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End of life ethics, serious illness, and veterinarian wellbeing: An upstreamist approach
Veterinary Ethics 101: Developing ethical literacy to cope with daily challenges in practices

Veterinary Informatics
Dr. Kelly Hall

Big data and trauma patient care

Dr. Rachael Kreisler

As you Likert: The anatomy and physiology of client surveys

Medical decision making: Evidence, values, economics, and best guesses

Veterinary Leadership

Dr. Betsy Charles

A better way to fight

Boomers are work-a-holics, X-ers are lazy, millennials are entitled and gen z is beyond help. How are we supposed to work together?
Can’t we all just get along?

Lead like a boss: Strategies for effective influence
Life is hard and not fair, so how do I thrive?

Who are you? Lessons from the greatest showman

Drs. Kimberly-Ann Therrien and Sarah Wooten

Don’t take your debt into retirement: Live with financial freedom sooner

Essential exchanges (Part 1): Are you avoiding conversations crucial to your (or your patients’) progress?
Essential exchanges (Part 2): Conversing with confidence in crucial situations
Fifty shades of greatness

Drs. Jeff Thoren and Robert Trimble; Elise Lacher, CPA and Peg Thoren

Enlightened Rebels make SHIFT happen! (Part 1): Escape the management matrix
Enlightened Rebels make SHIFT happen! (Part 2): Upgrade your operating systems for adaptability and collaboration
Enlightened Rebels make SHIFT happen! (Part 3): Unleash your team’s creativity, motivation, and accountability
Enlightened Rebels make SHIFT happen! (Part 4): Elevate your success through compelling
Veterinary Wellbeing
Dr. Katherine Goldberg

A social worker walks into a veterinary practice:
The why, what and how of veterinary social work
.............................................. Proceedings not provided

Veterinary social work and the bottom line: A session for practice owners and managers
.............................................. Proceedings not provided

Yoga won’t fix this: What do we really know about veterinarian wellbeing?
.............................................. Proceedings not provided

Dr. Sarah Wooten

Awaken your lionheart of courage and tame the fear of failure ........... Proceedings not provided
Our speakers are the best around

Celeste Allaband, DVM
Benita Altier, LVT, VTS (Dentistry)
Boaz Arzi, DVM, DAVDC, DEVDC, FAVDC
Mike Barletta, DVM, MS, PhD, DACVAA
Kevin Benjamino, DVM, DACVS
Mark Bobofchak, DVM, DACVO
Dawn Boothe, DVM, MS, PhD, DACVIM, DACVCP
Nicole Boyanosky
David Bruyette, DVM, DACVIM
Jamie Burkitt, DVM, DACVECC
Kara Burns, MS, Med, LVT, VTS (Nutrition)
Michelle Carnes, DVM, MS, DACVIM
Betsy Charles, DVM, MS
John Ciribassi, DVM, DACVB
Eli Cohen, DVM, DACVR
Brian Conrad, CVPM
Kelly Cronin, MBA, PHR, CVT, VTS (ECC)
Steve Dale, CABC
Lorelei D’Avolio, LVT, VTS (Exotics), CVPM
Darin Dell, DVM, DACVVD
Lena DeTar, DVM, DACVP, DABVP
Hilal Dogan, BVSc, CCTP
Robin Downing, DVM, MS, DAAPM, DACVSMR, CVPP, CCRP
Lauren England, DVM
Sue Ettinger, DVM, DACVIM
Eva Evans, DVM
Karen Felsted, CPA, MS, DVM, CVPM, CVA
Bronwyn Fullagar, BVSc, MS, DACVS
Mary Gardner, DVM
Jon Geller, DVM, DACVB
Katherine Goldberg, DVM, LMSW
Cheryl Greenacre, DVM, DABVP
Kristina Gulbrand, CVT, BS, CSP, ACC
Kelly Hall, DVM, MS, DACVECC
Bash Halow, LVT, CVPM
Cailin Heinze, VMD, MS, DACVN
Liz Hughston, Med., RVT, VTS (SAIM, ECC)

Jennifer Johnson, VMD, CVPP
Ari Jutkowitz, VMD, DACVECC
Kristin Kirkby Shaw, DVM, MS, PhD, DACVS, DACVSMMR
Rachael Kreisler, DVM, DACVP, MSCE
Elise Lacher, CPA
Yuri Lawrence, DVM, MS, MA, PhD, DACVIM
Heidi Lobprise, DVM, DAVDC
Tasha McNerney, CVT, CVPP, VTS (Anesthesia/Analgesia)
Karen Moriello, DVM, DACVD
Ashleigh Newman, VMD, DACVP
Jeff Nichol, DVM
Dave Nicol, BVMS, MRCVS
Mark Olcott, DVM, MBA
Gary Oswald, DVM, MS, DACVIM
William Rausch, DVM, DACVIM
Larry Rawson, DVM
Heidi Reuss-Lamky, LVT, VTS (Anesthesia/Analgesia, Surgery), FFCP
Bill Schroeder
Oriana Scislowicz, LVT, aPHR
Barbara Sherman, MS, PhD, DVM, DACVB, DACAW
Robert Silver, DVM
Jamie Snow, DVM
Laura Stokking
Kendall Taney, DVM, DAVDC, FAVD
Kimberly Ann Therrien, DVM
Peg Thoren, M.Ed., BCC
Jeff Thoren, DVM, PCC, BCC
Robert Trimble, DVM
Amanda White, DVM
Tina Wismer, DVM, MS, DABVT, DABT
Michael Wood, DVM, PhD, DACVIM
Sarah Wooten, DVM, CVJ
Bonnie Wright, DVM, DACVAA
Tosha Zimmerman, CVT
Anesthesia for the Animal with Gastrointestinal and Hepatic Disease

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Patients with gastrointestinal and hepatic diseases have an increased anesthetic risk. It is important to know if the disease is treated and under control before anesthetizing these patients. Ideally these animals should be stabilized before the procedure whenever possible. Fluid, electrolyte, and acid-base disturbances should be corrected prior to anesthesia. As in all patients, a thorough physical examination is paramount. A complete blood work including cell blood count, serum chemistry and electrolytes, and urine analysis are recommended. Abdominal radiographs and ultrasound should also be evaluated before anesthesia.

**Acute GI Diseases:**

**Pathophysiology**

These patients can present obstructions (foreign bodies, masses, intussusception) or a perforation of the GI tract. Nausea and vomiting can be present and can lead to dehydration, electrolyte abnormalities, acid-base imbalance. Loss of GI vascular and mucosal integrity can lead to leakage of toxic substances and sepsis. These patients are susceptible to bacteremia, endotoxemia, sepsis, hypotension, and arrhythmias. If abdominal distension is present, the animal can show signs of respiratory distress, poor tissue perfusion, and pain.

**Preanesthetic preparation**

Evaluate the hydration of the patient and treat shock if present. Aggressive fluid therapy may be necessary before anesthesia. Electrolytes and acid-base abnormalities should be evaluated and treated. Abdominal distension and pain need to be addressed as soon as possible. A large needle can be used as a trocar to decompress the stomach. Alternatively, a gastric tube can be used as soon as the animal is induced. Antiemetic drugs, such as metoclopramide and maropitant, can be administered in the pre-anesthetic period (avoid metoclopramide if a GI obstruction is suspected). Endotracheal intubation and protection of the airway should be established quickly to prevent aspiration in case of vomiting/regurgitation. Antimicrobial therapy should be administered in septic patients. Before induction of general anesthesia, preoxygenate the patient, evaluate the ECG and treat arrhythmias if present.

**Drug considerations**

In ill patients, opioids and benzodiazepines can provide analgesia and sedation with minimal effects on the cardiovascular system. In severely compromised patients combinations of these drugs can be sufficient to place an endotracheal tube. Acepromazine can cause hypotension, has a long duration, and provides no analgesia. For sick patients it is best to avoid this drug. Alpha-2 agonists should also be avoided due to depression of the cardiovascular system. Ketamine can be used in combination with benzodiazepines to induce general anesthesia. Alternatively, propofol can be administered. Propofol can cause hypotension (administer slowly to decrease this risk), has short duration, does not provide analgesia, and causes respiratory depression. Isoflurane and sevoflurane can be used to maintain general anesthesia, however they can cause hypotension. Use injectable drugs (i.e. opioids, lidocaine, ketamine) administered as CRI to decrease the amount of inhalant required.

**Intraoperative and post-operative monitoring/support**

Mechanical ventilation can improve O₂ delivery, however it can also decrease venous return to the heart (positive intrathoracic pressure during inspiration) and decrease cardiac output, blood pressure, and tissue perfusion. Minimize inhalant concentration by using drugs such opioids and ketamine, since high levels of inhalant can lead to hypotension. In most cases an arterial catheters should be placed to allow for invasive blood pressure and arterial blood gas monitoring. Positive inotropes may be needed before, during, and after the anesthetic episode. An ECG should be constantly evaluated for arrhythmias. Packed cell volume, total protein, glucose, and blood gas values should be checked regularly during anesthesia. Fluid therapy may include crystalloids, colloids, and blood products. Analgesic drugs should be administered before, during, and after the anesthetic episode. Monitor and
maintain a normal body temperature throughout the procedure. Oxygen supplementation may be required in recovery.

**Chronic GI diseases:**

**Pathophysiology**

Chronic GI diseases usually cause a decreased in nutrient availability with consequent weight loss, malabsorption and hypoproteinemia. Special attention should be given to the albumin level and glucose. Animals with chronic GI disease may need plasma or a synthetic colloid such as hetastarch prior to anesthesia if the protein level is less than 4.0 g/dl or the albumin is less than 1.5 g/dl since proteins are necessary to maintain a normal oncotic pressure. Most anesthetic drugs are protein-bound and a decrease in albumin causes and increase in free drug in the plasma. It may be important to consider decreasing the dose of drugs that are highly protein bound.

**Hepatic disease**

The liver plays an important role in the synthesis and homeostasis of several products, including glucose, plasma proteins (including albumin), clotting factors V, VII, IX, XI, XII, XIII, fibrinogen, prothrombin, prekallikrein, plasminogen, alpha2-antiplasmin, antithrombin and others. The liver is also responsible for the biotransformation and elimination of many drugs. Hepatic enzyme activity is not indicative of hepatic function. Pre- and postprandial bile acids and ammonia are used to assess hepatic function.

**Precautions for anesthesia in the patient with liver disease**

1. Avoid drugs requiring extensive hepatic metabolism or excretion. If this is necessary, decrease the dose if possible.
2. Maintain adequate cardiac output and blood pressure to prevent poor hepatic flow.
3. Avoid hypoxemia as it can lead to hepatic hypoxia.
4. Check the coagulation status of the patient and be prepared to treat. Be prepared for a blood transfusion if clotting times are abnormal.
5. Monitor and treat hypoproteinemia and hypoglycemia before and during the anesthetic event.

**Drug considerations an anesthetic management**

The use of short acting and reversible drugs is recommended. Phenothiazines (acepromazine) require extensive hepatic metabolism and can cause hypotension. In patients with hepatic disfunction these drugs can prolong the recovery time. Alpha-2 agonists cause depression of the cardiovascular system leading to decreased splanchnic blood flow and potential hypoperfusion and tissue hypoxia. They provide sedation and analgesia and can be reversed. Most opioids are metabolized in the liver, however hepatic blood flow is more important that the enzymatic activity for their biotransformation. They provide analgesia and are reversible. Benzodiazepines have a wide safety margin, are reversible, are metabolized in the liver and do not provide analgesia. Propofol undergo hepatic and extra-hepatic metabolism, has a short duration of action, and does not provide analgesia. Isoflurane and sevoflurane can be used to maintain general anesthesia.

Patients with hepatic disease may need intravenous colloids due to decreased oncotic pressure. If hypotension is present, inotropic therapy should also be considered. Monitor blood glucose and supplement with 1-5% dextrose if necessary.

Patients with portosystemic shunt can present with hypoglycemia, coagulopathies, hypoalbuminemia, abnormal response to drugs metabolized in the liver, and neurologic signs such as hepatic encephalopathy and seizures. Prior to and during anesthesia check plasma protein levels, glucose, and acid-base status. These patients often need colloids, glucose supplementation and correction of metabolic acidosis.
Inhalational Anesthetics

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Inhalational anesthetics are commonly used in veterinary medicine to maintain general anesthesia. They are delivered to the patient via the anesthesia machine through the lungs and they do not require hepatic metabolism for their excretion. The amount of inhaled anesthetic can be easily monitored and controlled. The response to these agents is somehow predictable, since it is very similar among species. The main disadvantage is that an anesthesia machine is required to deliver inhalants, and these machines are bulky, expensive, and they need periodic maintenance.

Gas anesthesia is the most common technique used for general anesthesia in small animals, however the mechanism of action of these drugs is still unknown. There are several theories that try to explain how these gases produce anesthesia, but none of them fully explains their mechanism of action.

Vapor Pressure
All inhalants, except for nitrous oxide, are administer as vapor. A vapor is and agent in its gaseous phase that can be condensed to a liquid by increasing the pressure without reducing the temperature. This is possible because the room temperature is lower than the critical temperature of that specific vapor. The critical temperature is the temperature above which the gas cannot be liquified by pressure alone. The boiling point is the temperature at which the vapor pressure equals the barometric pressure. The vapor pressure is the pressure exerted by a vapor when it exists in equilibrium with its liquid state. We refer to partial pressure when the gas is mixed with other gasses. In anesthesia, inhalants are measured in volume %, which represents the ratio between vapor pressure and barometric pressure (Vol% = vapor pressure/barometric pressure x 100). This means that 2% of isoflurane is equal to 0.02 x 760 mmHg (barometric pressure at sea level), which is 15.2 mmHg. The vapor pressure is temperature dependent and an increase in temperature will increase the vapor pressure of an inhalational anesthetic. According to Dalton’s law the total partial pressure of a gas mixture is the sum of each gas partial pressure present in the mixture.

<table>
<thead>
<tr>
<th>Inhalant</th>
<th>Boiling point (°C at 760 mmHg)</th>
<th>Vapor pressure (mmHg at 20°C)</th>
<th>Maximum concentration (Vol% at 20°C, 760 mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoflurane</td>
<td>48.5</td>
<td>238</td>
<td>31.3</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>58.6</td>
<td>157</td>
<td>20.7</td>
</tr>
<tr>
<td>Desflurane</td>
<td>22.8</td>
<td>669</td>
<td>88.0</td>
</tr>
</tbody>
</table>

Solubility
When we deal with injectable drugs, a solute is the substance dissolved in the solvent. When heat is applied to the solution, we can dissolve more solute in it. For gases dissolved in solvents, things are different. When we apply heat to a solution containing gas, more molecule of that gas escape from the solvent into the gaseous phase. When a gas is dissolved into a solute (i.e. liquid), there is a constant net movement of its molecules, until the equilibrium between the dissolved gas in the liquid and the undissolved gas above the liquid is reached. At this point the net movement stops. The amount of molecules dissolved into a solvent at equilibrium depends on the specific gas (and its partial pressure), the solvent, and the temperature. However, when equilibrium is reached, the partial pressure of the gas (but not the amount of molecules!) in the air above the liquid and the partial pressure of the gas dissolved in the liquid will be the same. The solubility of inhalational anesthetics in blood is measured using the blood/gas partitional coefficient, which describes how the gas partitions itself between dissolved (in the blood) and the undissolved (in the alveoli) phases. The higher the blood/gas partitional coefficient, the more soluble the anesthetic is in the blood. Inhalants with higher blood/gas partitional coefficient will take longer to reach equilibrium. In general this means that if an anesthetic agent is more soluble in blood it takes longer to induce general anesthesia using this inhalant via facemask, longer to recover, and longer to make a change in the anesthetic depth. In other words solubility means speed and control. The
The blood/gas partitional coefficient of isoflurane, sevoflurane, and desflurane at 37ºC are 1.4, 0.6, and 0.4, respectively.

**MAC**

The minimum alveolar concentration (MAC) is the volume % at 1 atmosphere (760 mmHg) of an inhalation anesthetic required to prevent movement in 50% of subjects exposed to a supramaximal stimulus. MAC is the equivalent of the effective dose50 (ED50) and it is a measurement of potency. The lower the MAC the greater the potency. MAC can be used to compare different inhalants and to predict their pharmacodynamic effects, since they cause similar changes at equipotent MAC multiples. MAC is species specific and allows to predict what percentage we should use to prevent movement during surgery. Usually 1.3-1.5 x MAC prevents movement in 95% of subjects. MAC values are additive and 2 inhalants used in the same patient work synergistically. All anesthetic drugs that have anesthetic-sparing effects, decrease MAC, and that is why when we use some of this drugs for premedication or during surgery (i.e. opioids), so we can use less inhalant to achieve a surgical plane of anesthesia. Other factors that decrease MAC include: age (geriatric patients), severe hypotension, hypoxemia, and hypercarbia, hypothermia, hyponatremia, pregnancy, and metabolic acidosis.

<table>
<thead>
<tr>
<th>Inhalant</th>
<th>MAC in the dog (%)</th>
<th>MAC in the cat (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoflurane</td>
<td>1.3-1.4</td>
<td>1.3-1.6</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>2.3</td>
<td>2.6-3.4</td>
</tr>
<tr>
<td>Desflurane</td>
<td>7.2-8.2</td>
<td>9.8-10.2</td>
</tr>
</tbody>
</table>

**Delivery and uptake**

Inhalational anesthetics move following a partial pressure gradient, from areas with high partial pressure to areas with low partial pressure until equilibrium is reached. At the beginning of the anesthetic event, the inhalant moves from the anesthesia machine to the patient's alveoli. The change in partial pressure of the anesthetic in the alveoli is influenced by the delivery and by the uptake. To increase the partial pressure in the alveoli:

- Increase delivery of inhalant to alveoli by
  - Increasing inhaled partial pressure
  - Turn up the vaporizer
  - Increase the fresh gas flow
  - Minimize the volume of the breathing circuit
  - Increasing alveolar ventilation
  - Increases delivery of the anesthetic to the alveoli

- Decrease the uptake factors
  - Solubility
  - Cardiac output
  - Alveolar-venous partial pressure difference

**Elimination**

This process is the opposite of the delivery and uptake, with some differences. During the first period of the anesthetic event, we can increase the delivery by using the overpressure techniques (high % of inhalant), however this is not possible during recovery (we cannot deliver less than 0%). The only thing we can do is flushing the breathing circuit with fresh O2 during recovery to increase the partial pressure difference between alveoli and anesthesia machine and to facilitate the elimination of the inhalant. The duration of anesthesia is also important. The longer this period is, the more anesthetic is absorbed in the tissues and the longer the recovery is. This is more evident for inhalants with a higher blood/gas partition coefficient (higher solubility).
Understanding Pain and Nociception: Pathway and Assessment

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The word "pain" comes from the Latin word "poena" meaning a fine, a penalty. The International Association for the Study of Pain (IASP) defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage”. Pain can be adaptive when it contributes to survival by protecting the organism from injury or promoting healing when injury has occurred or maladaptive when it becomes an expression of the pathologic operation of the nervous system. Acute pain is physiologic, however chronic pain is pathologic and can follow acute pain if not prevented or treated.

Physiology and pathophysiology

- Transduction occurs in the periphery and involves many different receptors called “nociceptors”. They convert energy from one form (which can cause tissue damage) to another (electrical impulse). Based on the type of stimulus that these nociceptors can process, they are classified as: mecanoceptors, thermoceptors, and chemoceptors. They are the peripheral end-terminal of Aδ and C-fibers. Mechanical, thermal, and chemical stimuli open different ion channels (i.e. potassium and sodium channels) located at the end-terminal of these nerve fibers activating this first phase of nociception (transduction). If this is not prevented/treated, it can lead to peripheral sensitization: tissue damage causes release of inflammatory mediators, such as histamine, bradykinin, tumor necrosis factor-alpha (TNF-α), interleukin-beta (IL-β), interleukin-6 (IL-6), nerve growth factor (NGF), adenosine, hydrogen ions (H+), adenosine triphosphate (ATP), and prostaglandin E2 (PGE2) which can bind to peripheral receptors and recruit more inflammatory cells (inflammatory soup). These inflammatory mediators will also decrease the nociceptors threshold (i.e. heat threshold for TRPV1 from 42°C to 35°C), they can activate silent nociceptors (see modulation) and cause hyperalgesia (increased reaction to a painful stimulus) and allodynia (pain caused by a stimulus which is not normally painful like touch or intense light).

- Transmission occurs when the electrical signal moves from the periphery to the spinal cord. The electrical impulse travels from the periphery to the spinal cord through the Aδ and C-fibers. The Aδ-fibers are myelinated with a diameter of 1-5 μm and conduct impulses rapidly at a rate of 5 to 30 m/sec. They have a small receptive field and specific high threshold ion channels that are activated by noxious thermal or mechanical input. There are 2 types of Aδ-fibers: type I with high mechanical and high heat threshold and type II with high mechanical and low heat threshold. Aδ-fibers can be unimodal (transmit only one type of signal, i.e. mechanical or heat) or polymodal (transmit more than one type of signal, i.e. mechanical and heat). They are responsible for sharp, fast, transient, and well localized pain. The C-fibers do not have myelin and their diameter is 0.25-1.5 μm with conduction velocity of only 0.5 to 2 m/sec. Their receptive field is larger compared to A-δ fibers and they are responsible for slow, poorly localized, burning, gnawing sensation of second pain, which persists after termination of the noxious stimulus. C-fibers are polymodal (i.e. mechanical/heat, mechanical/cold, and mechanical/cold/heat). In normal circumstances, Aβ-fibers, myelinated with fast conduction velocity and diameter ranging from 5 to 15 μm, are responsible for transmission of touch and pressure. During chronic or pathologic pain Aβ-fibers contribute to transmission of pain due to peripheral sensitization (inflammatory soup) and central sensitization (see modulation).

Once the nociceptors is stimulated in the periphery, the electrical signal travels from the periphery to the dorsal horns of the spinal cord through the nerve fibers via ion channels (sodium, potassium, and calcium channels). Aδ-fibers synapse in lamina I-V of the spinal cord and C-fibers in lamina II (also called substantia gelatinosa).

- Modulation takes place in the periphery, in the spinal cord, and in supraspinal structures. 1) Peripheral modulation. C-fibers and different substances can modulate (increase or decrease) response to stimuli. Silent nociceptors, a type of C-fibers, are normally unresponsive to noxious mechanical stimulation (high threshold) and become “awakened” (responsive) to mechanical stimulation during inflammation and after tissue injury. Peripheral opioid receptors, nociceptor sensitizers, such as prostaglandins, leukotrienes, bradykinin and nociceptor activators, such ad K+, ATP, substance P, and bradykinin can all play a role during peripheral modulation. 2) Spinal modulation. The response to peripheral nociceptive stimuli can be modified in the spinal cord due to release of local endogenous modulators and activation of descending pathways, which can decrease the response to nociception (i.e. opioids, serotonin, norepinephrine) or increase this response (i.e. glutamate, prostaglandins, substance P). When the response to nociception is enhanced, central sensitization can occur. This requires a brief intense nociceptor activity such a surgeon cutting through skin or can be the result of inflammation and nerve injury. Repeated impulse activity of C-fibers produces sensitization of the spinothalamic tract neurons over time, which leads to increased spontaneous impulse activity and enhanced response to
impulses (hyperalgesia and allodynia). The AΔ and C-fibers release a variety of neurotransmitters in the dorsal horn of the spinal cord such as, glutamate, ATP, calcitonin gene-related peptide (CGRP) and substance P and activate normally quiescent N-methyl-D-aspartate (NMDA) receptors, main receptors involved in the central sensitization. Down regulation of GABAergic and glycine receptor activity and activation of microglia also occur during central sensitization. 3) Supraspinal modulation. Descending pathways are able to modulate the pain response. Descending modulation is controlled by the periaqueductal gray matter (PAG) of the midbrain, the medulla and pons of the brainstem (nucleus raphe magnus), and thalamocortical structures. These structures release mediators, such as endorphins, enkephalins, dynorphins, serotonin, and norepinephrine which down regulate nociception.

- **Projection** involves transport of the electrical signal from the spinal cord to the supraspinal structures. The projection of the signal involved 3 main large tracts: 1) the spinothalmic tract – from lamina I-V to thalamus; 2) the spinoreticular tract – to reticular formation and thalamus; 3) the spinomesencephalic tract – to the midbrain.

- **Perception** occurs in the cerebral cortex where the electrical impulse in perceived as pain. Nociception involves all the physiological and pathological pathways and processes that lead to pain, except there is no conscious perception during only nociception.

**Pain recognition and assessment**

Recognizing pain in animals may be challenging and its assessment varies among different species and breeds. Training is important and some key points should be considered: Anticipate level of pain (severe, moderate, mild), keep in mind differences in pain behavior (i.e. demeanor, age, species), wide observation (recognize obvious and subtle signs), use response to pain therapy as a pain diagnostic tool, integrate physiologic, behavioral, and physical signs.

Different pain scoring systems, which may take in consideration different parameters, can be used to assess pain. They can include objective parameters, such as hear rate, respiratory rate, plasma cortisol/endorphins, and more subjective variables, such as observation alone or with verbal interaction and palpation of the painful site. Pain scales can be **unidimensional**, such as a simple descriptive scale (i.e. Colorado State acute feline/canine pain scale), numerical rating scale (NRS), visual analogue scale (VAS), and dynamic interactive visual analogue scale (DIVAS) or **multidimensional** such as the University of Melbourne Pain Scale (UMPS) and the Glasgow Composite Measure Pain Scale-short form (CMPS-SF).

The simple descriptive scale assigns a descriptor (i.e. no pain, mild pain, moderate pain, severe pain) based on the observation of the animal. The NRS is very similar, but it uses numbers instead of descriptors. These numbers act as a rating: the greater the number, the more intense the pain. For example, a score of 0 would indicate no discomfort present while a score of 10 would indicate extreme pain. The VAS is a scale consisting of a straight line 10 cm long with the left end, 0 cm, indicating no pain and the right end, 10 cm, indicating unbearable pain. The user places a mark on the line that best represents the intensity of the subject’s pain. The closer the mark is toward one end, the greater or weaker the intensity of pain the user believes the patient is suffering. Although the results of the VAS are somewhat subjective and the scale has been deemed unreliable by expert anesthesiologists, it has been and still is widely used in veterinary medicine. The DIVAS is similar to the VAS, but it also includes verbal and physical interaction with the animal.

The CMPS-SF is a questionnaire-based scaling system rather than a simple scale. The VAS and the NRS measure only one dimension (the intensity) of the pain experience, whereas the CMPS-SF and other multidimensional or composite rating scales also take into account the sensory and affective qualities of pain. This scale includes six behavioral categories with different descriptive expressions (descriptors) for each category: vocalization (4 descriptors), attention to wound (5 descriptors), mobility (5 descriptors), response to touch (6 descriptors), demeanor (5 descriptors) and posture/activity (5 descriptors). Descriptors are placed in increasing order of pain intensity and numbered accordingly. The user assigns the score that best describes the animal’s behavior. For example, under the category labeled “Vocalization,” there are four descriptors “Quiet”, “Crying or whimpering”, “Groaning”, and “Screaming,” marked 1, 2, 3, and 4, respectively. The user scores the animal by marking the descriptor that best describes the animal behavior (e.g. an animal whimpering would get a score of 2). The scores for each category are summed together, and the pain intensity is determined based on the total score. The CMPS-SF has good inter-observer correlation for postoperative pain assessment, but studies reported that sedation might play a confounding role during the evaluation. The maximum score for the 6 categories is 24, or 20 (for 5 categories) if mobility is impossible to assess. Recommended analgesic intervention level is 6/24 or 5/20 (if mobility is impossible to assess).
Sedation and Anesthesia for Donkeys and Mules

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Despite similarities with horses, donkeys and mules have unique characteristics that should be considered before sedating and anesthetizing these animals.

Behavioral differences:
- Donkeys become immobile when facing new situations (i.e. new environment)
  - After acclimation it is easier to handle them (patience is required!)
  - Nose twitches are not very effective (hard to place and they slide off easily)
  - Head ropes tied to a fixed object may work
- Mules can be even more difficult than donkeys
  - They are larger and can be more dangerous
  - Easier if they are trained
  - Individual variability
- Both can bite and kick without warning
- They are very stoic and assessing pain can be challenging

Anatomical differences:
- Jugular vein is deeper than in horses
  - Thick skin and cutaneous colli muscle
  - Since the cutaneous colli muscle is thick and is located over the jugular vein, distention of this vein can be achieved using a neck rope
  - Angle the needle more perpendicular to the skin than in horses
  - Use lidocaine SQ and make a small skin incision before placing the catheter
- Branches of the facial artery may have different anatomical location than in horses
  - Finding the pulse and placing arterial catheters can be more challenging

Physiological differences:
- In donkeys PCV increases only when severely dehydrated (20-30%). Mild dehydration is hard to assess
- Donkeys' body temperature can increase more than in horses when exposed to hot climate and after exercise
- Donkeys' resting respiratory rate is 20-30 breaths/min, which is higher than in horses
- Heart rate is similar to horses and it is a good indicator of pain (better than behavioral changes)
- Mules and donkeys metabolize drugs differently than horses, and they can be more or less sensitive to some drugs (doses and/or intervals should be modified)

Anesthesia

Premedication:
- Similar drugs used in horses
- Detomidine 5-10 µg/kg provides adequate analgesia, which becomes profound at 20-40 µg/kg. Sedation is adequate and increases slightly at 40 µg/kg. Duration of sedation is dose-dependent
- Sublingual detomidine gel has been used in donkeys (20-40 µg/kg)
  - Sedation and analgesia within 30-40 min
  - Sedation is dose-dependent (better at 40 µg/kg)
- Acepromazine 0.04-0.05 mg/kg provides adequate sedation but does not increase sedation when combined with detomidine in donkeys
- Mules require 50% more xylazine than horses and donkeys (1-1.6 mg/kg)
- Untrained, excited, and feral donkeys and mules are dangerous and might require higher doses
- Donkeys may lie down after premedication (general anesthesia can be induce in sternal recumbency)

Induction and maintenance of general anesthesia:
- Ketamine and diazepam are usually used to induce anesthesia
- Ketamine is more rapidly metabolized and redistributed in mules and donkeys
  - It only lasts 10-15 min
  - Needs to be re-dosed more frequently
- This rapid metabolism of ketamine is especially evident in miniature donkeys
- Anesthesia can be maintained with xylazine/ketamine IV boluses or GKX
- Donkeys are more sensitive to respiratory depression caused by guaifenesin
  - GKX for donkeys (5% GG 1L + xylazine 500 mg + ketamine 2 gr). Two grams of ketamine are recommended due to the rapid metabolism and to reduce the amount of GG administered.
- Tilazol (tiletamine + zolazepam) can be used for a slightly longer duration of anesthesia (1.1 mg/kg) in miniature donkeys
- Isoflurane and sevoflurane are used for procedures longer than 1 hour and their effects are similar to the ones described in horses
- Dwarf-like miniature donkey may have hypoplastic trachea and abnormal airway (keep head and neck extended and place endotracheal even if only injectable drugs are used)

**Monitoring:**
- Eye signs (palpebral and nystagmus) are similar to horses, but less reliable in donkeys
- Blood pressures are good indicator of the plane of anesthesia
- Respiratory rate is higher in donkeys compared to mules. Like in horses, breath holding might indicate a light plane of anesthesia

**Recovery and analgesia:**
- Donkeys lie down until they are able to stand. They usually experience a quieter and calmer recovery compared to horses
- Recovery in mules is variable and assisted recovery might be needed
- Myositis and myopathies can occur in mules, especially draft mules, but unlikely in donkeys
- For analgesia alpha-2 agonists and opioids can be used for analgesia
  - Mules require higher doses of xylazine
  - Butorphanol is used at 0.02-0.04 mg/kg in donkeys and mules
- NSAIDs can be used for pain management
  - Phenylbutazone 4.4 mg/kg IV, PO BID-TID in donkeys and bid in mules
  - Flunixin 1.1 mg/kg IV TID in donkeys
- Administration of analgesic agents might have to be repeated more frequently than in horses
- Frequent assessment for signs of pain is recommended
Sedation and Anesthesia in the Pig

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When general anesthesia or heavy sedation are planned, pigs should be fasted for 12-24 hours and water should be withheld for few hours before the procedure. Piglets should be fasted for 2-4 hours.

Pigs are difficult to restrain due to their demeanor and the shape for their body. Different techniques used to restraint pigs include:
- Snout snare
- Sling (used in research setting)
- The use of 1 or 2 boards to confine the pig
- The use of a bucket over the head and push the back end of the pig against a corner

When working with pigs, try to be quick and minimize the restraint to decrease stress.

Physical exam might be limited due to their demeanor and often they will need to be sedated to collect blood. Drugs can be injected IM in the neck, in the epaxial or in the gluteal muscle. Avoid these 2 last locations if the pig is meant for human consumption. When injecting drugs IM, use a luer lock syringe and a long needle (1.5-2 inch or longer in larger pigs). Use a bigger syringe that you think you might need (i.e. use a 6 ml syringe for 3 ml volume), this way the injection will be easier and faster.

IV access can be obtained by placing a catheter in the auricular vein in most pigs. In pot-bellied pigs this site might be very challenging. Alternative locations are the cephalic and saphenous vein.

- Drugs that can be used in pigs for sedation include:
  - Tranquilizers – acepromazine, azaperone. They provide sedation, however they do not provide analgesia, they are non-reversable and they have a long duration of action. They can cause hypotension.
  - Benzodiazepines – diazepam, midazolam. They provide muscle relaxation but not analgesia. They can be reversed (with flumazenil), however they are not good sedative in pigs if used on their own.
  - Alpha-2 agonists – xylazine. Good sedation and analgesia, but inconsistent response if used on its own. Pigs are more resistant to the effect of xylazine compared to other species and higher doses should be used to achieve adequate sedation.
  - Opioids – butorphanol, buprenorphine, hydromorphone. They provide analgesia, but they may cause dysphoria or aggression if given to healthy non-painful pigs without any sedation.
  - Dissociatives – ketamine, tiletamine. High therapeutic index and good restraint/anesthesia. They cause discomfort upon injection and they do not provide muscle relaxation.

Examples of protocols for sedation and anesthesia

- Example 1
  - Xylazine 2 mg/kg IM
  - Midazolam 0.2 mg/kg IM
  - Ketamine 2-5 mg/kg IM

The drugs should be mixed together in one syringe to allow for a single IM injection. If mild/moderate sedation is required, ketamine can be used at 2-3 mg/kg. If general anesthesia (i.e. using an inhalant agent) is planned, use ketamine at 5 mg/kg. If this is not enough to allow for endotracheal intubation, an IV catheter can be placed and more ketamine of propofol can be used until the animal reaches and adequate plane of anesthesia that allows for intubation. Alternatively, an inhalant anesthetic can be delivered via facemask until intubation is possible. In pigs weighing more then 150 kg, these doses can be reduced by 15-20%.

An opioid, such as hydromorphone (0.05-0.1 mg/kg) or buprenorphine (0.02 mg/kg) can be added if more analgesia is required.

If the procedure is not painful (i.e. of hoof and tusk trimming), xylazine can be reversed with tolazoline and midazolam can be reversed with flumazenil.
- Example 2
- Telazol, which is tiletamine + zolazepam (1 vial = 500 mg) + xylazine (5 ml of 100 mg/ml = 500 mg) → TX. Use 1 vial of Telazol (powder) and dilute it by using 5 ml of 100 mg/ml xylazine.

Give 1 ml/100 lb (2.2 mg/kg of telazol and 2.2 mg/kg of xylazine) IM for up to 300 lb of body weight. If the pig weighs more than 300 lb, give 0.5 ml/100 lb for every extra 100 lb (over the first 300 lb). For example, if the pig weighs 400 lb pig, give a total of 3.5 ml of TX. An opioid can be added if more analgesia is required. Xylazine and zolazepam (contained in the Telazol) can be reversed with tolazoline and flumazenil, respectively. This drug combination is associated with a longer recovery than the protocol described in example 1.

Orotracheal intubation can be challenging in pigs, due to their long snout, narrow dental arcades, small tracheal diameter, laryngeal diverticulum, and long soft palate. Laryngeal spasm can also occur during intubation.

- Equipment needed for orotracheal intubation:
  - Laryngoscope (use a long blade for larger pigs)
  - Lidocaine (in a syringe with long cannula to desensitize the arytenoids)
  - Stylet (longer than the endotracheal tube, useful in larger pigs)
  - Several sizes of endotracheal tubes
  - Gauze strips (to hold mouth open)
  - Lube
  - Cuff syringe

After induction, keep the animal in sternal recumbency. Use the stylet for larger pigs. When the endotracheal tube tip is between the arytenoid cartilages, rotate the tube 180 degrees while gently pushing it into the trachea. If using a stylet, make sure the tip exits the endotracheal tube at the patient end. Place the stylet tip between the arytenoids and while holding still the stylet from the other end (distal to the patient, where the endotracheal tube connects to the Y piece of the anesthesia machine), insert the endotracheal tube using the same technique described above.

The pharyngeal recess (or pharyngeal diverticulum) is located immediately dorsal to the esophagus. If the endotracheal tube is placed in the diverticulum, it will not advance. If this happens, start over and point the endotracheal tube (or stylet) more ventrally.

Monitoring pigs under general anesthesia is similar to other species:
- Mucous membranes and CRT
- Pulses and heart rate - 60 to 90 bpm (depends on the size)
- Respiratory rate - 15 to 40 bpm
- Eye reflexes - eye position is variable, loss of corneal reflex with a dilated pupil is indicative of excess anesthetic depth
- ECG
- Blood pressure – the cuff can be placed on the front or on the back leg. An arterial catheter can be placed in the auricular, medial saphenous or femoral artery for invasive blood pressure monitoring
- Temperature - it may increase if the animal becomes stressed before the procedure.

Malignant hyperthermia is an acute and potentially fatal syndrome reported in pigs. It is an autosomal recessive disorder due to the mutation of the ryanodine receptor 1 gene, which controls Ca++ efflux from sarcoplasmic reticulum. Myocytes of animals affected by the mutation are unable to control this efflux. The symptoms can present very quickly, and include:
- Increase in body temperature
- Tachypnea
- Muscle rigidity
- Vasodilation
- Hypercarbia
- Metabolic acidosis
- Death

This syndrome can be triggered by:
- Volatile inhalants (especially halothane)
- Succinylcholine
- Excitement, stress, and rough handling
- Exercise
- High ambient temperature.
Treat immediately if malignant hyperthermia is suspected. Treatment includes:
- Immediate removal of offending agent
- Ventilation with 100% oxygen (do not use the anesthesia machine as traces of inhalant can be present)
- Cooling
- Supportive therapy for acidosis and shock
- Dantrolene IV
Breeds that can carry the mutation includes: Piétrain, Landrace, Large White, Hampshire, and Poland China.

During recovery, leave the endotracheal tube in place until the pig can chew and swallow. Do not feed or give water to the pig until he can walk. Do not put a recovering pig with other subjects, as they may attack and injure the recovering pig.
EMERGENCY PRESENTATIONS AND PROCEDURES IN BIRDS

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Introduction

Bird emergencies are similar to dog and cat emergencies, but may vary slightly, for example: trauma (hit by ceiling fan, toe closed in door, big bird/little bird incidents, dog/cat attack, burns), toxins (lead, zinc, PTFE fumes from Teflon), metabolic (hypovitaminosis A, hypocalcemia in African Grey Parrots), or infection (due to bacteria, virus, fungus or chlamyphila usually involving the liver, GI or respiratory tract. Because subtle clues of disease may go unrecognized by the owner until late in the course of the disease, birds can present with a terminal manifestation of chronic disease that has just recently showed overt acute signs.

Clinical goals

First, if possible, obtain a history over phone so as to be as prepared as possible when the bird arrives. Be familiar with common species problems (i.e. for a seizuring African Grey parrot have hypocalcemia at the top of your rule-out list). Evaluate the history, cage and husbandry, droppings, and observe the bird for clues as to the etiology before stressful restraint. Perform a rapid, but thorough PE, and diagnostic collection (+/- cbc, profile, radiographs, fecal gram stain); sometimes the bird may be so stressed that the exam may need to be performed in less than one minute. Obtain an accurate weight with a gram scale so as to administer accurate drug dosing. Provide supportive therapy to stabilize the patient including providing a warm (85-90°F) and stress free environment (no barking dogs), +/- O2, offer familiar/favorite foods and water that are elevated to sit right in front of the bird, provide 10 hours of daylight and 14 hours of dark, provide a low perch (birds insist on perching on the highest available perch, even when severely debilitated.

ABC’s (Airway, Breathing and Cardiovascular system) for the unconscious patient

First, check to see that the patient has a patent airway. Examples of a mass include, aspergillus granuloma, neoplasia, diphtheric membrane. Examples of a foreign body include a millet seed in a cockatiel trachea (this can be directly visualized in the trachea with a rigid 1.0 mm endoscope. Second, check to see if the animal is breathing, and if not then intubate (use uncuffed ET tubes in birds since they have complete tracheal rings). Provide intermittent partial pressure ventilation (IPPV) in birds at 1 breath/5sec. Due to the unique respiratory system in birds an air sac tube can be placed in caudal thoracic or abdominal air sac and oxygenated air will flow through the lung. An air sac tube can be connected to O2 or anesthesia, and left in place 5 days. To place an air sac tube, make a skin incision over the sternal notch area (borders are the last rib, the femur and the lateral processes of the vertebrae) and use a pair of hemostats to penetrate body wall, and then insert an ET tube. Third, check for heartbeat. If a bird experiences cardiac arrest, then the prognosis to reverse this situation is poor/grave due to a bird’s high metabolic rate and oxygen demands. The following treatments can be attempted to reinitiate heart beat: rapid heart massage and ventilate (100 beats per minute and 1 breath/5 seconds), epinephrine IV or IT (intratracheally), atropine (usually used to prevent bradycardia), dopram IV or IT (stimulates respirations), bolus IV fluids +/- 2.5 – 5% dextrose and/or colloids.

COMMON EMERGENCY SITUATIONS AND THEIR TREATMENTS

Blood loss

The average blood volume of a bird is approximately 10% of its body weight (BW). A healthy bird can lose up to 10% of their blood volume (or 1% of BW) without any adverse side effects. Unlike mammals, a healthy bird can usually lose up to 30% of their blood volume without dying due to compensatory mechanisms. Because of the these compensatory mechanisms, it is important to realize that the PCV in a bird is not accurate (i.e. not equilibrated) for 24 hours after a hemorrhagic incident because birds can compensate their PCV during blood loss by shunting blood from large skeletal muscle capillary beds and away from the kidneys via the renal portal system to increase blood to central areas. Therefore, an equilibrated PCV <15%, or an immediate PCV <20% are similar and serious enough to contemplate a blood transfusion. Fluids, hetastarch, oxyglobin or a blood transfusion (5% of BW) will help a bird with severe blood loss. The anemic patient may require vitamin B complex, iron dextran and vitamin K1.
Dehydration and Fluid Therapy
Most sick birds are 5-10% dehydrated. Clinical signs of dehydration include depression, reduced skin elasticity over digits, sunken eyes, cool digits, decreased refill time of the basilic (cutaneous ulnar) vein. Maintenance fluids are 50 ml/kg/day. The most commonly used fluids are LRS or Normosol-R since they most closely resemble the fluid lost. WARM (about 90-100°F) FLUIDS are imperative. SQ fluids are generally administered into the inguinal area in birds. Severe dehydration or shock requires rapid circulatory expansion with IV or even better, IO (intraosseous) fluids. Peripheral indwelling catheters have been avoided in birds since they have small fragile veins that easily form hematomas, their demnis is highly mobile causing difficulties in stabilizing the catheter, and they have refractory temperaments and a powerful beak. IO catheters are most commonly placed in the distal ulna or proximal tibiotalarsus. IO catheters should not be placed in a pneumatic bone as this may drown the bird when fluids are administered, since pneumatic bones communicate with the respiratory system. Likewise, intracoelomic fluids should not be administered as this may also drown the bird if fluids get into an air sac. To place an IO catheter: 1) pluck and aseptically prepare the carpus, 2) position needle in center of distal ulna, 3) support ulna and rotate catheter, 4) past cortex the catheter passes easily, 5) aspiration produces a small amount of blood, 6) anchor to soft tissue of carpus, 7) apply a figure-8 bandage.

Crop Gavage
Before crop gavage be sure the patient is hydrated. Start with a thin carbohydrate supplement and later use a juvenile parrot hand feeding formula or specially made avian critical care diet (high calorie, easy to digest). If a bird is losing weight, then it needs to be tube fed about 1-4 times a day. While hospitalized a bird is weighed daily in the morning on a gram scale. Technique: 1) restrain the bird in a normal upright position so as to avoid regurgitation and aspiration. 2) I prefer a stainless steel feeding needle with ball tip, others use a red-rubber catheter and a speculum to prevent the bird biting the tube in two. 3) aim from left commissure to right crop area. 4) avoid the large trachea and avoid excessive force so as not to puncture the esophagus. 5) be absolutely sure of placement by palpating and visualizing tube in crop before administering the formula. 6) if the bird regurgitates, then place it on the floor immediately. Approximate amounts: budgerigar - 1 ml, cockatiel - 3 -5 ml, Amazon parrot- 15 - 30 ml, cockatoo - 20 - 40 ml, macaw - 30 - 60 ml.

Egg Binding or Dystocia
Egg binding describes a condition in which a fully formed egg in the uterus cannot be layed. There are many causes for this condition including too large of an egg or a missshapen egg, inability of uterus to contract sufficiently from low blood calcium, or other causes of uterine paralysis. Clinical signs usually include straining to lay the egg which sometimes owners think is straining to defecate, dyspnea, fluffed appearance, anorexia and being at the bottom of the cage. Diagnosis can be made on history, physical examination and radiographs showing or palpating an egg or eggs. The radiographs can be taken unanesthetized in a cardboard box since positioning is not necessary to determine the presence of a calcified egg. Total and ionized calcium are helpful, as is a CBC and chemistry profile, but this is a true emergency and injectable calcium, subcutaneous fluids with or without 2.5% dextrose, and warm incubator are needed immediately. The primary cause is hypocalemia, usually from insufficient calcium in the diet, such as a seed diet. Oxytocin, or if available, vasotocin, or prostaglandin E or F can be given, but only if the egg is deemed able to come out. If the egg is too large or irregular to come out naturally, or if the egg putting pressure on the kidneys has progressed to shock, then the egg should be imploded via ovocentesis. Implosion involves creating a negative pressure within the egg to collapse the egg in on itself, not crushing the egg. Prevention of egg laying in the future involves removing all sexual stimuli such as mirrors, more than 10 hours of light daily, and male presence. Leuprolide acetate, a GnRH agonist, can also be given monthly, or a salpingohysterectomy can be performed.

Heavy Metal Toxicosis:
Heavy metal toxicosis is usually caused by ingestion of lead or zinc. Sources of lead include fishing weights, curtain weights, bullets, paint, and costume jewelry. Sources of zinc include pennies minted after 1986, Monopoly® game pieces, powder coating, paint, and costume jewelry. The ventriculus (gizzard) of birds retains heavy particles for grinding food, but in the case of heavy metal particles, they are retained and slowly digested allowing constant absorption of the toxins. Clinical signs include depression, weakness,
regurgitation, and sometimes neurological signs. Diagnosis is usually made by visualizing the metal dense particles on radiographs (X-rays), but a definitive diagnosis can be made on only 0.2 ml of blood for lead, or 0.2 ml of serum for zinc. Treatment consists of a chelating agent such as CaEDTA or dimercaptosuccinic acid or d-penicillamine to bind with the heavy metal rendering harmlessly urinated out of the body. Stressful procedures such as surgery or endoscopy to remove a large particle should be done after some chelation therapy, since stress can cause lead to move suddenly from the bone where it is stored to the blood and worsen clinical signs. Other products such as lactulose to assist the liver with toxicosis, or lubricants such as corn oil or peanut butter, or bulking agents such as psyllium, can also be given. Differential diagnoses include other neurological diseases, proventricular dilation disease, or heart disease including atherosclerosis.

Trauma
Common causes of trauma include flying into windows, ceiling fans or pots of boiling water, being bitten by dogs, cats, or other birds, or having toes cut off while standing on closing doors. Following initial assessment and obtaining an accurate weight, the patient should be placed in a quiet incubator with no heat. Fluid therapy, analgesia and NSAID therapy should be initiated and the patient should be monitored for return to normal behavior. Mild cases should resolve with this level of care, whereas more severe trauma may need advanced imaging to rule out a fractures or internal bleeding. Trauma to the body or limbs from a bite or laceration should be quickly assessed to estimate the amount of blood loss and control hemorrhage if still actively bleeding. Once the bleeding is controlled and the patient is stabilized flush the wound and debride any tissue that is deemed nonviable. Sterile saline with or without dilute povidone iodine may be used to flush the wound. This procedure may need to be performed under anesthesia as it can be quite painful. The choice to manage the wound as open or closed will depend on the cause and duration of the wound. Antibiotic therapy should be based on culture and sensitivity results whenever possible, but realize if a predator bit the bird, Pasteurella sp. are sensitive to enrofloxacin but not sulfa drugs. Fractures of peripheral limbs can be caused by a number of different traumas, but the patient must be stabilized prior to attempting any repairs. A fractured limb can be quickly bandaged while the patient is being treated with supportive care. A stable patient will be much more capable of handling the stress of anesthesia and surgical repair.

Acute dyspnea
This may truly be an acute onset, or the result of a chronic, worsening condition. Tracheal obstruction is one of the more common causes of acute dyspnea and can be caused by seed inhalation or by a granuloma or aspergilloma decreasing the tracheal lumen. Radiography may show changes in the tracheal lumen but endoscopy will be needed to identify the obstruction. An air sac cannula can be placed providing the bird with an alternate input of oxygenated air. Masses outside of the trachea may be approached surgically but the risk may be quite high. Dyspnea may also be due to inhaled toxins. While many inhaled toxins such as polytetrafluoroethylene (PTFE) gas, certain air fresheners or other aerosolized agents are often rapidly fatal, some patients may present having just been exposed to them. These patients will typically present severely dyspneic, weak and potentially unconscious. Oxygen therapy is the mainstay of treatment for this condition as well as subcutaneous fluid, bronchodilators and nebulization with saline. Prognosis for these patients is quite poor. Dyspnea can also be due to coelomic fluid that can usually be palpated and partly drained by coelomocentesis. Heart disease is a common cause of dyspnea in older birds.
GERIATRIC DISEASES OF PET BIRDS
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Introduction

Older and geriatric birds are presenting more commonly for veterinary care and certain diseases are more common in older birds than younger ones such as arthritis, neoplasia, cardiac disease, obesity, atherosclerosis, chronic malnutrition, and cataracts. Below is an overview of these diseases, including the most common forms of neoplasia based on species of bird and location of tumor, as well as methods of diagnosing and treating common geriatric diseases of pet birds from basic to complex.

Companion avian patients are presumably living longer because they are receiving better care than in years past, or are they? The larger parrots purchased during the time when pet birds were popular, specifically the 1980’s and 1990’s, are now nearing a geriatric age and are now presenting with diseases that are accompanied by older age. Diseases that are more common in an older bird are neoplasia, arthritis, cardiac disease, obesity, atherosclerosis, chronic malnutrition, chronic aflatoxin exposure, and cataracts. Diagnosis of these diseases may require invasive techniques, but not always, and treatment of these diseases is possible with varying degrees of success depending on the disease. In most instances, if the disease process was detected sooner rather than later, a better outcome could be expected. Therefore, it is incumbent upon us to educate the public on the importance of regular examinations, especially in geriatric birds.

What is old for a bird?

Lifespan refers to the period of time that an individual is alive. Therefore, the average lifespan is the life expectancy for a particular group or breed. Longevity is the maximum lifespan that can be expected under ideal conditions. Unfortunately, ideal conditions are not present in many captive situations and an individual’s lifespan may not reach maximum lifespan, or get even close. The lifespan of an individual bird depends on many factors including species, amount of genetic inbreeding, size, concurrent disease, diet and environment. Chronic malnutrition, specifically hypovitaminosis A, can lead to a decreased lifespan.

Recently a study evaluated 83,212 parrot life history records in the International Species Information System (ISIS) from zoos around the world providing us with some information on average lifespan and longevity of some species of parrots. (Table 1)

It is very difficult to age birds accurately and we are often at the mercy of the history we receive from the owner as to the age of the bird. Susan Clubb wrote an excellent article many years ago showing that aging macaws start to develop old age cataracts in their mid to late 30’s and arthritis in their 40’s. This paper also showed anecdotally that sometimes the skin is wrinkled around the feet or hocks of an older bird, or on the face of birds with featherless areas on their face. It was also noted that the muscles mass over the pectoral muscles is not as robust. Jesse Fallon wrote an article evaluating the feathers of birds, especially wild birds, for the formation of a crosslinked compound called pentosidine, an advanced glycation endproduct, that correlates with age in various species of bird from a 2x2 cm skin sample.

Neoplasia:

A list of the most common type of neoplasia found in each tissue of parrots is listed in Table 2. The most common tumor of captive parrots is the lipoma. There are many reports of which tumor is most common in various tissues, and the lists vary somewhat depending on the population evaluated.
Lipoma

Lipomas are accumulations of fat usually in the subcutaneous area over the breast and coelomic cavity area, but can also occur within the coelomic cavity. They are most often seen in some middle-aged budgerigars, Rose breasted cockatoos, and some older Amazon parrots. Dietary changes to healthier, lower fat diet and exercise can help, but there seems to be a genetic component. Other treatments may include surgery but severe hemorrhage is a concern. One paper by Ryan Devoe showed adding taurine to the diet as well as diet change may help. Do not place the birds on thyroid supplement as life threateningly high T4 levels can easily occur.

Renal adenocarcinoma

Renal adenocarcinoma is very common in budgerigars and some state that over 90% of budgerigars over 5 years of age will develop a leg lameness secondary to a renal tumor (usually renal adenocarcinoma) pressing on the ischiatic nerve (some refer to this as the sciatic nerve). This the highest tumor rate of any animal. A presumptive diagnosis is based on breed, age and clinical signs. A definitive diagnosis is based on renomegaly observed on radiographs and/or ultrasound. Sometimes a barium series can outline the GI tract being pushed ventrally by the tumor. Unfortunately, there is no surgical treatment described for this tumor.

Testicular tumors

Male budgerigars over 5 years of age can have a seminoma tumor or more commonly a Sertoli cell tumor that secretes estrogen and causes a male’s blue cere to turn brownish like a female budgerigar. No surgical treatment is described, but a recent paper suggested that leupralide acetate seems to help with clinical signs for about 6 months.

Other carcinomas

Anecdotally, cockatoos and Amazon parrots tend to get adenocarcinoma more often than other species. Respiratory adenocarcinoma can be found in the wing, axillary or lung area in cockatoos. The respiratory epithelium lining of the pneumatic humerus can develop a respiratory adenocarcinoma. In Amazon parrots bile duct carcinoma is found in the liver, especially if the bird also has papillomatous masses in the cloaca due to Psittacine Herpesvirus-3. Radiographs may indicate bony involvement of a respiratory adenocarcinoma or hepatomegaly in the case of bile duct carcinoma (a CT would be even better). A PET scan could also help indicate an area of neoplasia or infection/inflammation. A needle aspirate and cytology may aid in diagnosis of bile duct carcinoma, but an endoscopic or key-hole access hepatic biopsy is better. Treatment is surgical removal/debulking if possible, which is most successful with wing amputation of a respiratory adenocarcinoma in the humerus of a cockatoo.

Squamous cell carcinoma (SCC) is common and may be more often seen in Amazon parrots, macaws, and African grey parrots. It can be found in the mouth or beak area, or under the wing of the Amazon parrots and macaws, whereas it often involves the uropygial gland in African grey parrots. Biopsy is the best method of diagnosis. Radiation therapy at higher doses than that used in mammals did not slow the growth of SCC in a Buffon’s macaw (Volgeneau and Greenacre) and has not generally been recommended for treatment. A study by Barron-Wilson showed birds can tolerate high levels of radiation therapy with little effect. Treatment of skin SCC has been attempted with Strontium-90 topical radiation and intralesional cisplatin or carboplatin with some success after initial debulking procedures.
Chronic aflatoxin exposure

Chronic aflatoxin exposure occurs most commonly from eating peanuts either in or out of the shell. Since peanuts grow underground they invariably have some degree of Aspergillus fungus which produce aflatoxins. Even peanut butter made for human consumption has “acceptable levels” of aflatoxin. It is one of the most potent toxins, which when ingested chronically even in small amounts can cause characteristic bile duct hyperplasia. Presumptive diagnosis is based on history and elevated bile acids, but definitive diagnosis is based on a liver biopsy. There is no treatment to reverse the damage done, but colchicine can prevent further fibrosis from occurring. Perhaps lactulose and milk thistle (silymarin) can help the liver function.

Chronic malnutrition

Chronic hypovitaminosis A is the most common malnutrition that we see in pet birds, usually from eating an all, or mostly, seed diet. Years of not ingesting enough vitamin A causes unhealthy epithelial tissues due to squamous metaplasia (especially skin, liver, kidney, mouth and sinuses, etc.) Blunted choanal papillae are characteristic of hypovitaminosis A. Unhealthy tissues easily become infected by bacteria and fungus. Aspergillosis occurring at the tracheal bifurcation is common secondary to hypovitaminosis A changing the epithelium in that area.

Arthritis

In Clubb’s aging macaw article, decreased range of motion and joint stiffness was noted typically in macaws over 40 years of age. Perching on the same size perch for years, lack of exercise, and old age are associated with arthritis. Low or no perches near food, pain relievers and nutritional supplements that increase fluid in the joints may help. Chondroitin sulfate and glucosaminoglycans orally may help. Injectable of these products have been associated with adverse events, including death.

Cataracts

In Clubb’s aging macaw article, they noticed immature cataracts in at least one eye in all macaws over 35 years of age. The cataracts tended to stay immature for many years after onset and the birds were still visual, but when the change to mature cataract and blindness finally occurred, onset was rapid and involved phacoelastic uveitis. The eyes of even the large macaws are too small for the tools generally used in phacoemulsification, therefore a needle is used to evacuate the abnormal lens, but fraught with problems due to the firmness of the lens.

Obesity

Birds in captivity rarely get enough exercise and sometimes their diet is too high in fat (seed diets especially). The typical “perch potato” is at risk for liver disease, obesity, atherosclerosis and heart disease. Many organs don’t function normally in an obese animal. Often darkened, even black, feathers are seen in green and blue feathers over the wing of birds with liver disease due to abnormal refraction of light on abnormal feathers.

Cardiac Disease

An enlarged, poorly functioning heart, specifically dilated cardiomyopathy, is being diagnosed more frequently in older birds. Diagnosis is based on echocardiogram. All the typical cardiac drugs used in mammals are used in birds (furosemide, pimobendan, enalopril). There is a great chapter on Cardiology by Dr. Brenna Fitzgerald in Dr. Brian Speer’s Avian Medicine book.
Atherosclerosis

Diets high in animal fats cause hardening of the arteries. Hugh Beaufrere has many excellent proceedings and articles on this disease. One study of Quaker parrots fed a 1% cholesterol diet quickly (induced at 2 months, advanced at 4-6 months) developed atherosclerosis. 8 In the other study age, female gender, being of the genera Psittacus, Amazona or Nymphicus, and having certain diseases (reproductive, hepatic, myocardial fibrosis) were associated with atherosclerosis. 5 Antemortem diagnosis is difficult and treatment is also difficult. It is much easier to prevent this disease with a good diet and exercise. If a high cholesterol is found (I consider over 200 high just like in humans although many say higher), then a lipid profile is recommended (and a diet change!). Oftentimes owners don’t realize sources of cholesterol for their birds such as egg yolks, butter, meat, and cheese (commercial instant cheese grits is not good but a common culprit in the southern US!) Recommend A diet change to add sweet potatoes, remove sources of fat and cholesterol, and slowly over months, change to pelleted diet with about 20% fresh vegetables and some fresh fruits. Atherosclerosis in birds leads to progressively decreasing lumen diameter and not necessarily a plaque breaking off and causing a stroke, therefore isoxuprine has been found to be helpful in these cases.

Laying first egg in life and it is huge!

Anecdotally, I have seen many older female birds presenting egg bound because for the first time in their life, at an old age, they decide to lay their first egg. Usually these eggs are larger than normal and are difficult to lay. They present as an emergency situation because the huge egg is placing pressure on the kidneys causing shock. Abnormally high estradiol levels can sometimes be measured in these birds and may be due to cystic ovaries which stimulate the bird to start laying eggs. Polyostotic hyperostosis (POHO) may be present as well which is abnormal calcium deposition in multiple bones. Palpation of an egg and radiographs are most helpful for a diagnosis. Standard procedures for an egg bound bird must be performed understanding that this case is different since it is usually an egg that is too big to be laid. Therefore first ovocentesis, is performed to relieve the pressure on the kidneys. Then determination of the underlying cause (usually chronic exposure to long light cycles) of more aggressive therapies such as surgery are performed.. Further study is needed in this area. For prevention, some give a monthly leuprolide acetate injection or a desloerelin injection every 3-12 months. A salpingohysterectomy may ultimately be needed. Sometimes just adjusting the light cycle is enough.

Prevention Program

Good diet, exercise and sunshine. Regular check-ups and bloodwork. Teach clients how to examine their bird, their eating and drinking habits, and their droppings routinely for any changes and to see a veterinarian familiar with birds immediately if any changes are noted.

References


Table 1: Lifespan and Longevity of commonly kept parrots

<table>
<thead>
<tr>
<th>Species</th>
<th>average lifespan</th>
<th>approximate longevity record</th>
</tr>
</thead>
<tbody>
<tr>
<td>Budgie</td>
<td>5-7 years</td>
<td>18 years</td>
</tr>
<tr>
<td>Cockatiel</td>
<td>5-7 years</td>
<td>32 years</td>
</tr>
<tr>
<td>Lovebird</td>
<td>10 years</td>
<td>13-34 years</td>
</tr>
<tr>
<td>Conure</td>
<td>20 years</td>
<td>6-60 years</td>
</tr>
<tr>
<td>Amazon</td>
<td>15-50 years</td>
<td>22-66 years</td>
</tr>
<tr>
<td>African Grey</td>
<td>15-40 years</td>
<td>48 – 60 (92) years</td>
</tr>
<tr>
<td>Cockatoo</td>
<td>15-30 years</td>
<td>27 - 92 years</td>
</tr>
<tr>
<td>Macaw</td>
<td>15-30 years</td>
<td>32-63 years</td>
</tr>
<tr>
<td>Lory</td>
<td>7 years</td>
<td>17-30 years</td>
</tr>
</tbody>
</table>

Table 2. Most Common neoplasms based on tissue. 4

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Neoplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle</td>
<td>Leiomyosarcoma</td>
</tr>
<tr>
<td>Bone</td>
<td>Osteosarcoma</td>
</tr>
<tr>
<td>Kidney</td>
<td>Renal carcinoma</td>
</tr>
<tr>
<td>Testicular</td>
<td>Sertoli cell tumor/seminoma</td>
</tr>
<tr>
<td>Ovarian</td>
<td>Granulosa cell tumor</td>
</tr>
<tr>
<td>Proventricular</td>
<td>Proventricular carcinoma</td>
</tr>
<tr>
<td>Hepatic</td>
<td>Bile duct carcinoma</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Pituitary adenoma</td>
</tr>
<tr>
<td>Hemolymphatic</td>
<td>Lymphoid neoplasia</td>
</tr>
<tr>
<td>Fat</td>
<td>Lipoma (most common tumor overall in captive parrots)</td>
</tr>
</tbody>
</table>
Proper Medication Use in Backyard and Companion Poultry

Cheryl B. Greenacre, DVM, DABVP-Avian, DABVP-Exotic Companion Mammal Professor, College of Veterinary Medicine, Department of Small Animal Clinical Sciences, 2407 River Drive, C-247, University of Tennessee, Knoxville, TN, USA

Introduction

Backyard chickens and backyard poultry are now commonly being presented to veterinary practices for individualized and flock care. It is important to realize that even though someone may present you their dear pet chicken that they would never eat, you are responsible for knowing that it is still considered, and regulated, as a "food animal species" according to the United States Food and Drug Administration (FDA) and their regulations must be followed. Below are descriptions of the regulations and terminology encountered in regards to medication use in backyard poultry.

Backyard chickens are birds and all of our knowledge of avian medicine can be used in their care including general husbandry and care, handling, approach to medicine and surgery, and anatomy and physiology within the confines of federal regulations regarding medication use. Consulting an avian textbook, or exotic animal formulary for general information on birds is a good starting point.

Backyard poultry usually brings to mind chickens, although the term backyard poultry also includes turkeys, pheasants, ducks, geese, swans, quail, and other species. The diseases and care of backyard flocks is somewhat different than that of commercial broilers, breeders or layers and the following will pertain to backyard poultry.

Prohibited Drugs

The FDA prohibits the use of these drugs with no allowable extra-label drug use in any food producing animal species such as chickens and turkeys: chloramphenicol, clenbuterol, diethylstilbestrol (DES), fluoroquinolone class antibiotics, glycopeptides (all agents including vancomycin), medicated feeds, nitroimidazoles (all agents, including dimetridazole, ipronidazole, metronidazole, and others), nitrofurans (all agents including furazolidone, nitrofurazone, and others), adamantane and neuraminidase inhibitors (in all poultry including ducks); cephalosporin class of antibiotics except cephaparin (in all classes of chickens and turkeys); gentian violet (prohibited from use in food or feed of food producing animals), and indexed drugs (some exceptions for minor use species); extra-label drug use (ELDU) restrictions apply to all production classes of major food animal species (no ELDU for purpose of disease prevention, no ELDU that involves unapproved dose, treatment duration, frequency or administration route, and agent must be approved for that species and production class); ELDU restrictions DO NOT APPLY to minor-use food animal species.

All fluoroquinolines (like enrofloxacin) and cephalosporins are PROHIBITED drugs in poultry, which means you CANNOT give them to poultry. It does not matter the use of the poultry, so even pet poultry cannot be given prohibited drugs. You cannot get around this by having the owner sign that it is OK to give it – these are prohibited drugs and cannot be used. The reason? For the fluoroquinolones, chickens amplify the creation of antibiotic resistant Campylobacter sp. which would create a situation in which humans could get a severe, even life threatening diarrhea from one of these organisms and physicians would have very limited to no treatment options.

Extra-Label Drug Use (ELDU)

There are other drugs that fall under the rules of Extra-label drug use (ELDU). ELDU is any of the following situations (with examples) where a drug is not given exactly as written on the label: use in another species (trimethoprim sulphamethoxazole directly orally to a duck when not labeled for use in ducks), use for a different indication (erythromycin administered as per label instructions but for pododermatitis rather than chronic respiratory disease, use at a different dose or frequency (administering
spectinomycin for more than the first 3 days of life to a chicken), use via a different route of administration (erythromycin directly orally, not in food or drinking water).

Contact www.farad.org for specific information and instructions on ELDU in poultry. FARAD (Food Animal Residue Avoidance Databank) provides an on-line service where you can submit a proposed drug dose, frequency, route, concentration, and duration, and within 48 hours they will provide you with a suggested withdrawal for that drug.

**Labeled Drugs**

A labeled drug must be used exactly as is written on the label to be considered as labeled drug use. If the labeled drug is an antibiotic that is to be given in the food or water, then a Veterinary Feed Directive is needed. An example of a labeled drug is: Erythromycin (erythromycin thiocyanate, Gallimycin, Cross Vetpharm Group Ltd., NADA 010-092) - 185g/ton of feed, to aid in the prevention and reduction of lesions and in lowering severity of chronic respiratory disease; feed for 5 to 8 days; do not use in birds producing eggs for food purposes; withdraw 48 hours before slaughter.

**Veterinary Feed Directive (VFD)**

A Veterinary Feed Directive (VFD) is needed for any feed additive given to a food animal. There must be a valid Veterinary Client Patient Relationship (VCPR). The VFD must include the following written directions (order): veterinarian’s information with signature, client information, where animals are located, and type and # of animals, date issued, expiration date, indication for use, dose, withdrawal time, and specific verbiage ["Use of feed containing this VFD drug in a manner other than as directed on the labeling (extralabel use), is not permitted].

**Eggs**

There are NO dewormers available for use in a chicken that is laying eggs to be sold. Furthermore, there are very strict rules if you are selling eggs to the public, and the only thing that can be used for attempted deworming is oregano, kitchen/food grade diatomoaceous earth, or pumpkin.

**Salmonellosis concerns**

Educate owners regarding the risk of salmonellosis especially for children under 5 years, the elderly, or immunosuppressed persons since the risk in them is potentially life threatening. Chicks and ducklings can carry Salmonella in their droppings normally with no clinical signs of disease. Education materials are available from www.cdc.org. Our hospital asks people to sign that they have read an informational sheet on salmonellosis when they present with any poultry or reptile.

**References**

2. [http://www.extension.org/poultry](http://www.extension.org/poultry)
9. [http://edis.ifas.ufl.edu/pdffiles/PS/PS03900.PDF](http://edis.ifas.ufl.edu/pdffiles/PS/PS03900.PDF)
11. [http://www.michigan.gov/dnr/1,1607,7-153-10370_12150_12220-26362--,00.html](http://www.michigan.gov/dnr/1,1607,7-153-10370_12150_12220-26362--,00.html)
Canine Body Language: But What Do You Really Mean?
John Ciribassi, DVM, DACVB
Chicagoland Veterinary Behavior Consultants
Carol Stream, IL.

Why is it so critical to understand body postures in dogs? There are several reasons why this is an important topic with any discussion of dog behavior. By understanding how dogs communicate we can diminish the amount of miscommunication that occurs between people and dogs, it can help us better predict future behaviors in the dogs we interact with, understanding how dogs communicate can help reduce the incidence of dog bites, and it can increase the enjoyment people can have in their relationships with their dogs.

Behavior evolves just as body type evolves. Behavior can change over time as a dog learns what behaviors work in a given situation and which do not. As a result the successful behaviors will flourish while those that are less successful will tend to fade. This evolution can be seen in the individual animal by observing body posture since this is the principle means by which dogs communicate.

The eyes, ears, tail, mouth and overall posture can give us the best indications of what dogs are trying to communicate. These structures can convey relaxation, anxiety, tension, or confidence and by understanding the subtleties of their expressions, much ambiguity can be eliminated.

Because aggressive can greatly influence the bond and attachment we have with our pets, an understanding of the progression of aggressive responses can help in minimizing exacerbation of problem behaviors. The “Ladder of Aggression” serves to provide a good model of how aggressive behavior can develop from relatively benign “calming signals” to more overt aggressive displays culminating in snapping and biting.
Canine Housesoiling
Doo It Here... Doo It Now
John Ciribassi, DVM, DACVB
Chicagoland Veterinary Behavior Consultants
Carol Stream, Ill.

One of the most common reasons for pet owners to relinquish a new puppy is due to the inability to accomplish consistent housetraining. Most dog owners recognize that puppies do not come pre-trained and there will be some effort required to communicate the appropriate location for these most basic of needs. However, some new owners fail to realize just how difficult and time-consuming the process can be. Combine this with the difficulty to train some breeds, the presence of distractions which occupy time away from the training process (such as having young children or time-consuming occupations) and uncooperative weather (rain or snow) and it can be easy to see how this simple task can become quite complicated.

The basic goal of housetraining a puppy involves the development of **Surface Preferences**. This refers to the tendency for puppies to seek out preferred elimination surfaces (soil, grass, carpet, tile, etc) based on early experiences with these surfaces. So, if the puppy has consistent access to grass in your yard, and is rewarded for eliminating on this surface from an early age, it will preferentially seek this material long term. Conversely, if the pup is allowed the freedom to choose a surface on its own, such as your new oriental rug, this may end up becoming the preferred surface. It’s all about what feels good and what is familiar. Eliminating on surfaces that you find objectionable is not related to the puppy being vindictive, or un-trainable. It is just about a biological need to eliminate waste material (urine and stool) and the animal looking for the best place to accomplish this. It is the pet owner’s responsibility to help direct the pup to an area that is mutually acceptable for both. So, how do we accomplish this?

Confinement

It is nearly impossible to achieve housetraining by allowing a puppy to roam the house freely when alone or overnight. This will allow the pup the opportunity to try various surfaces in the home until it finds one to its liking. As a result, it is imperative that the pup be restricted to an area of the home where it is least likely that it will choose to eliminate. Most commonly the choice is to use a cage or crate. However, some owners find that they can successfully use a small area to accomplish the same goal. Bathrooms, utility rooms or gated off areas of other rooms can serve to provide deterrent for the puppy. The idea is that most puppies typically do not soil in the same area that it rests. Whatever method you choose, the idea is that the puppy cannot have room enough to have a “bedchamber” (a place to sleep) and a “bathroom” (a place to eliminate). If using a cage, the cage should large enough to allow the puppy to lie down, stand up and turn around without giving enough room for the aforementioned multi-room dwelling. Some owners will purchase a cage that is large enough to accommodate their particular breed as an adult and then use a partition to narrow the cage down then gradually increase the space as the dog grows. Some cages now have these partitions built in or you can use a board which is secured to the sides of the cage.

Whatever method you use, it is critical that the pup is not forced to remain confined for a period beyond what it is capable of in terms of the ability to retain urine or stool. The rule of thumb is that you take the puppy’s age in months, convert to hours and then add one more. So, for example, a 2 month old puppy can be expected to last 3 hours (2 months, plus one) before it will need to eliminate. This time period should be kept in mind when not only the puppy is alone but also overnight while sleeping. Consider setting an alarm clock overnight so that you get up to take your puppy outside with a time frame based on the time frame suggested above. While pups prefer not to lie in their own waste material, they will if they have no other choice (if the puppy is not allowed to eliminate in the proper time), thus making confinement less effective.

Supervision

If allowed to proceed without close supervision, puppies will choose whatever feels right to eliminate on. In order to prevent this natural instinct from dominating the process it is imperative for you to closely supervise your dog at all times. NEVER, EVER should the pup be out of sight of a responsible person when loose in the home. If strict supervision is not possible for whatever reason, place the pup in the crate or appropriate confined space. Otherwise use one of these methods to achieve good supervision:

- Close doors to keep the puppy in the same room as the person supervising
- Put up baby gates to restrict the puppy’s access to the house
- Use the Umbilical Cord Technique. This is where the puppy is tethered to the supervising person by attaching a leash from the puppy to the waist of the person. This technique ensures that the puppy stays within range of the pet owner’s view.
If, while monitoring the puppy, you notice signs that it needs to eliminate you should quickly lead the pup to the desired location. You may notice the puppy sniffing the ground, circling a spot, moving towards the door where you take it outside or just behaving in an anxious manner. If you happen to find urine or stool in the house, there is no need to use any form of punishment. Punishment, even verbal correction, can merely result in the puppy learning to eliminate more secretly in order to avoid being punished. In addition, punishment after the fact results in confusion and anxiety for the pup since the act has no relation to the elimination that occurred earlier. I also believe in the use of a rolled up newspaper at these times…used to hit *yourself* in the head for not watching your puppy closely enough!

**Scheduling**

Like anything in a puppy’s life, consistency is critical to get a change in a behavior. That goes for housetraining as well. The first point to keep in mind is that a puppy’s urge to eliminate is often tied to eating and drinking. In general, a puppy will have an increased urge to urinate after drinking and will show a similar urgency to defecate after eating. This does not mean that what they drink immediately is urinated out nor does it mean that their food immediately turns to stool. The body has a natural reflex to void accumulated waste material in response to consumption of liquids and food. You can use this to help with training. I typically recommend feeding puppies three times per day and to remove the food AND water bowls within 20-30 minutes. Once the pup is finished eating and drinking, immediately take it out to the desired elimination spot. In this way, the puppy can anticipate having three clear opportunities to eliminate.

It should be routine to also allow the puppy an opportunity to eliminate before leaving it home alone and when you return. In addition, this should be repeated when waking in the morning and before going to bed at night.

**Reinforcing Elimination**

The challenge is being patient. Reinforcement only works when you deliver the reward (praise and food work best) immediately after your pup eliminates. So, you have to wait with it so you can be present to deliver the reward. This usually means having the puppy on a leash so that you can keep it near you and in the spot you desire for the puppy to eliminate at. By having it consistently eliminating at this one spot, this location will acquire a sort of “bathroom” quality for the puppy and will increase the likelihood of it using this spot long term. Reward appropriate elimination behavior by using praise, food treats and access to free exercise (allowing your pup to play in the yard) immediately AFTER it eliminates. Finally, do not allow your puppy to have free access to the house if it has not eliminated in the proper place. If it does not eliminate within a few minutes in the desired location, return to the house with the puppy on leash or in its cage then repeat the process until it eliminates.

**Other Causes of Housetraining Failure**

There are other reasons for a new puppy to have accidents in the home besides difficulty with housetraining. This is why your first move when you have a puppy having elimination issues is to contact your veterinarian. Your vet will be able to rule out problems such as urinary tract infections, urinary bladder stones, congenital abnormalities, diabetes and other hormonal abnormalities.

In addition, a common behavioral cause of elimination problems in puppies is Separation Anxiety in which anxiety associated with being separated from the owner can cause urinary or stool accidents in the home when the puppy is alone. Other symptoms of Separation Anxiety include destructive behavior, vocalization (howling and barking) and excessive drooling. Again, these symptoms occur when the puppy is alone if associated with Separation Anxiety.

### Key Points

- See your veterinarian to rule out medical causes of elimination problems
- Choose an appropriate method of confinement for use when you cannot supervise your puppy
- Choose an appropriate method of supervising your puppy
- Relocate puppy to proper location if notice it beginning to eliminate
- Do not punish for elimination accidents
- Reward proper elimination immediately afterwards using praise, treats and play
Cats are not asocial animals nor are they small dogs
Cats are social animals and are individuals.

For free ranging cats
- Home Ranges (area traveled during normal activities)
- Time shares (Overlap)
- Social groups
- Stable
- Co-operative parenting
- Little is definitively known about hierarchy
- Not “pack”

Affiliative gestures
Behaviors that ↓ distance between cats
- Characterized by Allogrooming and allorubbing
- Proximity
- Food sharing
- Play

Agonistic behaviors
Behaviors that ↑ distance between animals
- Vocalization
- Piloerection
- Body language
- Facial expressions
- Facial Expressions

Types of feline aggression
- Redirected aggression
- Territorial aggression
- Fear-related aggression
- Play-related aggression
- Petting-induced (Status-related) aggression
- Aggression in the Veterinary Office

Treatment options
- Desensitization and counterconditioning (DS/CC)
- “House of Plenty”/Proper Play Activities
- Remote punishment
- +/- medications
- +/- NILIF
- Desensitization and Counterconditioning
- Gradual (re)introductions
- Start introductions through closed door
- Screen door/carriers/leashes
- Gradually increase time together and proximity
- Rotate cats
- Create a group scent
- Towels
- Feliway?

Counter conditioning and desensitization (CC/DS)
CC/DS is characterized by reintroduction over good things, use of delicious food, brushing and petting, use of catnip and play.
“House of Plenty”
Enough of everything for all cats (food, litter boxes, hiding/resting areas, toys, food based toys, videos, and bird feeders). Functions to decrease competition over resources.
  • Decreases competition

Remote punishers
Used at FIRST sign of aggression
  Squirt gun, “Spray Shield” (Citronella Spray), SSSCAT®, Compressed air, Double-sided tape, upside-down carpet runner, Scat Mat®, Snappy Trainer®

Redirected aggression
  • Signalment: Any gender, breed, age
  • Target: Person or other animal
  • Cat aggressively aroused & redirects aggression on closest target with arousal lasting hours to days

Treatment
  • DO NOT ATTEMPT TO INTERACT
  • Isolation when unsupervised
  • Prevent exposure to arousing stimulus
  • Outside cats (Scarecrow, close blinds)
  • Odors from other cats

Territorial aggression
Signalment: Any gender (MC most likely), Any breed but usually adults
Target: Other animals, people
  • Guarding specific location
  • New cats introduced to a stable group
  • Similar to dispersion in wild ancestor
  • Spacing is critical
  • Personal territory vs. claiming an area as is seen in dogs
  • Can be among littermates

Treatment
  • “House of Plenty” – Provide ample food and litter
  • DS/CC
  • Prevent exposure to outside cats

Fear-related aggression
Signalment: Any gender, breed, age
Target: Other cats, people, can occur between “friends”
  • See a fearful-looking cat (“Halloween Cat”)
  • Hissing, growling
  • Same house and avoids other cats when possible (w/ fear based intercat aggression)
  • Inter-male: testosterone dependent
  • Does not seek out target, but may or may not actively avoid the target
  • Can be classically conditioned

Treatment
  • Separate from target (other cat or people)
  • “House of Plenty”
  • DS/CC
  • Anxiolytics
  • Fluoxetine/Reconcile®

Play-related aggression
Signalment: Kittens & young cats, any gender, breed, and may be more common in orphans
Target: People or other cats
  • Threatening posture, stalking, ambushes
  • Usually no vocalization
• Becomes a problem when injurious
• Bites, Scratches, Falls
• Victims may become afraid of cat

**Treatment**
• Encourage object directed play
• Add a playmate
• Encourage independent play
• Redirect to more appropriate play
• Remote punishment

**Petting-induced aggression (“Don’t Pet Me” bites)**
Signalment: Any gender, breed, age
Target: People
• Owners may notice change in body posture
• Cat may solicit petting & tolerate some petting

*Many consider petting-induced aggression to be part of Status Related Aggression which is owner or cat directed and is stimulated by attempting to control or dictate some aspect of the cat’s behavior (petting, being picked up or moving the cat).*

**Treatment**
Stop petting at earliest sign
DS/CC
Remote punishment

**Aggression in veterinary office**
Signalment: Any gender, breed, age
• Can occur as kittens or following neutering surgery
• Posture is consistent with fear (hissing, piloerection, arched back, flight)
• Hissing, Growling, Swatting, Biting
• May develop over time into offensive display
• May be exacerbated by painful experience
• Associated with rushed veterinary visits
• Excessive Restraint
• Anxious or Socially Inadequate Cats

**Treatment**
• Alter handling techniques
• Remove from carrier by dumping or taking carrier apart
• Move slowly with handling
• Use towel to cover head
• Remove from kennel using slip lead and “scoop” technique

**Adding a new cat**
• Gradual Introductions
• Separate the newcomer
• Start introductions through closed door
• Screen door/carriers/leashes
• Gradually ↑ time and proximity
• Rotate cats
• Create a group scent
• Towels
• Feliway
• DS/CC

**Retrospective study on adopted cats**
Compared introducing cats:
• immediately
• after a week
• after a month
Equal success rate! Outcome seems to be dependent on resident cat
Medical Causes – LUTD

- Cystic Calculi
- Crystaluria
- Bacterial Infection
- Neoplasia
- Interstitial Cystitis
- Viral, Stress Induced, Idiopathic

Medical Causes – PU/PD

- Chronic Renal Failure
- Diabetes Mellitus
- Pyometra
- Estrus
- Hyperthyroid

Medical Causes – Fecal Abnormalities

- Inflammatory Bowel Disease
- Dietary Intolerance
- Gastrointestinal Parasitism
- Neurological or Locomotion Abnormalities

Minimum Database

- Urinalysis
- Urine Culture if indicated by U/A or blood work (ex. If Azotemic)
- CBC
- Chem. Profile
- Total T4

The Goal in Making a Behavioral Diagnosis is deciding Between: Marking Vs. Toileting

Minimum Behavioral Database

- Location of elimination and substrate - Marking typically occurs on vertical surfaces vs. horizontal
- Along walls, center of room, near windows or doors - Marking can often occur along perimeters
- Personal items vs. flooring - Horizontal marking can occur on personal items
- Type of elimination - Stool vs. urine (domestic cats do not mark with stool)
- Volume of urine - Marking commonly associated with small volumes
- Length of time problem has been occurring (Chronic vs. acute) - Can give an indication of prognosis
- Began as adult or kitten - Marking usually begins as kitten ages (after successfully using the litter box)
- Frequency of housesoiling incidents - increased frequency can be seen with marking behavior
- Number/Types of surfaces - marking commonly involves multiple surfaces
- Number of litter boxes and location – (Rule of Thumb: 1 box per cat + 1 and boxes should be separated in space to increase number of “core areas”
- Type of box - Covered vs. Uncovered
- Liners Used
- Size of box
• Litter types used (scented vs. unscented, clay vs. clumping)
• How long were the litters used
• Cat’s response to each litter
• Cats in household
  o Number of cats in household - Increased marking with increased # of cats
  o Correctly ID problem cat - Use of fluorescein and non-toxic crayons
  o Relationship between cats
• Access to outdoor animal activity - Territorial marking near viewing areas
• Changes in household (people and pets)
• Routine change in the home prior to onset of problem
• Previous treatments and results

Behavioral Causes

Toileting Issues:

• Substrate Preference - Cats will strive to reach proper substrate material.
• Substrate Aversion - Unacceptable litter type and can also occur secondary to LUTD or de-claw
• Location Preference - Cat finds an alternate location that it prefers in place of where litter box is located. Could be an area where cat feels safe or prefers secretive elimination.
• Location Aversion - Cat may have been frightened in the litter box area or had been attacked by another cat in the home while using the litter box.

Marking Behavior

• Vertical Marking (Spraying) - Typical Posture with tail raised, quivering and urine projected in a horizontal fashion
• Horizontal Marking - not as common. Characterized by depositing urine on personnel items
• Middling (Fecal Marking) not suspected to occur in domestic cats.

Characteristics of Marking

• Small Amounts of Urine
• Deposited on vertical surfaces (spraying) or on personal items (horizontal marking).
• Locations - No commonality of surface types (carpet, tile, wood, etc)
• Litter Use - Normal frequency of litter use. There is typically no issue with acceptance of litter. Remember, marking is for communication purposes.
• Elimination Posture - Spraying (tail raised and quivering)

Treatment Options

• Toileting Issues
  Place Litter Box in Cat’s Preferred Location - consider placing a litter box in this area in order to determine if the problem is location-related.
• Litter Trial - Offer several litter choices and record frequency of use of each.
• Confine with Preferred Litter - The goal is to increase the likelihood of the cat re-acclimating to the litter of choice
• Prevent Access to Soiled Areas
• Enzymatic Cleaners (Anti- Icky Poo, KOE)
• Litter Box Care
  o scoop daily
  o open litter boxes
  o no liners
  o clean with hot water only
  o 3-4” of unscented litter
• Appropriate Number of Litter Boxes - 1 box per cat plus 1 additional and distributed around the home.

It is important to gradually reintroduce cat to living area after proper interval of confinement. Slowly increase access to increased number of areas of the home. Be sure to provide additional litter boxes (with the preferred litter) in those areas to increase the likelihood of the cat using the box with the proper litter material.

**Treatment Options**

**Marking Behavior**

• Treat as for Toileting Issues - Evidence suggests that, even for marking behavior, proper litter management (#of boxes, dispersed throughout the home, proper litter cleaning protocol) can increase the tendency to utilize the litter box for elimination

• **Medication**
  - Clomipramine – 0.25-0.5 mg/kg bid
  - Fluoxetin – 0.5-1.0 mg/kg sid

• **More effective, safer and less recidivism rates as compared to Diazepam and Buspirone**

• **Treatment Options**
  - Feliway – synthetic Feline Facial Pheromone. Apply to marked areas and prominent spots in the home. Available as a spray or a plug-in diffuser.
  - Provide alternate marking opportunities
    - scratching posts or scratch boxes (in a proper location)
    - scratching combs (*Cat A Comb*)

• Manage relationship issues in the home - Address aggression issues between cats (indoor and outdoor) as well as relationship with human members of the household.
The focus of the discussion

- Which individual in a dyad (pair of animals) is considered to be dominant in the relationship?
- What criteria is used to make that determination (acquisition of resource vs. defense of resource)?
- Does aggression over the control of resources equate with dominance based aggression?

“Dominance: the assertion of one member of a group over another in acquiring access to a piece of food, a mate, a place to display, a sleeping site or any other requisite that adds to the genetic fitness of the dominant individual…” E.O. Wilson from Sociobiology: The New Synthesis Belknap Press of Harvard University Press, 1975. pg 257

Resource holding potential


“Dominance is a concept found in traditional ethology that pertains to an individual’s ability, generally under controlled conditions, to maintain or regulate access to some resource.” Karen Overall (“Clinical Behavioral Medicine for Small Animals” Mosby 1997. pg. 115

“Relative dominance is usually tested by giving two dogs access to one bone. The dog that gets possession is considered the higher-ranking dog.” Katherine Houpt (“Domestic Animal Behavior for Veterinarians and Animal Scientists” Iowa State U. Press 1982 pg 65)

“…a single bone was brought in, shown to the puppies, and laid between them……”

“The dominant dog shows a self-assured gait, a large, confident body posture, raised head, raised ears, large eyes and curled lips, all in different intensities and combinations depending upon the degree of dominance, superiority, or self-confidence.” Roger Abrantes (“Dog Language” Wakan Tanka Publishers 1997 pg. 93)

“…Once everyone knows his place, the alpha male need only move toward a lower-ranking male to have that individual hurry out of the way or otherwise signal submissiveness……” John Alcock (“Animal Behavior” Sinauer Associates, Inc. Publishers 2005 pg. 332)

Equal opportunity tests (EO tests)

“In equal opportunity tests (EO tests), both members of a pair had equal chance to seize the bone when it was tossed into the arena” Beach, Beuhler and Dunbar (“Competitive behavior in male, female, and pseudohermaphroditic female dogs.” J Comp Physiol Psychol. 1982 Dec;96(6):855-74)

Established possession tests (EP tests)

“During an EP test, the loser of the preceding EO test was given possession of the bone before the former winner was returned to the test arena” Beach, Beuhler and Dunbar (“Competitive behavior in male, female, and pseudohermaphroditic female dogs.” J Comp Physiol Psychol. 1982 Dec;96(6):855-74)

“…for a meaningful formal test of dominance, and to rule out differential motivation as a confounding factor contaminating the results, both animals must be motivated equally for the same resource.” Wendy van Kerkhove (“A Fresh Look at the Wolf-Pack Theory of Companion-Animal Dog Social Behavior” JOURNAL OF APPLIED ANIMAL WELFARE SCIENCE, 7(4), 279–285)

“A reasonable hypothesis is that the physical restrictions and limitations of captivity define environmental circumstances, engendering the formation of dominance hierarchies in wolves. Much the same might be said for dogs living together in a household.” Wendy van Kerkhove (“A Fresh Look at the Wolf-Pack Theory of Companion-Animal Dog Social Behavior” JOURNAL OF APPLIED ANIMAL WELFARE SCIENCE, 7(4), 279–285)

Possessive aggression

Aggressively guarding or maintaining control of a valued object (bone, chew item, stolen items or food, etc.). Guarding is considered to be normal behavior but can increase with opportunities for learning or can be exaggerated as a consequence of fear or defensive behavior/conflict.
“….food guarding was the most common circumstance for bites to familiar children (42%) and territory guarding for bites to unfamiliar children (53%). Behavioral screening of the 103 dogs examined revealed resource guarding (61%) and discipline measures (59%) as the most common stimuli for aggression.” Reisner IR, Shofer FS, Nance ML; “Behavioral assessment of child-directed canine aggression.” Department of Clinical Studies, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA 19104-6010, USA.

- Food Guarding
- Resource Guarding
- Possessive Aggression

These are all terms describing the use of aggressive behaviors to maintain possession of valued items. The aggression can be directed towards humans or other animals. Items can include anything which motivates an individual animal. In companion dogs these can be:

- Food
- Bones
- Rawhide
- Stolen Items

**Possessive aggression**

The sphere of guarding (critical distance in which a dog may react to approaching individuals) can increase over time to the point of the animal guarding a space that the valued object is contained within.

The behavior can be seen concurrently with Conflict Aggression and Territorial Aggression. Punishment or forced removal of items or food can increase the likelihood of the animal escalating aggressive displays to maintain control of items. This fear based response can result in the aggressive guarding of benign items that may not contain the same value as the original objects possessed by the dog.

The aggressive behaviors can be directed to both familiar and unfamiliar individuals when the appropriate circumstances exist to motivate the guarding response. Fear based body postures may be present initially but over time, as the dog learns the value of using aggression, body language may appear more confident.

**Other possible diagnoses**

- Disease Conditions - Is there a medical condition causing the dog to use aggression to prevent pain inducing activities
- Conflict Related Aggression - Does the aggression extend to other situations where the dog is using aggression to have an individual cease certain activities
- Dominance Related Aggression - Does the dog displace another individual from a valued resource?

**Medical examination**

Always begin with having the animal evaluated medically and appropriate testing should be performed. Conditions which cause pain or conditions which increase appetite may result in an increase in food acquisition and guarding behaviors.

**Treatment**

- Avoid known triggers (secure food, control access to toys and highly valued items, isolate during feeding and feed small meals)
- Consistent periods of play and exercise
- Avoid confrontation over retrieval of objects
- “Nothing in Life is Free” routine in order to increase consistency of interactions and put control of resources in owner’s hands
- Provide alternate items and activities, especially at high risk times, to substitute for the animal focusing on other valued items
- Trade for valued items that must be retrieved
- Utilize a leash and head collar to facilitate redirecting the dog’s behavior when needed

Once the level of tension has reduced between the dog and owner, if desired, the owner can work on teaching:

- “Drop It” and “Leave It” commands for managing object possession
- Desensitization to the presence of the owner around the food bowl in order to manage food guarding behaviors

Possessive Aggression is typically managed and controlled and not cured. As with most forms of aggression, the only guarantee can be made with a recommendation of euthanasia. Short of this option, the owner is always accepting some degree of risk.
Sibling Rivalry: When Roommates Come to Blows
John Ciribassi, DVM, DACVB
Chicagoland Veterinary Behavior Consultants

Risk factors

Household instability

- One or more dogs in household achieving social maturity (1-3 years)
- New pet or person added to home
- Illness in one or more pets in the home
- Pet returning from an absence
- History of one or more dogs in the home of having poor early socialization with dogs (genetics, early health issues, inadequate exposure)
- Anxiety related condition(s) in one or more dogs in the home (Separation Anxiety, Noise Phobia, CCD, General Anxiety, Fear Based Aggression, Conflict Aggression)
- Medical condition causing irritability (Otitis, Dermatitis, etc.)
- Deprived environment (fewer than ideal resource load; food, resting areas, owner interaction
- Same-sex pairs in the home.
- Most commonly females. Particularly in spayed females
- Young dogs being added to a household or dogs rehomed to a household are more likely to initiate fights

Typical history

- Often between two specific dogs even in a multiple dog household (>2 dogs in the home)
- Various stimuli
- Excitement in the home (greetings, passing through narrow openings, territorial barking, laughter or arguing in the home or running through the home)
- Resources (food, owner attention, toys, space) – recognize the relative value of the items to each individual dog in the household (Resource Guarding Potential)

Hierarchy conflicts – behaviorally appropriate dogs are similarly motivated to maintain or acquire access to similar resources.

- Competition can be over one specific person in the home
- Owners undermine appropriate social structure between the dogs
- Aggressor may persist in attacks even if victim offers proper deferent signaling

Differential diagnosis

- Medical conditions
- Dominance Hierarchy – Resource Related
- Anxiety Related
- Redirected aggression
- Play Related Aggression

Differential diagnosis

Commonly seen with newly introduced housemates

- Fear Based Aggression
- Territorial Aggression

Typically increased social contact between housemates diminishes the likelihood of these interactions. However, socially inept dogs may show a reduced inability to adapt to prolonged exposure and continue to display behaviors more common with contact between unfamiliar dogs.

Medical conditions

Any condition which causes increased pain or irritability can increase the likelihood of an aggressive response between dogs

- Otitis Externa
- Osteoarthritis
- Dermatitis
Dominance hierarchy – Resource related
If there is equal motivation between dogs in a household over the acquisition or holding of a resource we can see an escalation of aggression between those individuals. Commonly a factor between intact males in the same household.

Equal opportunity and established possession testing

Anxiety related
- Behaviorally inappropriate dogs
- Do not adequately recognize normal signaling in other dogs (deference cues such as lip licking, yawning, turning away, moving away or exposure of underbelly, for example)
- Excessively reactive. More likely to target another dog in the home in situations characterized by high arousal (exposure to excitement stimuli)
- Can have poorly inhibited bites

It is critical to recognize, in these instances of aggression between dogs in the same household in which the attacker is socially inappropriate, the victim’s quality of life may suffer greatly. These dogs are doing everything they know how to diffuse the aggression and communicate deference or submission to the attacker but the attacks persist.

Stress escalates when the individual has minimal control over the outcome of a situation. This chronic stress results in continued activation of the Hypothalamic Pituitary Axis and thus prolonged cortisol exposure for the victim.

Redirected aggression
- The victim of the attack is the secondary target. The attacker cannot access the primary focus (another dog passing the home, for example, and then targets the other dog in the home which is more available).
- Can result in extreme fear in the victim, who can respond in a likewise aggressive manner thus escalating or maintaining the aggressive relationship between the dogs

Play based aggression
- Typically occurs between younger dogs
- Bites are usually inhibited so that significant injury does not occur
- Frequent reversal of roles during fights such that each dog will take turns showing dominant displays (mounting or biting over the dorsal aspect of the neck, for example)
- If excessive, can escalate to more serious encounters necessitating the owners to intervene

Fear based aggression
- Fearful animals may elect to utilize aggressive responses in order to manage or cope with stressful situations involving new dogs in a household
- May be initiated by the newcomer or the resident dog
- Depending on the age and experience of the fearful animal you may or may not see typical fearful signs (tail tucked, cowering, ears down and back, etc.) Dogs with a longer history of fear based aggression may have abandoned these postural strategies due to perceived ineffectiveness and now depend on aggression as a better coping response.

Territorial aggression
- Resident dog responds to newcomer by preventing access to valuable space.
- May be the home itself, certain areas of the home, the yard or valued sleeping areas.

Prognosis
- The likelihood of a successful outcome is good if both dogs are behaviorally appropriate, if resources can be identified, and the resources can be adequately managed.
- Prognosis is poor if one or both dogs are behaviorally inappropriate (anxiety or fear is a component of the behavior), particularly if response to medication is inadequate
- Prognosis is also poor if aggression occurs immediately whenever dogs come into sight of one another….

Diagnostic evaluation
- Physical Exam
- Neurologic Exam
- CBC, Chemistry Profile and Thyroid Screen
- Further labs as indicated by basic work up
Questions

- Household composition
- When aggression began
- Frequency
- How are resources managed between the dogs
- How do dogs interact outside of aggressive episodes
- How do fights occur. Give examples from most recent to previous fights as well as description of earliest fights.
- How do the fights resolve
- Are there injuries

The most important question is which dog, if any, is acting appropriately in the interactions. In this way, the attention can be centered on the correct dog. That may be changing the response of the dog acting inappropriately in the relationship or, if both dogs are appropriate, managing the resources in the household.

Treatment

- Manage resources (food, toys and attention) – “dogs are not best thought of as a pack in a home environment. They are best thought of as roommates who need to learn to share”
- Identify all situations which trigger aggression and avoid these triggers or separate the dogs at these times
- Safety
- Provide owners with means to break up fights (head collars with drag leashes, blankets, air horns, water, instruct in removing dog by pulling on rear legs)
- Isolate pets when unsupervised
- Address triggers (food, toys, resting areas, access to owners)
- Feed dogs separately
- Do not leave toys out but apportion them as needed
- Deny access to elevated surfaces and have dogs resting remotely away from owners (on mats or dog beds, for example)
- Basket Muzzles

These can be used whenever there is a higher likelihood of aggression between the dogs where the owners are not as likely to be able to quickly intervene. Can result in increased comfort for the owner in knowing the dogs are at least safe from severe injury.

- Separation with gates or tethers
- Used when dogs cannot be closely supervised
- NILIF or "SIT" protocol
- Goal here is to increase the dog’s attention to the owner for direction
- Regular periods of basic training (clicker training)

By increasing the dog’s level of responsiveness it allows the owner better ability to direct their dog’s behavior and therefore having them show less focus on each other. A good recall is important in that it gives the owner the ability to call the dogs away in potentially problematic situations.

- Have owners ignore BOTH dogs if owner attention is causing hierarchy issues between the dogs

The goal here is to reduce the value of the owner as a resource for either dog. Increased owner attention to either dog (as opposed to trying to figure out which dog is higher ranking with respect to this particular resource) can escalate the owner’s value and thus increase conflict and also elevate emotionality in the home (problematic for the behaviorally inappropriate dog).

- Support higher ranking dog?

There are several problems with this approach

- Difficulty for owners to identify accurately
- Owners may be reluctant to demote an older, favored dog
- Dogs who are behaviorally inappropriate may not be signaling correctly and thus owners red these dogs incorrectly thus favoring a dog who is showing aggression at the wrong times and putting the victim in a difficult situation
- The aggression in the household may not involve hierarchy at all

Response substitution (operant counter conditioning)

- This involves interrupting the dog and then redirecting to more appropriate sets of behaviors (that the owners have been rehearsing with the dog on a regular basis in non-distracting situations) and reinforcing those behaviors.
- Does not reinforce the aggression since the dog is being relocated and not reinforced until it complies with a request to perform an alternate behavior. We are conditioning a behavior that is counter to the problem behavior.
Counter conditioning and desensitization to graded triggers such as sounds in the environment

If there are triggers which can be identified as causes of the aggression, and the intensity of these triggers can be adjusted, the owners can gradually expose the dog(s) to the trigger at slowly increasing levels (desensitization) while asking the dog to perform more appropriate competing behaviors (counter conditioning).

Example: Door bell triggering excessive greetings and resulting aggression.

Reintroduction

In some cases dogs have to be separated for an extended time while owners work on getting consistent responses from each dog separately and each dog learns it will receive positive rewards for attending to the owner. This would be needed if the dog’s cannot be in each other’s company without immediately reacting.

Once each dog is responding well separately from each other, then they can be reintroduced on walks. First at a comfortable distance while going through training individually then gradually decreasing the distance between them as they adjust.

Treatment

If treatment proves to be unsuccessful, other options include:

- Rehoming
- Permanent Separation of the dogs
- Euthanasia (particularly if one of the dogs is behaviorally inappropriate)

Should dogs “fight it out”?

In one study, 42% of dog fights did not require intervention to break them up.

However, if there is a history of injury to either of the dogs involved in fighting, it would be inappropriate to allow them to continue to fight without intervening. The injuries demonstrate that the dogs have been unable to arrive at a mutually beneficial agreement over partitioning or resources. If the fights are motivated by fear or anxiety in behaviorally inappropriate dogs, they will be incapable of regulating the level of violence and injuries are likely.

In these cases, owners need to learn how to safely break up fights

Options in breaking up dog fights

- Wheelbarrow the attacker by picking up the rear legs and lifting while moving back and to the side
- Compressed air or citronella
- Water
- Sudden noises such as with pot lids
- Board to wedge between the dogs
- Blankets or cushions
- Leashes attached to both dogs (with or without a head halter)

Drug therapy

- ONLY if one or both dogs are abnormal in terms of fear/anxiety
- SSRI (0.5-2.0 mg/kg SID)
- Fluoxetine
- Sertraline
- Paroxetine
- Selegiline if Canine Cognitive Dysfunction (1 mg/kg SID)
- As Needed Options
- Clonidine (0.01-0.05 mg/kg 1-2 hours before needed or up to tid)
- Trazodone (3-5 mg/kg 1 hour before needed up to tid)
- Benzodiazepines (not indicated in fear based aggression due to the possibility of disinhibition).

Pre-treatment blood work

CBC/Chemistry profile/Thyroid profile

Post-treatment blood work (4-8 weeks post onset of therapy)

CBC/Chemistry profile

- Pheromones (Adaptil)
- Neutraceuticals such as Anxitane

Surgery (if hierarchy related)

- Castration
- OHE? No indication that OHE is successful at reducing aggression between females in the same household.
Client education
- Discuss canine body posturing and communication methods
- Regular communication with client to enable adjustment of treatment plan

Prevention
- Add dogs to home of different genders and ages
- Regulate access to resources
- Castration to help prevent intermale aggression
- Proper socialization
- Puppies stay with litter until about 8 weeks of age
- Socialization classes between 8-14 weeks of age and reward based obedience class at around 4-6 months of age
Canine Separation Anxiety: A common behavior problem and welfare concern
Barbara L. Sherman, MS, PhD, DVM, DACVB, DACAW
College of Veterinary Medicine, North Carolina State University

Learning Objectives: At the conclusion of this lecture, attendees will be able to:
1. Recognize the clinical signs of canine separation anxiety
2. Design a multifocal treatment plan for management of canine separation anxiety.

Introduction:
Canine separation anxiety is a common behavioral disorder, with signs reported in up to 17% of dogs that obtain regular veterinary care. Separation anxiety is thought to be an extreme consequence of the social nature of dogs and their normal attachment to specific individuals. Lack of early socialized may predispose animals to separation anxiety as dogs from shelters are over-represented. Signs of canine separation anxiety occur when an affected dog is left alone or separated from its “significant person.”

Separation anxiety is expressed by distress vocalization, pacing and orienting toward the door of owner egress, destructiveness, housesoiling (in an otherwise well housetrained dog), hypersalivating, and other signs. Dogs may exhibit one or more of these signs, and may live with other dogs who are not affected. The dog’s behavior when left alone is usually in marked contrast to its behavior in the presence of the owner, when it may never exhibit these misbehaviors. The owner may be unaware that the dog’s behavior is due to an anxiety disorder, and may attribute the behavior to spite. Diagnosis is made on the basis of the behavioral history and the exclusion of differential diagnoses, which may be medical or behavioral. To make a definitive diagnosis, the behavioral signs must be restricted to times when the dog is left alone or separated from its “person.” A web cam may confirm the diagnosis, especially in a multi-dog household, and may be useful in assessing the effectiveness of treatment. A video recording of the dog is useful in helping the client appreciate the severe distress that the affected dog is experiencing.

--Clinical signs: Destructiveness
Destructiveness is a common presenting sign that may be very costly to repair. Often the dog’s destructiveness is focused around the door that the owner has exited, but may include other sites of egress, including windows and other doors. The moulding around doors or windows may be chewed and clawed, and the door itself may be extensively damaged. Digging may occur at the base of doors, so that carpeting, or other floor covering, is damaged. Articles of clothing, including shoes worn by the owner may be moved to a new location and chewed. Furniture may be chewed and trash cans overturned. The owner may return home to find the appearance of chaos and destruction inside the house where the dog has been left. Dogs left outside may direct their destructiveness toward the outside of doors and windows as they attempt to get inside the house, which is the route of departure of the owner. Dogs left in crates when the owner leaves may exhibit destructiveness by tearing up bedding or newspapers left in the crate or chewing on the bars of the crate or attempting to dig out of the crate, causing self trauma. Differential diagnoses include general destructiveness, thunderstorm phobia, and territorial behavior. General destructiveness is especially common in young dogs, who may be particularly destructive when unsupervised by the owner. Dogs fearful of noises such a thunderstorms may exhibit destructive behavior when frightened and the owner is gone. Territorial behavior may be manifest as chewing around the front door and adjacent windows, in an attempt to deter postal workers or delivery persons.

--Clinical signs: Housesoiling
Fecal or urinary elimination in the house is a common presenting sign of canine separation anxiety, related to autonomic arousal. If the dog is crated, the dog may have soiled itself with elimination products. Differential diagnoses include inadequate housetraining, male urine marking, noise phobia, gastrointestinal or urinary tract disorders, or seizures. The behavioral history should be investigated to verify that the dog never eliminates in the house unless the owner is absent.

--Clinical signs: Vocalization
Dogs may vocalize within 10 minutes of the owners’ departure and may continue intermittently or unabated, resulting in complaints from neighbors. The vocalizations include plaintive “distress” whining, howling, or barking.
--Clinical signs: Hypersalivation
Profuse hypersalivation may occur as a clinical sign of separation anxiety. In these cases, the owner may return to find the dog's face, chest, and forelimbs soaked with saliva. Dogs left in crates may be standing in a puddle of saliva at the end of the day. The owner often reports excessive water consumption when he or she returns, as the dogs compensate for transient dehydration. When hypersalivation occurs only in the owner's absence, it may be considered pathognomonic for canine separation anxiety.

--Clinical signs: Other behaviors
Dogs with separation anxiety may show altered patterns of motor activity for several hours after the owners' departure, in contrast to normal dogs, including ritualized pacing or circling. Some dogs may exhibit decreased motor activity, often described by the owners from cam recordings as "depressed" or "catatonic". Such dogs may seek a special location, such as a closet or under a bed, and remain immobile until the owner's return. Some dogs tremble when left alone; others lick the floor or object continuously or may self traumatize themselves.

In many cases, dogs with separation anxiety have highly affiliative relationships with their owners. The following traits are frequently observed: The dog remains close to the owner, following him or her about the house, the dog becomes distressed with increasing distance of the owner, such as when retrieving the mail from the mailbox or taking out the trash. The dog may show signs of agitation or depression as the owner prepares to leave. The dog may be anorexic when the owner is absent and may eat only in the presence of the owner.

Environmental and Behavioral Treatment:
--Treatment consists of environmental control, behavior modification, and pharmacotherapy. Best long-term treatment success is obtained when all of these modalities are used.
--Do not punish the dog retrospectively. Punishment will increase the dog's anxiety and will worsen separation anxiety.
--If possible, do not leave the dog alone until medical therapy has been initiated. It may be possible that the dog can be taken with the owner, boarded for the day, or left with a neighbor.
--About 30 minutes before departure, make any household changes, such as turning off lights, turning on the radio, at least 30 minutes prior to departure to avoid these becoming signals. On departure, be low key.
--Leave the dog in a safe place when it cannot harm itself, with a special food filled toy, such as a rubber "Kong" filled with favored food items, such as cheese, crackers, p-nut butter, etc. When the owner returns,
--Practice independence training when the owner is at home, rewarding the dog for lying on a comfortable dog bed on the floor while the owner sits in a chair, without physical contact. With success, over weeks, the dog should be taught to lie down while the owner goes to another room for gradually longer periods of time, then returns to reward the dog. These exercises should not be linked to departures.

Behavioral Medication
--Appropriate antianxiety medication is critical to successful treatment. A daily "baseline" drug (Table 1), such as fluoxetine or clomipramine should he started immediately at a starting dose, then increased within a week. In addition, a situational agent (Table 1) should be given 1-2 hours prior to departure, regardless of the length of departure.
--Recent studies suggest that the GIT plays a role in anxiety. The addition of a "calming probiotic supplement" (Calming Care, Purina) shows efficacy in treatment of canine anxiety and may be helpful in cases of separation anxiety. It may be given with any medication.
### Table 1: Drugs commonly used to treat separation anxiety

<table>
<thead>
<tr>
<th>Generic Name of Drug</th>
<th>Proprietary Name</th>
<th>Baseline, Given Daily</th>
<th>Situational, Given Prior to Departure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluoxetine</td>
<td>Reconcile, Prozac</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Clomipramine</td>
<td>Clomicalm</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Sertraline</td>
<td>Zoloft</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Bifidobacterium BL999**</td>
<td>Calming Care</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Trazodone</td>
<td>Desyrel</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>BZ Alprazolam</td>
<td>Xanax</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>BZ Lorazepam</td>
<td>Ativan</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>BZ Clonazepam</td>
<td>Klonopin</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Clonidine</td>
<td>Vasopres</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Dexametomidine gel</td>
<td>Sileo</td>
<td>x</td>
<td></td>
</tr>
</tbody>
</table>

** GIT supplement, not a drug.

Other Resources: (access via [https://scholar.google.com/](https://scholar.google.com/))

--Ogata N. Separation Anxiety in dogs: What progress has been made in our understanding of the most common behavioral problems in dogs in dogs? Journal of Veterinary Behavior: Clinical Applications and Research 2016;16:28-35.
Oh, no a behavior problem! What do I do now?
Barbara L. Sherman, PhD, DVM, DACVB, DACAW
North Carolina State University College of Veterinary Medicine

Learning Objectives: At the conclusion of this lesson, attendees will be able to:
--Explain steps in approaching behavior problems in general SA practice
--Outline the components of a SA practice model that incorporates prevention and treatment of common behavior problems
--Consult with veterinary behavior specialists to solve common behavior problems

Introduction
Behavior problems are the most common cause of death of pets in the United States. This is because a pet with an unresolved behavior problem is likely to be relinquished to an animal shelter where it may be euthanized. In one study, behavior problems were cited as the cause for relinquishment in 47% of dogs and 32% of cats (Salmon et al, 1998). According to the National Council on Pet Population Study and Policy, approximately 56% of dogs entering member shelters were euthanized. By this unfortunate process, it is estimated that approximately 6 million dogs and cats are euthanized each year, many on the basis of their misbehavior. Preventing and resolving behavior problems before they become life-threatening is a way for small animal practices to help animals’ live long healthy lives.

Studies show that 45-90% of owners report behavior problems with their pets Although in an American Animal Hospital Association (AAHA) study, 78% of pet owners consider their veterinarian to be the first person to contact when seeking help for behavior problem, many veterinarians feel unprepared. Some may even deliberately avoid asking routine questions about a pet's behavior to circumvent such a discussion. One reason is that veterinarians are often untrained in the field of behavior and lack the expertise to treat problems in this area. Another reason is that, in the past, there have been few standardized treatments for common behavior problems, making the practice time consuming and the fee structure challenging. Further, some veterinarians have felt that treating behavior problems is outside their domain as practitioners. These findings have been ameliorated by the increased availability of research, continuing education, veterinary information web sites (such as VIN), and books on clinical behavior written by board-certified veterinary behaviorists.

Clinical approach
The standard protocol for addressing behavior problems is to first rule out medical etiologies. This is accomplished with a behavioral and medical history, physical examination, neurological examination, and appropriate laboratory tests and procedures. The next goal is to conduct a behavioral evaluation in order to establish a behavioral diagnosis (see Table 1). Finally, a treatment program is initiated. Treatment modalities include environmental management, behavior modification techniques, and pharmacotherapy. In some cases, surgical procedures such as castration can be part of the management plan.

Case Scenario:
On a busy clinic day, you conclude a routine uneventful annual appointment with a healthy 5 yo terrier mix named “Jack.” As the client, Ms. Jones, turns toward the door, she asks: “Doctor, I am concerned that Jack has recently started to bark at children who come to play after school with my 8 year old daughter, Heather. What should I do?” Your first thought: “Oh no! A behavior problem! What do I do now?”

Your approach:
--Medical evaluation: “Ms. Jones, I’m so glad you mentioned this problem so we can address it. Although Jack looks good today, I’d like to be certain there are no underlying problems that might explain his behavior. Can you leave him with us until later this afternoon so we might run some diagnostic blood tests?” Plan: Drop off CBC, Chem, UA to more fully evaluate the dog’s health.

--Immediate safety. “Ms. Jones, until we review the situation, please keep Jack on a leash when children visit and tell the kits that Jack is in training and can’t be petted and cannot play with them. Give Jack treats for sitting and staying next to you on leash.
--Plan: “Our technician, Sue, will give you a handout on this approach, and a behavior history form (or she can e-mail it to you) and she’ll help you set up a behavior consultation (extended appointment).” Be sure Sue will follow up and get history form in advance so you can review and develop a plan.

History form:

Download a canine and feline history form (www.ncsubehavior.com) and customize them for your practice and use.

--Prepare your hospital for behavior cases:

--Take the Fear Free course (https://fearfreepets.com/) or the Low Stress Handling Course (https://lowstresshandling.com/).

--Identify the RVT in your practice most interested in behavior (maybe all technicians!) and enroll her/him in the Society of Veterinary Behavior Technicians (http://svbt.org).

--Identify trainers in your area who are members of at least one of the following organizations and ask her/him to come in and meet with you to work with you on behavior cases.
  --Pet Professional Guild https://www.petprofessionalguild.com/
  --CCPDT: http://www ccpdt.org/certification/dog-trainer-certification/
  --Karen Pryor Academy Trainer: https://karenpryoracademy.com/certification/choose-kpa-certification/

DO NOT simply refer difficult cases to a trainer.

-- Purchase resources on Behavior for your library:
  BOOK 3: Horwitz D, Ciribassi J, Dale, S (editors). Decoding Your dog. Mariner books, 2015. Written by the diplomats of the ACVB. Become familiar with this book and recommend it to clients. You might wish to sell it in your practice!

--Find an ACVB board-certified veterinary behaviorist who will consult with you (for a fee). Find the closest person to you: https://www dacvb.org/ or call NC State Behavioral Medicine Service (www.ncsubehavior.com).

--When Ms. Jones and Jack some in for their extended appointment: Be prepared by reading the history form and determining what handouts will be helpful. We will discuss how to address this case during the lecture.
Overview of common behavioral drugs: A guide to usage, class, contraindications, and side effects
Barbara L. Sherman, MS, PhD, DVM, DACVB, DACAW
North Carolina State University College of Veterinary Medicine

Learning Objectives: At the end of this lesson, participants will be able to:
--Identify major indications for the use of behavioral drugs
--Recognize 3 classes of behavioral drugs and their usages
--Recognize common contraindications for behavioral drugs
--Recognize common side effects of behavioral drugs and how to manage them

Introduction:
In veterinary medicine, behavioral drugs are used to manage the behavior of conscious animals to reduce fear and anxiety, to manage pathologic states, and to improve welfare. Their use is now an important aspect of clinical veterinary medicine, particularly for pets, companion animals in confinement, such as shelter animals, and zoo animals. Not discussed here are the behavioral effects of drugs, such as analgesics, antihistamines, or anticonvulsants, primarily used in conscious animals for medical indications. Discussed here are behavioral drugs that are particularly useful to reduce fear and anxiety without excessive sedation, and for which clinical data have been published. This introductory discussion will focus on 3 drug classes (SSRIs, SNRIs, α2-adrenergic agonists) and a representative drug within each classes. For reference, 6 drug classes are shown in Table 1. The greatest efficacy of behavioral drugs is achieved when used in combination with environmental improvements and positive behavior modification.

Usage:
Behavior modifying drugs have important application to the treatment of behavior problems encountered by the veterinary practitioner. A number of studies confirm that behavioral drugs can increase the number of animals that response to behavioral therapy and can increase the latency to response. Behavioral drugs are most effective when used in combination with a positive behavior modification program. In clinical trials, use of behavior modifying drugs in combination with behavior modification instructions achieved greater success than behavior modifying drugs alone. Behavior modification instructions may be given to clients in the form of a standard handout or customized program. Refer to the list of useful veterinary behavior reference books at the end of these notes for examples of behavior modification handouts and instructions.

Behavioral drugs may have general or specific effects. General effects include a decrease in affective behaviors such as arousal, reactivity, or impulsiveness that can drive unacceptable behavior. Such nonspecific effects can sufficiently alter the problematic interactions between the pet and owner to allow behavior modification programs to be more effective. Behavioral drugs may also be used to treat specific diagnoses. For example, anxiolytic drugs are used to treat anxieties and fears, such as separation anxiety, and noise aversion, including fear of thunderstorms or fireworks. Other drugs may also be used to improve the functionality of pets suffering from organic states that affect behavior, such as compulsive behaviors or cognitive dysfunction.

Drug Classes:
Commonly used drug classes of behavioral drugs are shown in Table 1. Emphasized during the lecture, with examples, will be selective serotonin reuptake inhibitors (SSRIs), Serotonin norepinephrine reuptake inhibitors (SNRIs), and alpha-2 agonists. Further details will be provided in behavior lectures that follow this introduction.

Contraindications:
In general, behavioral drugs are well tolerated when used in combination with other routine medications, such as heartworm and flea prophylaxis, antibiotics and anesthetics. In all cases, the health of the animal should be assessed before prescribed behavioral drugs. Most behavioral drugs are metabolized through the liver and excreted by the kidneys, so optimal function of these organs are critical. Some drugs, such as alpha-2 agonists, have cardiovascular effects, which are generally well tolerated in cardiovascularly-healthy animals. Often, behavioral drugs, such as fluoxetine and trazodone will be combined for optimal
effectiveness. However, combining some behavioral drugs may be contraindicated, primarily due to the risk of “serotonin syndrome.” This risk will be discussed in the lecture. To minimize side effects, often drugs are started at relatively low doses initially, then increased as drug tolerance is achieved.

Side Effects:
Muted affect and mild ataxia may occur with behavioral drugs. Sleepiness and decreased appetite with initial doses is a common transient side effect. These usually usually resolve within 7-10 days. Side effects may be avoided by starting at a low dose for 1 week, then increasing.

Medication Management:
Successful treatment of behavioral problems requires a schedule of follow-ups with the client, in person or by telephone. If problematic side effects or inefficacy occur, the dose can be adjusted. If a medication is not effective after dose adjustment, selection of an agent from another drug class is recommended. If a drug in one class is not sufficiently effective, and appropriate dose adjustments have been made, that drug should be discontinued and a drug from another drug class should be prescribed, according to the "wash-out" instructions for the specific agents. In some cases, more than one drug is used concurrently, although this practice requires knowledge of the available drugs, their general behavioral actions, side-effects (including potential drug interactions), and previously reported therapeautic effects. Understanding the neurochemistry of each drug helps to distinguish between agents likely to differ in their effects, so that if one drug is ineffective or poorly tolerated an alternative can be selected with different mechanisms of actions, side effect profile or behavioral action.

Conclusions:
Behavioral pharmacotherapy is an important component of veterinary behavior therapy. Psychoactive drugs are most effective when used as part of a comprehensive program involving behavior modification and environmental management. In general, behavioral drugs are well tolerated, although knowledge of the action and potential side effects of these agents is necessary to guide the practitioner in their use.

Table 1 Commonly used behavioral drugs

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Drug Name</th>
<th>Usage</th>
<th>Contraindications</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSRI</td>
<td>Fluoxetine (Reconcile®)</td>
<td>Fear &amp; anxiety, feline urine marking</td>
<td>Use of MAOI</td>
<td>Sleepiness, ↓ appetite</td>
</tr>
<tr>
<td>SARI</td>
<td>Trazodone** (Deprenyl®)</td>
<td>Arousal, Excitation</td>
<td>Use of MAOI</td>
<td>Sleepiness, agitation</td>
</tr>
<tr>
<td>TCA</td>
<td>Clomipramine (Clomicalm®)</td>
<td>Fear &amp; anxiety, feline urine marking</td>
<td>Use of MAOI</td>
<td>Anticholinergic signs</td>
</tr>
<tr>
<td>BZ</td>
<td>Alprazolam** (Xanax®)</td>
<td>Fear &amp; anxiety</td>
<td>Human abuse potential</td>
<td>Hyperactivity, Hepatic necrosis (cats only)</td>
</tr>
<tr>
<td>BZ</td>
<td>Clonazapam (Klonopin©)</td>
<td>Fear &amp; anxiety</td>
<td>Human abuse potential</td>
<td>Hyperactivity</td>
</tr>
<tr>
<td>α2-adrenergic receptor agonist</td>
<td>Dexmedetomidine** (Sileo®)</td>
<td>Fear &amp; anxiety; noise phobia</td>
<td>Oral gel administration</td>
<td>Cardiac, respir, kidney, liver dz</td>
</tr>
<tr>
<td>MAO I</td>
<td>Selegiline (Anipryl®)</td>
<td>Cognitive Dysfunction</td>
<td>Concurrent use of SSRI, SNRI, TCA</td>
<td>Agitation</td>
</tr>
</tbody>
</table>

** Situational use

For drug doses: [https://www.merckvetmanual.com/behavior/](https://www.merckvetmanual.com/behavior/)
Additional Resources (access via https://scholar.google.com/)
Pharmacologic treatment of situational fear and anxiety in cats: Travel, vet visits, and visitors to the home
Barbara L. Sherman, MS, PhD, DVM, DACVB, DACAW
College of Veterinary Medicine, North Carolina State University

Learning Objectives: At the end of this lecture, participants should be able to:
--Describe how cats manifest signs of fear and anxiety.
--Name 4 situational drugs used to attenuate fear and anxiety in cats.
--When presented with case of fear/anxiety in a cat, describe a pharmacologic treatment plan.

Introduction
As both predators and prey, cats are extremely sensitive to changes in their environment, in the form of novel visual, auditory, or olfactory signals. Common behavioral expressions of anxiety and fear are flight (escape, hide), fight (aggression), or freeze, which may indicate fear and/or anxiety. For caged or indoor cats experiencing fear, an inability to escape may lead to a worsening of their welfare. This lecture will address pharmacologic treatment of fear and anxiety in cats. In all cases, behavioral and environmental management should also be implemented, including crate and car travel conditioning and low stress handling in the veterinary hospital. At home, a safe confinement area should be established where the cat may be comfortably confined away from visitors and workers. This safe place, perhaps the master bed room, should be provisioned with key environmental resources, and use of feline pheromone and familiar odors. Cats should not be punished when they exhibit anxiety or fear.

Pharmacologic treatment
A number of recent clinical studies have established safe, well-tolerated single dose oral behavioral medications to attenuate situational fear and anxiety in cats (Table 1). Benzodiazepines are not included on this list, due to the rare but sometimes fatal condition, hepatic necrosis, has been reported in response to oral use of this medication in cats. The medications are most affected when administered to a calm cat. Thus, the medications should be administered at least one hour prior to the sequence of events that triggers fear and anxiety. For example, the cat should be medicated before the crate is taken out.

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Drug Name</th>
<th>PO Dose &amp; Freq.</th>
<th>Side Effects, Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricyclic Antidepressant</td>
<td>Amitriptyline (Elavil®)</td>
<td>10 mg tablet 2 hours prior</td>
<td>Decreased fear/anxiety, mild sedation; bitter taste. Coat pill in butter before administration.</td>
</tr>
<tr>
<td>*Anti-convulsant, anxiolytic</td>
<td>Gabapentin (Neurontin®)</td>
<td>50 mg/cat single dose before veterinary visits</td>
<td>Mild sedation. Drug must be compounded as a flavored suspension</td>
</tr>
<tr>
<td>*Serotonin Agonist and Reuptake Inhibitor (SARI)</td>
<td>Trazodone (Desyrel®)</td>
<td>50 mg/cat single dose before veterinary visits</td>
<td>Mild sedation. Pill may be given per os, or tablet may be broken into pieces and hidden in wet cat food.</td>
</tr>
</tbody>
</table>

For drug doses: [https://www.merckvetmanual.com/behavior/](https://www.merckvetmanual.com/behavior/)
Additional Resources (access via https://scholar.google.com/)
Pharmacologic treatment of situational fear and anxiety in dogs: Travel, vet visits, thunderstorms, and fireworks
Barbara L. Sherman, MS, PhD, DVM, DACVB, DACAW
College of Veterinary Medicine, North Carolina State University

Learning Objectives: At the conclusion of this lesson, participants will be able to:
--Recognize behavioral signs in dogs associated with fears and anxieties
--When given a case scenario of an anxious or fearful dog, develop a pharmacologic treatment plan

Introduction:
Dogs suffer from a number of behavioral disorders, including fear, anxiety, phobia, and stress. Since there is some confusion about the meaning and use of these words, to avoid confusion, they are defined, below.
--Fear is an emotion of alarm and agitation caused by a real or threatened danger. Among animals, fear is manifest by physiological responses such as tachycardia, hypersalivation, or elimination, and behavioral responses associated with escape, avoidance or defensiveness. Fear responses occur in response to the presence or proximity of an object, individual, or social situation.
--Anxiety is a reaction to an unreal or imagined danger or uncertainty. Anxiety includes both physiological signs (increased respiratory and heart rates, vasomotor changes, trembling or paralysis, increased salivation or sweating, gastrointestinal disturbances, etc.) as well as behavioral signs. The behavioral signs may include changes in activity (immobility, pacing, circling, restlessness), changes in nearest neighbor distances, reluctance to eat, etc.
--A phobia is a marked, persistent and excessive fear of clearly discernible, circumscribed objects or situations. Exposure to a phobic stimulus almost invariably provokes an immediate anxiety or fear response. The response may take the form of a situationally-bound or situationally-predisposed “panic attack”. Phobias often lead to avoidance behavior.
--“Stress” is a term used to describe a state of chronic anxiety and distress that impairs normal functioning and competence, and may be associated with frustration, fatigue, agitation, and inability to relax. Scientifically, stress refers to permutations in the hypothalamic-pituitary-adrenal axis, causing an increase in endogenous steroids and is characterized by increased heart rate and respiratory rate. In dogs, these negative welfare states may be difficult to differentiate.

Animals in these states show physical signs, including: ↑ resp. rate, ↑ heart rate, trembling, hypersalivation, dilated pupils, loss of bladder/bowel control, and anal gland expression. Behavioral signs include: aggression, anorexia, attention-seeking, barking, catatonia, destructiveness, hiding, hyperattachment, immobility, pacing, restlessness, self-injurious, and whining as well as postural signs.

The cause of fears, phobias or anxiety states varies from individual to individual. Often a specific cause cannot be identified. In certain cases, a specific frightening event triggers futures responses. Some potential etiologies include: inadequate socialization and impoverished early experience, innate (heritable) response, breed or individual temperament predispositions, learned responses through traumatic experiences, or a combination of one or more of these potential etiologies.

In this talk, pharmacologic management of dogs’ specific responses to travel, veterinary visits, thunderstorms, and fireworks will be discussed. Since these events are transient and episodic, situational medication is indicated (Table 1). Management is most successful when events are predictable, allowing affected dogs to be medicated in advance. Common situational medications include benzodiazepines, trazodone, and transmucosal dexmedetomidine, and clonidine.

Treatment Strategy (Table 1):
--Client should “test drive” the situational drug and dose before the inciting event.
--If possible, administer the situational drug BEFORE starting travel preparations, i.e. getting in car to go to vet, before thunderstorms, and before fireworks.
--If this is not possible, give the situational drug as soon as possible when the event begins.
--Positive behavioral management (not punishment) should be utilized in combination with the drug(s)
The first dose of the situational drug may be given the day prior to the event to increase the effectiveness of the situational drug. May increase event of situational drug to give first dose the day before the event.

All situational drugs may be used in combination with daily SSRIs, such as fluoxetine.

Table 1

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Drug</th>
<th>Approximate Onset of Action</th>
<th>Approximate Duration of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzodiazepine</td>
<td>alprazolam</td>
<td>60-90</td>
<td>medium</td>
</tr>
<tr>
<td></td>
<td>diazepam</td>
<td>30-60</td>
<td>long</td>
</tr>
<tr>
<td></td>
<td>lorazepam</td>
<td>60-90*</td>
<td>medium</td>
</tr>
<tr>
<td></td>
<td>clonazepam</td>
<td>60-90</td>
<td>long</td>
</tr>
<tr>
<td>SARI Serotonin Antagonist &amp; Reuptake Inhibitor</td>
<td>trazodone</td>
<td>1-2 hr</td>
<td>4 hr</td>
</tr>
<tr>
<td>Alpha-2 agonist</td>
<td>dexmedetomidine</td>
<td>20-40 min</td>
<td>2 hr (may redose)</td>
</tr>
<tr>
<td></td>
<td>Sileo®</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>clonidine</td>
<td>1 hr</td>
<td>4+ hr</td>
</tr>
</tbody>
</table>

*No active metabolites

Additional Resources (access via https://scholar.google.com/)

Puppy socialization to reduce the risk of behavior problems in adulthood
Barbara L. Sherman, MS, PhD, DVM, DACVB, DACAW
College of Veterinary Medicine, North Carolina State University

Learning Objectives. At the conclusion of this lecture, attendees should be able to:
--Explain to clients the importance of puppy socialization and what it means.
--Train a staff member to apply positive behavior methods to handle a puppy for a vaccine and to teach a client how to housetrain a puppy.
--Provide a written resource to all puppy owners to explain how to crate train, confinement train, housetrain, greet people, and how to provide for puppy’s behavioral needs.

Introduction
Many studies have documented the existence of a special learning period in dogs, called the socialization period. This stage, from approximately 3-14 weeks of age, is a time of brain maturation and plasticity. Positive experiences acquired during this time have effects during the dog’s entire lifetime. As adults, dogs well socialized as puppies, unlike inadequately socialized dogs, exhibit increased exploratory behavior, increased learning ability, increased sociability toward people, and decreased relinquishment and behavior problems. Higher retention in the home was reported for dogs that participated in humane society puppy socialization classes (Duxbury et al 2003). This is especially important for households with young children. If isolated from people during the critical socialization period, pups will be fearful and retreat from people. In extreme cases, this fearfulness cannot be overcome and dog is handicapped its entire life.

Clients should be educated to understand the importance of the socialization period so that they adopt their new puppy at 7-8 weeks and use the next 8 weeks for socialization. Concepts critical to the socialization process are listed on Table 1. The puppy should be exposed in a positive way to many people of different sexes, races, and ages, including children, and other dogs. The veterinary hospital should be part of this socialization experience. Dogs should be handled in positive ways and given small treats when they exhibit calm, friendly behavior. Gentle handling should characterize all experiences so that the puppy does not become fearful or defensive.

During puppy vaccination visits, basic instruction in puppy care and handling should be given and questions related to behavior answered. In educating owners, the need for appropriate exercise and play should be emphasized. Many behavior problems can be avoided if a puppy is properly housetrained, taught to be alone, and taught acceptable chew toys. Behavior problems that develop in puppies and can cause young dogs to be relegated outside or turned in to an animal shelter include: chewing, house soiling, excessive vocalizing, and aggression.

Veterinary clinics should either offer “in house” puppy classes or refer puppies to such classes elsewhere. “Puppy parties” can be held in the reception area of a veterinary hospital, where puppies of about the same age can be handled, exposed to different people, and allowed to play. During these sessions, clients can learn how to teach their puppy acceptable behavior. Each owner should reward calm, obedient behavior with praise and treats and not reward jumping up or whining (turn away and withdraw attention).

Table 1

<table>
<thead>
<tr>
<th>Age</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3 weeks</td>
<td>Primary period</td>
</tr>
<tr>
<td>3-7 weeks</td>
<td>Period with dam, siblings &amp; caretaker: species-specific and social behavior</td>
</tr>
<tr>
<td>3-14 weeks***</td>
<td>Socialization period of puppies</td>
</tr>
<tr>
<td>7-8 weeks</td>
<td>Best time to be placed in home</td>
</tr>
<tr>
<td>7-14 weeks</td>
<td>Provide: Behavioral needs (play, exercise, social interactions), positive manners training (including walk on leash, sit/stay, recall), “house rules,” confinement (crate or pen) conditioning, housetraining, meeting kind strangers without eliciting fear, car travel; avoid punishment</td>
</tr>
<tr>
<td>7-14+ weeks</td>
<td>Puppy socialization classes, Puppy “parties”</td>
</tr>
</tbody>
</table>
3-14 weeks | If isolated from people, pups are fearful and behave like wild dogs
14 weeks – social or sexual maturity | Juvenile period
Adult | 

The role of the veterinary hospital:
--Model to clients positive handling of puppies for lifting, exam, vaccines
--Explain the importance of socialization and what it means
--Offer “puppy parties” or recommend “socialization classes” for healthy puppies
--Be a resource for puppy questions (designate 1-2 interested staff members)
--Provide puppy socialization materials to clients (Table 2)

**Table 2**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Reward obedient, calm behavior (“pay” with praise/treats for what you want!)</td>
</tr>
<tr>
<td>2</td>
<td>Do not reward (with attention) pushy or demanding behavior</td>
</tr>
<tr>
<td>3</td>
<td>Do not punish: walk away or remove puppy from situation</td>
</tr>
<tr>
<td>4</td>
<td>Teach the puppy to be alone (use crate or confined space)</td>
</tr>
<tr>
<td>5</td>
<td>Teach manners (sit, stay, come, walk on a leash, no jumping up)</td>
</tr>
<tr>
<td>6</td>
<td>Provide and encourage use of appropriate chew toys</td>
</tr>
<tr>
<td>7</td>
<td>Prepare for vet visits: practice picking up puppy, placing it on a table and giving small treats for immobility and gentle examination.</td>
</tr>
<tr>
<td>8</td>
<td>Housetraining and confinement (crate or pen) conditioning</td>
</tr>
</tbody>
</table>

Benefits of socialization:
--Dogs that are appropriately socialized as puppies are less likely to exhibit behavioral problems as adults, including aggression and fearfulness.
--If socialized well as puppies, dogs are more likely to engage in positive social behaviors with humans
--If socialized well as puppies, dogs can learn to play games with humans better than dogs without proper socialization.

Additional Resources (access via [https://scholar.google.com/](https://scholar.google.com/))
--AVMA. [https://www.avma.org/KB/Resources/Reference/AnimalWelfare/Pages/Socialization.aspx](https://www.avma.org/KB/Resources/Reference/AnimalWelfare/Pages/Socialization.aspx)
--Martin KM, Martin D. Puppy Start Right, Sunshine Books, 2011. FOR CLIENTS
AIMS OF THIS TALK

The ECG provides unique diagnostic information and our ability to make a timely and accurate rhythm diagnosis often has immediate therapeutic implications. Murphy’s Law of Electrocardiography suggests that the most perplexing of ECGs will usually belong to the patients most in need of a quick and proper rhythm diagnosis. This is a 2-part lecture with each component presented consecutively in 50 minute segments. The first part will review ECG basics and help attendees develop a systematic diagnostic approach to ECGs. The goal is that a more thorough understanding of normal and abnormal complexes can aid identification of more complex arrhythmias when they occur. In the second half of the talk we will use case example ECGs presented as unknowns. We will use these as a springboard to discuss a wide spectrum of common arrhythmias with a focus on when and why they occur and the hemodynamic consequences that often determine if and how we intervene. We will discuss therapeutic options and why some treatment choices are better in particular situations and how we should measure success/failure of therapy.

ECG BASICS

The ECG is a graphic representation of the depolarization and repolarization of cardiac muscle cells as recorded from the body surface. Time is expressed in millimeters/second and is plotted on the horizontal axis versus voltage, which is expressed in millivolts on the vertical axis. The ECG is a superb diagnostic tool for determination of heart rate and rhythm and is uniquely better than even a trained ear at these two often critical determinations. Many electrocardiographic recording devices will automatically count the heart rate, but these automatic counts can be inaccurate - especially in veterinary medicine. The relatively wide breadth of morphologic normality for T waves and QRS complexes in our patients can lead to markedly inaccurate heart rates in machines calibrated for humans. Likewise, ECG monitors that purport to automatically yield a rhythm diagnosis can often prove misleading. These machines have a particularly difficult time interpreting the variations in sinus rate that are often a normal variant in our patients. Thus, there is still no substitute for careful assessment of cardiac rhythm and an accurate count of heart rate. Acquisition of an accurate heart rate is especially important when managing bradycardic and tachycardic patients and this heart rate often plays a role in guiding the necessity for medical intervention. Accurate manual heart rate assessment simply involves counting the number of QRS complexes in three or more seconds and multiplying appropriately in order to yield a per minute rate. If the cardiac rhythm is highly irregular or slow, then using at least six seconds of an ECG strip to extrapolate a heart rate will yield a more accurate count. In the setting of single early beats or non-sustained runs of tachycardia, it is also often helpful to measure an instantaneous heart rate for the early beats and a simple method will be discussed in this talk.

Indications

One of the most important indications for performing an ECG is determination of heart rate and rhythm and proper arrhythmia diagnosis. Multiple case examples will be used to illustrate this evaluation. While rate and rhythm may be accurately inferred following a careful auscultation in many patients, there are special circumstances in which an ECG may be indicated. These include irregular rhythms suspected of potentially being pathologic, bradyarrhythmia, tachyarrhythmia, or as part of a complete workup in patients with cardiac disease. Other indications for an ECG include pre-operative evaluation, anesthetic monitoring, monitoring of patients with non-cardiac conditions that predispose to arrhythmias, evaluation of collapse or weakness of unknown origin, patients whose body condition or disease prevents reliable auscultation, and screening certain patients with breed-associated arrhythmias.

Limitations

While the ECG is uniquely poised to give information regarding cardiac rate and rhythm, it is important to realize its limitations. A major limitation is that the ECG is a relatively insensitive test for detecting cardiac disease and is generally non-specific as to the underlying disease in the presence of abnormal ECG findings. Thankfully there are other tests that are better suited to determine if primary heart disease is present, make a cardiac diagnosis, evaluate hemodynamic status and characterize cardiac function. The ECG is but one indispensable part of the cardiac evaluation. Another important limitation is that the transient nature of some rhythm disorders can lead to false negative ECG findings if the arrhythmia does not occur during the recording period. This is a limitation that can often be overcome with ambulatory ECG monitoring and case examples utilizing these technologies will be discussed.

Accurate ECG assessment can be limited by the quality of the recording. Proper recording technique can help to avoid many of the pitfalls associated with artifact. The standard position for recording a small animal ECG is right lateral recumbency. If the patient is too dyspneic for lateral recumbency a standing or sternal ECG can be recorded. Gentle limb restraint will often help to minimize tremble artifact. It is important to avoid contact between the leads and to limit
lead and wire movement with respiration. Such movement can be avoided by proper placement of the limb leads away from the chest (just above the olecranon and in the area of the patellar ligaments). Case examples will be presented that focus on methods to determine if abnormal "complexes" represent arrhythmia or artifact. A helpful guideline to keep in mind is that an actual arrhythmia has to make physiologic sense in the greater context in which it is seen. Thus, the complexes surrounding the suspect deflection can often yield important information that alters the likelihood that the deflection represents an arrhythmia. For example, a deflection that may represent a VPC is likely to be so if it is immediately followed by a non-conducted P wave, but more likely to be artifact if it appears to fall in the QT interval of a sinus beat without altering the T wave of the sinus complex. An ECG device that is capable of recording multiple leads simultaneously can also be helpful when trying to distinguish artifact from arrhythmia, as certain motion artifact will be detected in some leads but not others. Such a finding would suggest artifact, rather than arrhythmia, as at least some portion of an arrhythmic complex would be expected to be seen in each lead tracing.

Physiologic Correlates and ECG Assessment

A regular sinus rhythm emanating from the sinus node is the normal rhythm of the heart. The sinus node is a highly organized cluster of specialized cells located in the right atrium near the junction with the cranial vena cava. The cells within the sinus node have the ability to spontaneously depolarize during phase 4 of the cardiac cycle. While this is not a trait exclusive to cells of the sinus node, the sinus node assumes the role of the dominant cardiac pacemaker under normal physiologic conditions because of two basic physiologic properties of sinus node pacemaker cells. These cells have both a low level of resting diastolic membrane potential and a relatively rapid rate of rise of phase 4 diastolic depolarization, as compared to other cells in the heart that have the ability to act as pacemakers. Thus, the sinus node normally controls the heart rate and rhythm, and by doing so suppresses the activation of subsidiary pacemakers. The importance of these phenomena in understanding bradyarrhythmia and tachyarrhythmia will be discussed in this lecture.

The initial depolarization of the cells of the sinus node is a relatively small electrical event and is not significant enough to be detected on a surface ECG. After emerging from the sinus node the cardiac impulse traverses both atria in its path to the atroventricular (AV) node. The normal P wave represents the depolarization of the atria that results from sinus node firing. The sum of the wavelets depolarizing the atria is an electrical wave that predominantly moves caudally and from right to left, and thus the normal P wave is thus a positive deflection when viewed from lead II. Disease processes that structurally alter the atria can affect the height or width of the P wave. A tall P wave (P pulmonale) is classically associated with an enlarged right atrium and wide P waves (P mitrale) are often associated with an enlarged left atrium. These are not entirely specific findings however, and these ECG changes are relatively insensitive markers of chamber enlargement. The AV node is the only electrical pathway between the atria and the ventricles under normal circumstances. Electrical activation of the AV node begins somewhere during the period of the P wave on the ECG.

Conduction proceeds relatively slowly through the AV node, which has implications in the medical management of arrhythmias as will be discussed in this lecture. Because it cannot be known precisely when AV nodal conduction begins, the PR interval (the time from the beginning of the P wave to the beginning of the QRS complex) is used as a surrogate for measuring AV conduction velocity. Normal ventricular activation occurs via the rapidly conducting specialized Purkinje system. Cardiac or pulmonary diseases that alter the relative distribution of myocardial mass can affect QRS morphology and the mean electrical axis of the ECG. Cardiac diseases that lead to eccentric or concentric myocardial hypertrophy can lead to changes complex size and sometimes cause conduction disturbances.

With this information as background, in the 2nd part of this lecture we will evaluate a collection of challenging arrhythmias. Once we make a diagnosis we will consider how it is that we decide if a particular arrhythmia warrants therapy. Many arrhythmias that we see in practice have severe manifestations which require intervention and less severe forms that may not. We will use case examples to explore in detail the following questions - the answers to which will determine if and how we choose therapy.

1. Is the arrhythmia hemodynamically significant?
2. Does it appear to be causing clinical signs?
3. Can a correctable or self-limiting underlying cause for the arrhythmia be identified?
4. Does the arrhythmia appear likely to degenerate into a less stable rhythm?
5. Is there a consideration for interactions between anti-arrhythmic therapy and current therapy?
6. Is the patient under or soon to be under general anesthesia?
7. What are the risks of beginning therapy?
8. What measures will I use to determine if my therapy is effective?
Acquired degenerative mitral valve disease (DMVD) is the most common cause of cardiac morbidity and mortality in our canine patients. Although this is a chronic disease that generally has at least a multi-year period with no or few obvious outward symptoms, many patients may eventually present for care because of acute life-threatening symptoms associated with left-sided congestive heart failure. However, if we are able to successfully get them through these acute crises, they have the potential to return to a good-quality life for a meaningful amount of time.

The diagnosis and successful treatment of left-sided congestive heart failure resulting from DMVD depends upon timely and correct diagnostics and therapy. Although a certain percentage of patients with left-sided congestive heart failure resulting from DMVD will respond positively to a formulaic treatment approach, many will not. This is partially due to variation in individual patient parameters such as the degree of disease, the severity of pulmonary edema on presentation, the level of pre-existing cardiac therapy, the presence or absence of arrhythmias or pulmonary hypertension and the health of other organ systems among other factors. Additionally, the unique combination of this disease and it's therapies can lead to complications including systemic hypotension, azotemia, tachyarrhythmias or simply worsening respiratory status despite CHF therapy. It is in these patients in particular that a more nimble individualized approach can lead to better outcomes. Such a nuanced approach requires careful observation of the patient and their response to our initial therapeutic choices. This live lecture will first review the natural history, pathophysiology and common clinical presentations seen with this disease. We will then discuss how a skilled history and physical exam and careful clinical observations during initial diagnostics and treatment can positively influence our choices at critical decision points. Your observations and judgements at these inflection points are a large determinant of better or worse patient outcomes.

The first challenge is correctly identifying these patients upon arrival at the hospital. There are clues in the presenting complaint, patient signalment, physical exam and clinical history that can raise or lower your index of suspicion for this diagnosis. Patients suffering the symptoms of left-sided congestive heart failure commonly present because their owners notice some combination of tachypnea, dyspnea, lethargy, syncope, cough or inappetance. Many dogs in this condition will show some, but not all, of these symptoms. These symptoms do not make the diagnosis though as they are not exclusive to dogs in congestive heart failure. Many dogs presenting with some combination of these complaints will be doing so for other medical reasons.

DMVD does occur commonly in both male and female dogs, although some reports suggest that males often acquire the condition at a slightly younger age. Although most dogs with DMVD will never develop congestive heart failure, those who do tend to be middle-aged to older. Importantly, they usually have had a multi-year period of exhibiting DMVD without serious symptoms prior to developing left-sided congestive heart failure. Therefore if they have been patients of your hospital prior to this time with regular visits there is a strong chance that a left-apical systolic heart murmur would have been noted during past visits. On emergency presentation dogs with clinically significant DMVD should have an auscultable systolic heart murmur heard best at the left cardiac apex. In an emergency presentation it can occasionally be more difficult to hear because of sounds associated with increased respiratory effort or because of the presence of arrhythmias. Keep in mind that the presence of this murmur with the clinical symptoms described above is consistent with congestive heart failure but not diagnostic for it. However, the clear absence of a left-sided cardiac murmur is strongly suggestive that a dog does not have serious DMVD. There are other clues in the heart rate and rhythm that can suggest whether congestive heart failure from DMVD is more or less likely. Dogs with this presentation most commonly will have either sinus tachycardia or a relatively fast sinus rhythm. The presence of a relatively slow sinus rhythm should raise serious suspicion that the clinical signs seen may not be the result of congestive heart failure. Ventricular and supraventricular (including atrial fibrillation) arrhythmias are not uncommon in this setting. Serious tachyarrhythmias may require anti-arrhythmic treatment at the same time the congestive heart failure is being treated in order to optimize patient outcomes and their use in this setting will be discussed. Thoracic radiographs are of course an obvious outward symptoms, many patients may eventually present for care because of acute life-threatening symptoms associated with left-sided congestive heart failure. However, if we are able to successfully get them through these acute crises, they have the potential to return to a good-quality life for a meaningful amount of time.

With this as background, in this lecture we will discuss use cases and the potential benefits and risks of various classes of medications including diuretics, inodilators, ACE-inhibitors, positive inotropes, vasodilators, anxiolytics, anti-anti-arrhythmias and oxygen therapy. We will discuss common complications in treated patients (hypotension, tachyarrhythmias, azotemia, anxiety, worsening failure) and how we might react to these complications by re-calibrating our approach for a particular patient's needs.
GOALS OF THIS LECTURE

Cats with cardiomyopathy present significant diagnostic challenges. The most common cardiomyopathies in cats (hypertrophic and restrictive cardiomyopathy) tend to be associated with no obvious outward clinical signs that an owner might notice until cats reach a crisis stage as a result of congestive heart failure or thromboembolic complications. Moreover, even our annual physical exams are not always as useful as we would like. This is because some well-feeling cats with heart disease have no murmur or arrhythmia or gallop on physical exam. As if that did not make your job challenging enough, a significant number of asymptomatic cats with heart murmurs will end up having a relatively benign cause for their murmur with no serious underlying heart disease. Echocardiography by a highly trained sonographer is considered the gold standard for diagnosis and characterization of these cardiomyopathies. However, it is not practical to perform an echo study on every feline patient and not even every cat with a heart murmur has an owner who is able or willing to have an evaluation by one of the limited number of cardiology specialists. It would thus be ideal if there were a test that could be performed in practice they could reliably help to let you know which asymptomatic cats were more likely to have serious heart disease and which were not. NT-proBNP is a test that can be useful as a screening test for cardiomyopathies in certain common clinical settings - provided we choose our patients to test carefully and understand the limitations of the test. In this talk we will discuss the utility of this test in different feline patients - as well as how patent selection significantly must impact our interpretation of the results.

BACKGROUND

B-type natriuretic peptide (BNP) is a naturally circulating hormone that promotes vasodilation and renal sodium and water loss. BNP is produced and secreted into the blood by cardiac myocytes - the muscle cells of the heart. Low basal concentrations of BNP can be detected circulating at all times, but the heart increases production and secretion of BNP in response to excessive stretching of heart muscle cells - as occurs with excess intracardiac volume expansion. This volume expansion - or cardiac enlargement - is a common late-stage result of many serious cardiac diseases in our patients including feline cardiomyopathies. While not a precise measure in any one individual cat, the degree of an increase in circulating BNP tends to be correlated to the severity of the underlying cardiac disease. The BNP pro-hormone is secreted into the circulation during periods of cardiac stress/stretch and is cleaved into the carboxy-terminus (C-BNP) and the amino-terminus (NT-proBNP). The NT-proBNP segment is more stable than proBNP or C-BNP and has a longer half-life and these facts make it a more reliable subject to measure and track. A commercial assay for feline NT-proBNP has been available for a number of years and more recently a qualitative in-house ELISA SNAP test was introduced.

UTILITY IN DIFFERENT CAT POPULATIONS

In order to most effectively use NT-proBNP we need to consider the patient we are performing this test on. Any test that tests for a disease can yield a positive or negative result. In reality, the patient may or may not have the condition being tested for. A test result that is positive when the patient truly has the condition is a true positive and a test result that is positive when the patient does not have the condition is a false positive. A negative test result can likewise be a true or false negative. The Positive Predictive Value (PPV) of a test refers to the likelihood that a positive result in an individual patient is a true positive.

A critically important consideration is that the PPV of any test is influenced by the prevalence of disease in the population that is being tested. If a test is performed in a high prevalence setting, it is more likely that a patient who tests positive truly has the disease than if the test were performed in a population with a lower prevalence of the disease. For example, consider the use a Doppler device to test for feline systemic hypertension. This condition is far more common in geriatric cats with chronic renal failure than it is in three-year-old cats with normal kidney function. Now imagine that a geriatric cat with chronic renal failure and a three-year-old normal cat are each put in carriers and driven to a veterinary clinic. Blood pressure measurements are performed and both measurements measure 180 mmHg. These cats are not equally likely to truly have chronic systemic hypertension. This “positive” result is far more likely to be a true positive in the geriatric cat because this cat is from a population with a far higher incidence of systemic hypertension. In the three-year-old cat this measurement, while true in the moment, most likely is a transient “white-coat” effect if so, then this would represent a false positive false positive.

This principle of the incidence of disease in the tested population affecting the PPV is true for any test including NT–ProBNP. This has important implications when using NT-proBNP to screen a cat for a serious cardiomyopathy, as a clinician can increase the PPV (increase the chances that a positive result is actually true) for example by employing the
test in cats who are likely to have an increased risk of having a serious cardiomyopathy - such as cats with a heart murmur, arrhythmia, gallop and/or radiographic cardiomegaly. In such a population of cats, a recent study found that when a commercial qualitative cage side ELISA NT-proBNP assay was used to try and identify which cats from this high risk population truly had a moderate or severe cardiomyopathy, the PPV of the assay was 62%. Therefore, an asymptomatic cat with a heart murmur, gallop, arrhythmia and/or cardiomegaly who tested positive on the ELISA assay had a 62% chance of having a significant cardiomyopathy (and a 38% chance of being a false positive). It is important to keep in mind that this is a population of cats with a higher than average risk for cardiomyopathy and that this same test run on a population of cats less likely to have heart disease (all cats undergoing anesthesia for dentals for example) would lead to a higher rate of false positives. We will review these findings and discuss their implications in detail in this live lecture. The Negative Predictive Value (NPV) of this cage side quantitative test for a serious cardiomyopathy in this same population of high risk cats was relatively high. Therefore, in this population of high risk asymptomatic cats with murmurs, gallop sounds, arrhythmia and/or cardiomegaly, a negative visual SNAP result meant that the cat was unlikely to have a serious cardiomyopathy. About 6% of these cats who tested negative actually had a serious cardiomyopathy and were thus false negatives. Because the positive predictive value of the SNAP test in this population of cats was lower than the negative predictive value, this means that a negative result was less likely to be false than a positive. This suggests that in an asymptomatic cat with a heart murmur, NT-proBNP might be a better (albeit imperfect) test to rule out serious heart disease than to rule it in. In the live lecture we will discuss various ways that you can use these facts when presented with an asymptomatic cat with a heart murmur.

Cats who present because of serious respiratory clinical signs (dyspnea, tachypnea, cough) most commonly have underlying heart disease that has led to congestive heart failure or serious primary respiratory disease such as asthma, neoplasia, pneumonia, pleural space disease, etc. The treatment options for these often critical cases vary widely based upon the underlying condition. History and physical exam can often yield important clues that guide our index of suspicion for the underlying problem. Thoracic radiographs and/or a brief ultrasound check for effusion are important first order diagnostics but the sometimes fragile nature of these patients can make such procedures challenging. The quantitative NT-proBNP assay has been shown to be useful and a reasonable differentiator in these challenging cases, but the fact that this has to be sent out to a diagnostic lab limits its timely utility in these critical patients. As of yet the cage side clinical SNAP test has not been evaluated for accuracy in cats with respiratory signs. We will discuss some cases in which the SNAP test might still be useful when combined with other first order diagnostic tests. NT-proBNP has not been evaluated for accuracy at predicting likelihood of heart disease in the general population of cats who are not considered to have increased risk of heart disease - such as those with no respiratory symptoms or heart murmur or other known risk factors for heart disease. If NT-proBNP were tested in such a cat, the risk that a given positive result would be a false positive is undoubtedly higher than in the populations described above who have known risk factors for heart disease.
This talk is designed for an audience of veterinarians and we will review findings from the veterinary literature involving the use of pimobendan. We will focus primarily on what we have most recently learned about it’s use in canine patients with myxomatous mitral valve disease.

**BACKGROUND**

Pimobendan is a benzimidazole pyridazinone that possesses both positive inotropic and vasodilatory properties. Two separate mechanisms contribute to the positive inotropic effect, an increase in calcium sensitivity of cardiac contractile proteins and an inhibition of cardiac phosphodiesterase activity. Pimobendan also results in selective peripheral phosphodiesterase inhibition, which leads to systemic vasodilatation. This combination of increased contractility of the heart combined with vasodilation that decreases afterload (afterload being the pressure resisting forward cardiac output) can result in improved cardiac output and reduction of cardiac size and filling pressures.

Myxomatous mitral valve disease (MMVD) is the most common cardiac disease in the dog. While MMVD can remain chronically mild and well-tolerated in many dogs, it remains the most common cause for cardiac morbidity or mortality. The progressively degenerative valvular change results in mitral valve insufficiency (regurgitation) and this can lead to a gradually increasing chronic volume load on the left side of the heart. In some dogs, the volume load will result in enlargement of the left heart that can be detected radiographically or with echocardiography. Some of these dogs with MMVD and secondary left-sided heart enlargement will eventually develop signs of congestive heart failure (CHF), that is, pulmonary venous congestion and edema. Dogs with MMVD can thus progress through various stages of the disease with not all dogs reaching all stages.

For the sake of classification a widely used staging system divides affected dogs into 4 categories. Those whose signalment puts them at risk for developing MMVD (but they are not currently affected) are considered to be at stage A. Dogs with evidence of mitral regurgitation (MR) and no signs of CHF are considered to be at stage B. Those who go on to develop clinical signs of CHF are considered to be at stage C. Even if the clinical signs of CHF are successfully managed and subside, the dogs are still considered to be at stage C given their reliance on therapy to keep them out of congestive heart failure. Those dogs who have signs of CHF refractory to treatment are considered to be at stage D.

Within stage B this disease tends to progress at very different rates in different dogs. However, given the generally slowly progressive nature of this condition Stage B is usually a long multi-year period. Because of the wide clinical variance and time in stage B, stage B dogs are further divided into stages B1 and B2. Dogs who have demonstrable stage B disease (generally with a left apical systolic murmur) but not yet any evidence of cardiac enlargement are categorized as stage B1. Dogs in stage B1 who then go on to develop volume loading and cardiac enlargement, but have not yet developed signs of CHF, are categorized as stage B2. Dogs who are in stage B2 tend to breathe comfortably, have a good appetite and energy and overall enjoy a relatively high quality life. Because of this, any treatment that can help to prolong the B2 stage is especially meaningful to MMVD dogs and those who adore them.

**EPIC TRIAL**
Pimobendan has been previously shown to be of benefit in dogs with MMVD who have reached CHF/Stage C and the goal of the EPIC trial was to investigate whether pimobendan - when given as monotherapy to dogs with stage B2 MMVD - would prolong time in B2 by delaying either onset of congestive heart failure or euthanasia due to cardiac disease or other death presumed to be cardiac in origin. The EPIC study was designed as a prospective, multi-center, blinded, randomized and placebo-controlled trial. In order to be included in the trial dogs had to be at least 6 years of age, weigh between 4.1 and 15 kg, have a left apical systolic murmur of grade III or louder, have an echocardiographic diagnosis of advanced MMVD, and have BOTH echocardiographic evidence of left atrial and left ventricular enlargement and radiographic evidence of left-sided cardiomegaly.

Dogs were excluded if there were any other significant cardiac disease found on echocardiography, or if there were substantial pulmonary hypertension discovered. Dogs with advanced MMVD do have an increased risk for having arrhythmias (especially SVPCs and VPCs) but any dog with an arrhythmia significant enough that it required anti-arrhythmic therapy was excluded. Dogs were also understandably not allowed to have any other known severe systemic disease that might prevent them from completing the study.

Three hundred and sixty dogs were enrolled in the trial. This made it the largest controlled clinical trial to date investigating the benefit of medical therapy at this stage of disease. One hundred eighty dogs were randomized to receive pimobendan (as a chewable tablet) PO at a target dose of 0.4–0.6 mg/kg/day divided into two administrations approximately 12 hours apart. The other 180 dogs who were randomized to the placebo group received a visually identical placebo tablet. All dogs were followed and examined on a scheduled recheck plan or whenever there was concern that they were possibly developing CHF or having other new clinical signs. Dogs who received the active drug had a median prolongation of 462 days until the time that they reached the primary endpoint (EITHER CHF OR euthanasia due to cardiac clinical signs OR death thought to be related to cardiac disease). This approximately 15 month delay in that primary composite endpoint was statistically significant. There was not a statistically significant delay in the time to each individual component of that composite endpoint. For example, dogs receiving pimobendan had a longer but not statistically longer time until reaching CHF compared to placebo. However, lumping these 3 events into one composite primary endpoint seems very reasonable since these are 3 of the most likely serious complications to occur in this population of dogs and the delay of any one of them is clinically meaningful and in fact the goal of therapy.

The results of this trial demonstrate a benefit of pimobendan (versus no treatment) in this population of dogs who met the entry criteria. It is worth noting that most of the dogs enrolled had no serious outward symptoms when they entered the trial. For example, less than 15% of the enrolled dogs had a frequent or persistent cough and less than 3% of enrollees had a history of syncope. Ninety-six percent of enrollees were said to have good to very good exercise tolerance when the trial began. This tells us that there are many dogs with MMVD who could benefit from pimobendan well before they ever show clinical signs. This strongly argues for investigating moderate-loud left apical murmurs in dogs where the index of suspicion for MMVD is high - even if clinical symptoms are not present.

It is also worth keeping in mind that the population of dogs who received benefit from pimobendan in this study had been selected because they showed both echocardiographic evidence of MMVD, left atrial and left ventricular enlargement (LA;Ao ratio ≥ 1.6 and body-weight normalized LVIDD ≥ 1.7 respectively) as well as radiographic suggestion of cardiomegaly (Vertebral Heart Score ≥ 10.5). However, breed differences in VHS normals and variations in chest
confirmation can impact radiographic interpretation. There are a significant number of dogs with
the appearance of cardiomegaly on radiographs who do not have any such significant change
on echo. Further, many small dogs with a grade 3 or louder murmur will upon further evaluation
not (or in some cases not yet) have cardiomegaly that meets these criteria. Thus, these com-
plexifiers and the results of the EPIC trial emphasize the importance of careful investigation of a
loud left apical heart murmur to make rational decision as to what treatment might or might not
be indicated. In the lecture we will discuss various diagnostic strategies in symptomatic and
asymptomatic in small dogs with heart murmurs that take into account the diagnostic limitations
of our tests as well as the logistical limitations of having an echocardiograph for some patients.
A complete blood count (CBC), chemistry, and urinalysis (UA) are often referred to as a minimum database. Just as you approach a physical examination with a systemic, repeatable approach, you should do the same for these baseline diagnostics.

Starting with the CBC, start by characterizing the erythron (the red blood cell parameters). Typically the hematocrit (Hct), RBC count, and hemoglobin (Hgb) will trend together. However, there are scenarios where they may not, typically as a result of pre-analytical interferences (see proceedings on "Getting the most out of your clinical pathology samples..."). Sick patients are often either anemic or have an erythrocytosis. Starting with the approach to the anemic patient, you look to the MCV, MCHC, and if provided absolute reticulocyte count to further characterize the anemia. RBC morphology, whether noted by you looking at the blood smear, or provided on a diagnostic lab report can also be helpful with determining the mechanism of anemia (see proceedings on "You have an anemic patient, now what?"). As for an erythrocytosis, this is most commonly a relative erythrocytosis secondary to hemoconcentration, as a result of fluid losses. To help confirm this, you should refer to the chem panel and look for an increase in total protein and an albumin at the high end or increased above the reference interval (RI).

Quick interlude, keep in mind that a value within the RI does not rule out pathology. There can be mechanisms favoring an increase in a value (e.g. hemoconcentration), while another mechanism counteracting that with a decrease (e.g. negative acute phase response) resulting in a value within the RI.

Next, move on to the leukon (the white blood cell parameters) and characterize the leukogram. Inflammatory leukograms are common in sick patients, but keep in mind they can come in many different flavors depending on the severity and time course. For example, with acute severe inflammation you can have a neutropenia with a degenerative left shift (immature neutrophils outnumber segmented neutrophils), whereas with more long-standing inflammation, you can see a neutrophilia with or without a left shift and a monocytosis. To further support the presence of inflammation, you can look to albumin, which may be decreased due to a negative acute phase response, and the globulins, which may be increased due to antigenic stimulation. If there is an inflammatory leukogram, you want to try and determine the source of inflammation. This should prompt you to look at the chem panel. Keep in mind, the chem panel gives you information about certain body systems (e.g. liver, kidney), but can't help you when it comes to others (e.g. respiratory, cardiac). The latter systems really depend on a clinical history and your astute physical exam abilities to direct you there. A stress leukogram (neutrophilia +/- lymphopenia, eosinopenia) is another common finding in sick patients and can be supported by a mild to moderate hyperglycemia. When evaluating leukocytes, it’s also important to evaluate morphology on a blood smear. This becomes even more important in cases of bicytopenia or pancytopenia or a marked leukocytosis, which may signal an underlying neoplastic process (see proceedings on “How to make and evaluate a blood smear and reasons why you should...”).

Lastly on the CBC are platelets. If you have a thrombocytopenia, it is important to look at a blood smear as a quality check to evaluate for platelet clumping, which can falsely decrease the automated platelet count. Unfortunately if clumps are present, there isn’t a calculation to account for them. Just know that whatever count or estimate you get from a blood smear, is a minimum. Speaking of estimates, if you don’t have an automated analyzer, you can look at a blood smear to get an idea of a platelet count. This is done by finding the monolayer and counting platelets in ten 100x high power fields and multiplying the average by 15,000/ul.

Moving on to the chemistry panel... while it’s tempting to scan to see what’s high or low or to just look at one value at a time in isolation, I recommend breaking it down into systems (remember a normal result doesn't rule out pathology! See above). First up are electrolytes (Na, K, and Cl) and acid base (bicarbonate and anion gap). Even without a blood gas, you can detect metabolic acid-base disturbances with a chemistry panel. If an anion gap is not provided on your report, I would encourage you to manually calculate it [(Na + K) – HCO3 + Cl]). An increase in anion gap signals a primary titration metabolic acidosis caused by the accumulation of “unmeasured” anions such as lactate, uremic acids, ketones, or some toxins (e.g. ethylene glycol). If this is the only metabolic acid base disturbance, there will be a roughly proportional decrease in bicarbonate. However, if the bicarbonate is normal, this should signal a counteracting acid-base disturbance resulting in its increase (i.e. hypochloremic metabolic alkalosis).
To further support the presence of an acid-base disturbance involving chloride, evaluate sodium and chloride together. You can either “eye-ball” to see if they are roughly increased or decreased to the same degree, or calculate the “corrected chloride” (normal Na / patient Na) x patient chloride. The normal Na is half-way between the RI you are given. If the corrected chloride falls within the chloride RI, then the changes in Na and Cl are interpreted to be due to changes in free water. Whereas if the corrected Cl falls outside the RI, this signals an additional acid-base disturbance present. Then, it helps to think of Cl as an acid: so an increase would be an acidosis and a decrease would be an alkalosis. A disturbance affected chloride may be primary or in compensation for a respiratory disturbance. The most common disturbance affecting chloride in small animals is a primary hypochloremic metabolic alkalosis due to vomiting.

Next up are the renal values (BUN and creatinine) and minerals (calcium, phosphorous, and magnesium). When an azotemia is present, your eye should go straight to the urinalysis in order to classify it as pre-renal or renal. The urine specific gravity evaluates for concentrating ability and is considered adequate at 1.025 in a large animal, 1.030 in a dog, and 1.035 in a cat. Other UA results can help evaluate for tubular and glomerular function, including protein and glucose. As for post-prandial azotemia, look at the potassium to see if it is increased signaling decreased excretion. In an azotemic patient, Phos and Mg are often increased due to decreases in GFR. With renal disease, calcium can be quite variable and a total calcium should not be used to predict an ionized. Also, forget that correction formula you may have been taught! It doesn’t work. Measure ionized instead. When interpreting calcium derangements, it is helpful to evaluate it in conjunction with Phos to determine the underlying mechanism at play (e.g. alterations in PTH or vitamin D).

Then come the slew of results related to the liver. First are the leakage enzymes (ALT, AST), that when increased indicate hepatocellular injury. Keep in mind that AST (and to a lesser degree also ALT) are also located in muscle, so if CK is increased, there is concurrent muscle injury. CK has a huge range that it can increase – so even a result of 1,000 or so is considered relatively minor. A marked increase is around 10,000 or even higher. Think of hit by cars, ATEs, or rhabdomyolysis as causes of marked muscle injury. Then are the induction enzymes (ALP and GGT), which are increased with cholestasis and biliary hyperplasia (the latter for GGT). Remember that in dogs and dogs only, increases in corticosteroids (whether endogenous or exogenous) can result in an increase in ALP. Also, don’t forget the bone isoform of ALP, which will be higher in young, growing animals as a normal age-related change. As for evaluating liver function, remember the four synthetic products available on a chem panel: urea, albumin, glucose, and cholesterol. Decreases in some or all of these, should make you concerned about decreased liver synthetic function, potentially as a result of severe liver injury or decreased blood flow (as in a portosystemic shunt). Thinking a shunt? Go back to the CBC. Is there a microcytic anemia? Go to the UA. Are there ammonium biurate crystals? (See how we’re bouncing back and forth between all of our data?!). Then, there’s bilirubin. Some labs only provide a total bilirubin, whereas other labs may provide a split with indirect (unconjugated) and direct (conjugated) bilirubin. A split can be helpful when discerning the mechanism of a hyperbilirubinemia (e.g. increases in unconjugated bilirubin with a hemolytic anemia), however in many cases there are increases in both (i.e. often there’s concurrent cholestasis with an increase in conjugated bilirubin in a hemolytic anemia).

When evaluating proteins, it’s helpful to determine if there is a selective or non-selective change in proteins, by this I mean are albumin and globulins changing in concert or not. This helps to determine the mechanism of their changes. For example, a protein losing nephropathy is typically selective resulting in just a hypoalbuminemia, whereas blood and GI losses are non-selective and result in a panhypoproteinemia (decreases in both albumin and globulins). Inflammation, on the other hand, results in discordant changes with a decrease in albumin due to a negative acute phase response and an increase in globulins with antigenic stimulation.

Lastly, you can evaluate the metabolic parameters including glucose, cholesterol, and possibly triglycerides. Remember, before you panic about sepsis due to a hypoglycemia – make sure you didn’t leave that red top for too long before separating off the serum! This is a common pre-analytical error, and is not prevented by using a serum separator tube. Remember to try and get a fasted sample if you’re going to interpret triglycerides. However, increases in cholesterol (particularly marked increases) will not be attributed to a recent meal and should not be ignored, as this may be a sign of an underlying endocrinopathy.

And that’s about all of it! While I can’t go into everything in depth, hopefully this provides guidance in how you can approach laboratory data in your future cases! If you need help remembering mechanisms for bloodwork alterations, I recommend checking out Eclipspath.com, which is a free website out of Cornell University!
Cytology of common skin lesions and lymph nodes
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Cytology is a quick and minimally invasive diagnostic test that can help narrow down your differential diagnoses and at the best of times provide a definitive diagnosis. Some cytologic samples are challenging, even for clinical pathologists, and leave us interpreting the lesion as only “tumor,” and listing differentials, as the origin is unclear. This session is not about those cases. The goals of this lecture are to 1) provide a systematic approach on how to evaluate a cytology smear, 2) review some of the common skin masses readily diagnosed by cytology, and 3) provide guidelines on how to evaluate lymph node aspirates. With both skin masses and lymph nodes, the limitations of cytology will be highlighted.

Gross evaluation
Prior to staining your cytology sample, grossly evaluate the slide after you’ve performed your squash smear (see proceedings on ‘Getting the most out of your clinical pathology sample…’ for tips on smear prep). If the slide looks grossly greasy, that’s fat! This could mean one of two things: 1) you aspirated a lipoma or 2) you aspirated normal subcutaneous fat surrounding the mass. It’s important to know that cytologically they look identical, so this distinction is best made based on your clinical judgment of what the mass palpates like. If you’re trying to aspirate a lymph node, try again! It’s not uncommon to aspirate the fat surrounding a lymph node, rather than the node itself.

Microscopic evaluation of skin masses
When evaluating a cytology smear, first start at low power (10x objective). At this altitude, scan the entire slide and evaluate sample cellularity and staining quality. If it appears understained (too pink without hints of purple), then dip it in the 3rd (purple) solution some more prior to putting any oil on the slide. If you have cells (other than just blood), then you’re ready to move on!

Next, get a sense of the cell population(s) present. Are all of the cells the same (i.e. a homogeneous population, such as all neutrophils) or is there a mixture (i.e. a heterogeneous population, such as neoplastic cells and inflammatory cells)? It’s often easiest to start off by asking yourself – do I recognize these cells? This is often the case with inflammatory lesions. Although, don’t be discouraged by the sneaky macrophage. It can look like a lot of different things, including a multinucleated giant cell. Mild inflammation can be difficult to be confident of, since there is often blood present (with associated leukocytes) in the background. So you have to ask yourself, “do I think there’s more leukocytes than are just present from blood?” It’s helpful to know the peripheral WBC count in the patient to make this assessment (if you know it, provide it to your clinical pathologist in the history on the request form!). If not, that’s alright – you won’t miss severe inflammation. Also keep in mind, if you’re seeing cells such as macrophages or plasma cells, which are not normally in peripheral blood, that’s support for tissue inflammation, as well.

At low power, it’s also beneficial to evaluate the background of the smear. Other helpful such as extracellular matrix or the lining up of cells in a row (“windrowing”) may provide hints for the tissue of origin.

In the case of non-inflammatory cells, assess their arrangement at low power. Are the cells present in cohesive clusters (suggesting an epithelial origin)? Are they together in loose aggregates and also present individually, if so are the cells spindled (suggesting a mesenchymal origin) or round to oval with more distinct cell margins (as with round cells)? Are the cells in loose packets with lots of free (or “naked”) nuclei in the background (suggesting endocrine or neuroendocrine tissue)? Sometimes it can be difficult to tell at low power and takes a closer assessment at higher power to be sure.

Lastly at low power, find a nice well spread area of the smear with intact cells to go to higher magnification for further evaluation. Most clinic scopes have a 40x objective, which is a “high dry” (i.e. you should not use oil on this lens), whereas oil can be used on 50x and 100x objectives. At this objective, then you can start identifying inflammatory cells with more confidence: neutrophils, macrophages, lymphocytes, plasma cells, eosinophils, etc. For non-inflammatory cell populations, the above arrangements, as well as morphologic details about the cells themselves, can be assessed to try and classify their origin. Epithelial cells tend to be round, cuboidal, columnar or polygonal. Mesenchymal cells are often spindled or stellate (like a star or triangle). Round cells are you guessed it, round, but they can also be oval (especially histiocytes and plasma cells).
If it seems like you can’t make out cell borders and there are just nuclei with rare intact cells, this may be a result of excessive pressure in making the slide (i.e. rupturing the cells), however this may also indicate an endocrine or neuroendocrine origin. Lastly, non-inflammatory cells should be assessed for cytologic criteria of malignancy.

Normal structures (or benign lesions) have a relatively uniform appearance versus malignant neoplastic processes typically display three or more criteria of malignancy. However, there are some malignant tumors, which are cytologically “bland” and do not display features of malignancy (e.g. anal sac adenocarcinoma). Cytologic criteria of malignancy include variation in cell size (anisocytosis), variation in nuclear size (anisokaryosis), abnormally large cells, variation in N:C ratio, increased and bizarre mitotic figures, binucleation and multinucleation with intracellular anisokaryosis, multiple prominent nucleoli, macronucleoli, variably sized nucleoli, and abnormal nucleolar shapes. It’s important to note that cytology cannot always reliably rule out malignancy. In addition to those tumors that we know are cytologically bland but biologically aggressive, cytology cannot assess for tissue invasiveness, which may be displayed by some low-grade tumors and is only detected on histologic sections. There are also some unique aspects to evaluating for criteria of malignancy in lymph node aspirates (see section on Cytologic interpretation of lymph nodes).

Cytologic interpretation of skin masses

Non-diagnostic
There are several reasons why a sample may be non-diagnostic. Low cellularity samples are very common, and may be due to a poorly exfoliating lesion or poor collection technique. Sample cellularity is key for a confident cytologic diagnosis of neoplasia. Splat smears (see proceedings on “Getting the most out of your clinical pathology…” are also common and may be non-diagnostic or at minimum limit the certainty of a cytologic interpretation due to the distortion of cells and their arrangement. Excessive blood contamination creates a thick background, which complicates evaluation of individual cell detail. Also, blood-associated leukocytes complicate the interpretation of inflammation, as previously discussed. Lastly, a sample may be highly cellular, but if the cells are all ruptured, they can’t be evaluated. This can be due to exuberant collection methods or poor smear technique, although some tumor cells are excessively fragile and prone to rupture.

Then there are scenarios where the sample isn’t non-diagnostic per se, but isn’t helpful or the diagnosis you were looking for. This includes sampling error, in which surrounding tissue is aspirated instead of the desired mass or structure: for example, aspirating salivary gland when attempting mandibular lymph node aspiration. It’s also possible to aspirate a non-representative area of the lesion. For example, aspiration of inflammatory areas of a tumor or aspiration of reactive fibroplasia associated with an inflammatory lesion. This is where providing a clinical history is key to the clinical pathologist. This allows us to use our clinical judgment to suspect if what we’re seeing isn’t the whole story.

Inflammation
For inflammatory lesions, the types of inflammatory cells can help to narrow the list of differential diagnoses / tell you what you should be searching for.

- Suppurative / neutrophilic
  - Neutrophils are judged as either non-degenerate or degenerate. Non-degenerate neutrophils have crisp, lobulated nuclei, and appear similar to segmented neutrophils in peripheral blood. Whereas degenerate neutrophils have swollen nuclei, which appear lighter and have less crisp/obvious nuclear lobation. The terms “non-degenerate” or “degenerate” only apply to neutrophils in fluids or tissues (not peripheral blood).
    - Non-degenerate neutrophils: suspect immune-mediated, sterile irritants (bile, urine), neoplastic lesions
    - Degenerate neutrophils: suspect bacterial infection. Look carefully for phagocytized (intracellular) organisms.

- Histiocytic (macrophages)
  - Macrophages predominate, may see multinucleate forms.
  - Suspect foreign body, fungal, or higher bacterial infections (such as *Mycobacterium*, *Nocardia*, or *Actinomyces* spp.).

- Eosinophilic
  - >10-20% eosinophils
  - Suspect hypersensitivity/allergic conditions, parasitic disease, some fungal infections, or paraneoplastic processes (e.g. mast cell tumors or T cell lymphoma).
• Lymphocytic or lymphoplasmacytic
  o Heterogeneous mix of lymphocytes (mostly small lymphocytes) along with plasma cells, and other inflammatory cells.
  o Suspect antigenic/immune stimulation, early viral infections, insect bites, or chronic inflammation.
  o A homogenous population of lymphocytes in the absence of other inflammatory cells is suggestive of lymphoma.
• Mixed
  o Neutrophils and macrophages +/- lymphocytes, plasma cells, eosinophils
  o Suspect chronic tissue injury such as lick granulomas, but can also be seen in reaction to foreign bodies, fungi, and bacteria.

Keratin-containing lesions
These lesions are signaled by the presence of abundant anucleate keratinized squamous epithelial cells. They are very common, may be single or multiple, and may be non-neoplastic (i.e. follicular cyst) or neoplastic (e.g. trichoepithelioma, pilomatricoma). Neoplastic lesions tend to have clusters of nucleated epithelial cells, as well, but they may not be present in the aspirate. While these are typically benign tumors, surgical excision is a good idea, as the cystic component of these lesions may rupture releasing keratin into the subcutaneous tissue inciting a robust pyogranulomatous inflammatory response. Histopathologic examination is also needed for a definitive classification of tumor type and to assess for malignancy in these cases.

Neoplasia
Any category of neoplasia may occur in the skin. The following are some common examples with some notes / words of caution:

- Epithelial: perianal gland adenoma, trichoblastoma, squamous cell carcinoma
  o Low cellularity samples consisting of well-differentiated cells may represent a hyperplastic, rather than neoplastic lesion. Histopath is needed to make this distinction.
  o Squamous epithelial cells may display prominent atypia (dysplasia) in the face of inflammation. And squamous cell carcinomas are often very inflammatory. Best to submit to a clinical pathologist or obtain a surgical biopsy for histopathology, to prevent “overcalling” on this tumor type!
- Mesenchymal: soft tissue sarcomas
  o Non-neoplastic (reactive) fibroblasts associated with inflammatory lesions may display prominent cytologic atypia mimicking a sarcoma. If there’s inflammation and spindle cells – send it to a clinical pathologist for interpretation or obtain a surgical biopsy for histopathology.
- Round cell: mast cell tumor, plasmacytoma, histiocytoma
  o Plasmacytomas and histiocytomas can sometimes be challenging to differentiate.
  o Histiocytes may actually be in the minority as a histiocytoma regresses. Small lymphocytes dominate at this point.
- Endocrine / neuroendocrine: anal sac adenocarcinoma
  o Note, while the anal sac adenocarcinoma is technically an epithelial tumor, its cytologic pattern is that of an endocrine (e.g. thyroid) or neuroendocrine (e.g. carotid body) tumor.
  o This is the category in which even tumors that lack cytologic criteria of malignancy, may be malignant / biologically aggressive.

Cytologic interpretation of lymph nodes
There are several potential cytologic interpretations for lymph node aspirates:

- Reactive / lymphoid hyperplasia
- Lymphoma
- Metastatic neoplasia

There’s a different approach to cytologically evaluating lymph nodes, as compared to skin masses. First of all, just to state the obvious – lymphocytes should predominate. Beyond that, it’s all about proportions. A normal or reactive lymph node consists of a heterogeneous (mixed) population of lymphocytes with mostly small lymphocytes and fewer intermediate and rare to no large-sized lymphocytes. Few plasma cells may also be seen.
An interpretation of no cytologic abnormalities is technically possible if the node that is being aspirated is normal in size (i.e. aspirated for staging purposes), however typically normal sized lymph nodes are either not palpable and/or non-diagnostic owing to low cellularity. If the node is at all enlarged, it’s at least reactive / hyperplastic to some degree. A well-spread neutrophil is used in cytology to size lymphocytes. A small lymphocyte could fit inside of a neutrophil. An intermediate lymphocyte’s nucleus is approximately the same size as a neutrophil. And the nucleus of a large lymphocyte is larger than a neutrophil. With more reactivity / antigenic stimulation, there will be slightly increased numbers of large lymphocytes with deep blue cytoplasm and plasma cells, possibly even Mott cells (an activated variant of a plasma cell). Note, that rare mitotic figures can be seen in a reactive lymph node. Their mere presence does not signal neoplasia.

The general principle with lymphoma on cytology is a loss of this heterogeneity. So if the lymphocytes all appear the same, this should raise concern. (This is in contrast to other tumors, in which anisocytosis and anisokaryosis are criteria of malignancy). However, there are some forms of lymphoma that cytologically closely mimic a reactive lymph node (e.g. T cell rich B cell lymphoma, Hodgkins-like lymphoma) and require histopathologic examination to identify loss of normal lymph node architecture. Evaluation of lymphocyte chromatin is another key feature with diagnosing lymphoma, which Diff-Quik staining is not ideal for.

Cytology is best at definitively diagnosing large cell lymphomas. However, cytology cannot definitively immunophenotype (B vs T cell) based on morphologic appearance. Additional testing such as flow cytometry or immunohistochemistry is needed for this. The most common type of lymphoma affecting lymph nodes in small animals is diffuse large B cell lymphoma (DLBCL). Cytologically this typically appears as a homogenous population of large lymphocytes with fine chromatin and visible nucleoli. Mitotic figures may be few or numerous. Macrophages containing phagocytic debris (including phagocytized lymphocytes) are also often scattered throughout. It can be challenging to determine the size of a lymphocyte when all of the cells appear the same. Remember to find a neutrophil to help guide you. Large T cell lymphomas are far less common.

Since small lymphocytes normally predominate in lymph node aspirates, small cell lymphoma is not a cytologic diagnosis. However, there are two exceptions. One is a lymphoma of granular lymphocytes, which is an aggressive form of lymphoma, most often originating from the small intestinal tract of cats. This type of lymphoma is characterized by magenta cytoplasmic granules, however the cell size may be small, intermediate, or large. It’s important to note that these granules do not stain well with Diff-Quik and may be missed. This reinforces the importance of submitting at least one unstained smear to allow for methanol-based staining (e.g. Wright’s stain) in the diagnostic lab. This form of lymphoma is more often diagnosed on internal and not peripheral lymph nodes.

Another form of small cell lymphoma, known as T-zone lymphoma has a morphologic signature that can lead to a probable cytologic diagnosis. Remember the mantra “B is bad, T is terrible”? Well, not all T cell lymphomas are terrible. This form of T cell lymphoma has an indolent disease course and better prognosis than DLBCL. Cytologically this form of lymphoma is composed of a monomorphic population of paracortical T cells, which are referred to as “hand-mirror” cells based on their single small cytoplasmic projection / uropod (the handle of the mirror). Increased mitotic figures are not a feature. Note that these cells may also be increased in reactive conditions, therefore the mere presence of them does not equal a diagnosis of lymphoma. If this form of lymphoma is suspected, a CBC is recommended, as these dogs may also have a lymphocytosis. Interestingly in peripheral blood, this hand mirror morphology is not appreciated. In addition to evaluation of the sample by a clinical pathologist, flow cytometry (on a lymph node aspirate or peripheral blood if a lymphocytosis is present) or histopathologic examination (of an excised lymph node) may be pursued for confirmation. These lymphocytes have a classic aberrant phenotype on flow cytometry and histopathologic examination can be done to assess lymph node architecture to help reach a definitive diagnosis.

Lymph nodes are often aspirated as a part of staging to evaluate for the presence of metastasis. An interpretation of metastatic neoplasia is challenging with some tumors (e.g. mast cell tumor) and straight forward in others (e.g. squamous cell carcinoma). Few mast cells may be seen in aspirates of normal or reactive lymph nodes. Therefore when mast cell numbers are mildly increased in a dog with a mast cell tumor in the drainage area of the node, it can be difficult to be certain if there is truly lymph node metastasis or not. The more mast cells there are, particularly in groupings, the more confident you can be of metastasis. On the other hand, epithelial cells are never a normal component of lymph nodes. Therefore the presence of any epithelial cell population (as long it’s not just a skin contaminant) is considered a criteria of malignancy and indicates metastasis. Leukemias (chronic and acute) may also metastasize to lymph nodes.
Cytology is not recommended in the staging of chronic lymphocytic leukemia (unless of granular lymphocytes), since as previously stated, small lymphocytes normally predominate in lymph node aspirates. Acute leukemia in a lymph node aspirate can appear cytologically identical to a large cell lymphoma. This is why a CBC and clinical history are so important, and details of which should be provided to the clinical pathologist on the request form. The presence of a bicytopenia or pancytopenia and one or two enlarged lymph nodes raises the suspicion of an acute leukemia, whereas one or no cytopenias and generalized peripheral lymphadenopathy are more suggestive of lymphoma. In both cases, hemodilution should be kept to a minimum, as a heavily blood contaminated lymph node aspirate in a patient with leukemia would complicate evaluation for metastasis.
With clinical pathology testing, the phrase “garbage in, garbage out,” rings true. The pre-analytical phase of testing including sample collection, handling, and submission, and is critical to setting up a sample for success, and will be the focus of these notes. The analytical phase of testing occurs at either an in-clinic laboratory or an external diagnostic laboratory. If you are running an in-house laboratory, I would recommend you read an article I wrote with a colleague entitled, “Quality assurance and quality control in point-of-care testing” in the journal of Topics in Companion Animal Medicine. QA/QC is beyond the scope of this talk.

Hematology
When collecting a sample for hematology testing, it is important to try and minimize in vitro hemolysis. Good venipuncture technique (obviously patient dependent!) is a big part of it, but also avoiding small gauge needles and removing the needle and tube cap prior to transferring the sample to the tube can also help. Samples for hematology testing should be collected into an EDTA (purple top) tube. After quickly transferring the sample, gently invert the tube several times to ensure thorough sample contact with the anticoagulant. Unfortunately a clotted sample cannot be run through a hematology analyzer, as the counts will be falsely decreased to an unknown degree rendering the sample non-diagnostic. However, a blood smear made at the time of sample collection (prior to sample clotting) can be evaluated and provide a lot of information! (see proceedings on How to make and evaluate a blood smear…).

It is important to adequately fill the tube to the fill line or use a microtainer if only a small volume can be collected. If the tube is underfilled (also known as a “short sample”), the excess EDTA will cause an osmotic pull of water out of the RBCs resulting in cell shrinkage and thus a falsely decreased MCV. This will result in a falsely decreased spun PCV, as well as Hct, which is a calculated value based on the MCV and RBC count. The MCHC will also be falsely increased (due to the same amount of hemoglobin [Hgb] present in a smaller cell). Remember that a high MCHC is almost always an artifact, and should raise the suspicion of a pre-analytical error or interference, such as lipemia or hemolysis. In the scenario of a short sample, the Hgb and RBC counts are still reliable.

Once the sample is obtained, prompt submission (i.e. overnight shipping) is important to minimize in vitro storage changes that will affect both automated results and cell morphology on blood smear examination. With storage, RBCs take on water and swell resulting in a falsely increased MCV (and subsequently increased PCV and Hct) and falsely decreased MCHC. While platelet clumping is hard to avoid, it is exacerbated with in vitro storage. Even overnight, there are morphologic changes to cells, particularly neutrophils, that can affect interpretation, such as the ability to definitively determine the presence of a left shift. To overcome this, you can make a blood smear at the time of sample collection and submit this, along with the EDTA tube of blood. (see proceedings on How to make and evaluate a blood smear…).

Chemistry
Chemistry testing can be performed on either serum (red top / non-anticoagulant tube) or heparinized plasma (green top tube). It is imperative not to perform chemistry testing on plasma from an EDTA tube or contaminate your sample with EDTA. EDTA functions as an anticoagulant by chelating calcium. Obviously this will cause a falsely decreased calcium, but it also will chelate other divalent cations including magnesium and iron, resulting in a falsely decreased magnesium and iron, and falsely increased total iron binding capacity (TIBC). Most EDTA tubes also contain potassium (rarely sodium instead), which will also result in a falsely increased potassium.

After collecting your sample and putting it in the proper tube, you’re not done! Next, it is important to centrifuge the tube and separate off the serum or plasma. Failure to do so results in the RBCs continuing to metabolize and consume glucose, resulting in a falsely decreased glucose. Keep in mind that serum separator tubes (with the gel at the bottom) do not prevent this artifact. Contents of RBCs will also leak out resulting in a falsely increased AST, phosphate, and potassium in large animals and Japanese dog breeds (e.g. Akita) with high potassium RBCs.

Serum protein electrophoresis
This test is beneficial in the work-up of hyperglobulinemia to determine if there is a monoclonal or polyclonal gammopathy. A monoclonal gammopathy is typically due to a neoplastic process such as multiple myeloma or B cell lymphoma or leukemia, whereas a polyclonal gammopathy is a reactive response to antigenic stimulation.
It is important to submit serum (as the name implies) and not heparinized plasma, because fibrinogen (present in plasma and not serum) will appear as a sharp, narrow peak mimicking a monoclonal gammopathy. It typically falls in the beta region, therefore a sharp, narrow peak in this location would be suspect for a plasma submission.

Cytology
Cytology is a quick and minimally invasive diagnostic test that can sometimes provide a definitive diagnosis or at least narrow the list of differentials. However, non-diagnostic submissions do happen, and these outcomes are heavily dependent on the quality of the sample submitted. A clinical pathologist can’t make magic happen out of a slide with only blood, no cells, or all the cells smooshed up into a thick droplet. Keep in mind, that even a high quality sample may not be representative of the underlying pathology due to “sampling error.” For example, you may have aspirated an area of inflammation or necrosis adjacent to a tumor. This is why providing a complete description of the aspirated site and relevant clinical history are important. A history or description may raise red flags in the mind of the clinical pathologist that may make us suspicious that what we’re seeing may not be the whole story and we may recommend follow-up testing (i.e. surgical biopsy) to be sure. A blank request form limits the potential of your cytologic submission and our ability to provide an interpretation that fits clinically with your patient. Here are some tips when collecting and preparing your cytologic samples.

For fine needle aspirates of masses, it’s helpful to start with multiple slides laid out ready to go. If you have a particularly bloody aspirate, you can drip droplets of blood onto multiple slides prior to expelling the air in the syringe. This helps to prevent having one smear that is heavily hemodiluted and thick. Be sure to label the slides in pencil with the patient’s name and sampled site. This is particularly important when multiple sites on the same animal are being aspirated. You want to be sure the right sample goes with its corresponding location, especially if the results vary by site and surgical excision is recommended. Do this as you go, rather than at the end (or ask an assistant to label as you move on to the next site).

Sometimes a “stab” technique is preferred over the aspiration technique, in which a syringe is attached and negative pressure applied during the procedure. With the stab technique, you simply poke the lesion, keep the needle inserted in the mass and redirect multiple times while holding the hub of the needle. Afterwards, reattach the needle to a syringe pre-filled with several (~3cc) of air, then expel the contents onto a slide. This method works well for small lesions or enlarged lymph nodes, in which potentially neoplastic lymphocytes are fragile and prone to rupturing. The aspiration technique is often preferred for firmer masses, in which cells may be less likely to exfoliate.

Perhaps the most important aspect to set your FNA cytologic sample up for success is making a good quality smear. When expelling the contents of an aspirate onto the slide, orient the needle and syringe at about a 45 degree angle to the slide, face the bevel of the needle down towards the slide and have the tip of the needle, just in front of the frosted edge of the smear. After quickly and forcefully expelling the air from the syringe and spraying the contents of the aspirate onto the slide, you will have one or more droplets of material on the slide. Don’t stop here! Submitting a slide at this point is what we call “a splat smear.” These droplets are thick resulting in the cells being smooshed up together, often precluding, but most certainly hindering their evaluation. Cells will appear smaller. Cells can appear cohesive, when they may be in fact more individualized. To prevent this, take another slide, hold it above the first in a parallel direction, then gently place it on top of the slide and swiftly pull the two slides apart in opposite directions. This will spread the droplets, and sometimes causing them to coalesce into a single oval monolayer (depending on their size). This is often referred to as a “squash prep,” however this doesn’t mean you need to push hard with your spreader slide. Excessive pressure will rupture cells resulting in nuclear streaming.

Prior to sending your patient away or waking your patient up from sedation or anesthesia, take the time to stain and examine what you think is your “worst” slide. This allows you to ensure adequate cellularity of the sample (i.e. nucleated cells and not just blood), as well as aspiration of the desired tissue (e.g. lymph node, rather than perinodal fat). If you don’t have what you think is a diagnostic sample, this allows you to take another stab (pun intended) at your aspirate. Even without staining, you can often tell when you expel the contents of the aspirate, if there are “chunkier” pieces of tissue, rather than purely blood. It is important to submit at least one unstained smear, since Diff-Quik staining doesn’t stain the granules in mast cells and granular lymphocytes, well. In addition, Diff-Quik is not recommended for lymphoid tissue, because the staining of chromatin makes it challenging to differentiate mature from immature lymphocytes.
Here’s my tip on Diff-Quik staining – 10 dips in the first solution. Then for each of the second and third, dip the slide completely in and out until there is a solid film of the color across the whole slide. This prevents under and overstaining. Definitely don’t put the slides in the solution and walk away! Also be sure that prior to staining, the slide is sufficiently dry without water droplets on it. You can place a hair dryer on low setting underneath the slide to help rapidly air dry samples.

There are other methods for collecting cytologic samples including skin scrapes and swabs, when an FNA isn’t feasible. Keep in mind that you want to avoid getting ultrasound gel or lubricant onto the sample, as this material takes up stain and shows up as a purple granular material that obscures evaluation of the sample. Impression smears can also be made of surgical biopsy samples to provide a preliminary diagnosis, while waiting for histopathologic results.

For cytologic evaluation of urine (i.e. when concerned about transitional cell carcinoma), in addition to submitting urine in a red top tube for urinalysis, submitting a “line smear” made at the time of sample collection is recommended. A line smear is made in a similar manner as a blood smear, except instead of making a feathered edge, you stop abruptly with the spreader slide and lift it straight up, leaving a concentrated line of cellular material about ¾ across the slide. This smear can then be stained and evaluated. Even with making a fresh smear of urine, sometimes this is insufficient for making a diagnosis of TCC, as cells that sit in urine degrade making morphologic evaluation difficult. In this case, performing a traumatic catheterization is useful to get fresh cells to exfoliate from the mass.

If fluid is obtained when aspirating a mass, place the fluid into an EDTA tube. This also goes for all of your cytologic fluid needs, including body cavity effusions, BALs, tracheal washes, CSFs, and joint fluids. EDTA helps to preserve cellular morphology. If you’re suspecting an infectious etiology and want to culture, then an additional aliquot of the fluid should be placed into a red top tube. This is important, as EDTA is bacteriostatic interfering with culture results. After obtaining your samples, quick shipping (i.e. overnight) is then needed to limit in vitro cell aging, such as cell swelling. For fluid-filled masses, you should also perform an additional aspirate aiming for more solid portions of the lesion, such as the wall, as fluid in the masses is often poorly cellular and may contain few macrophages only. For fluid samples (except for CSF due to its inherent low cellularity), it is also recommended to make a direct squash smear prepared at the time of sample collection and submit this along with the fluid in the EDTA tube. This allows for the evaluation of fresh cells, as they were in vivo, and can help provide a more useful description and interpretation. For example, aged neutrophils with nuclear swelling mimic degenerate neutrophils. In addition, neutrophils and macrophages can also phagocytize objects (e.g. RBCs, bacteria) in vitro, which complicates the interpretation of hemorrhage and sepsis, respectively. When submitting fluids, do not concentrate the sample prior to submission. This will skew automated cell counts and confound the clinical pathologist’s interpretation of the cellularity of the sample when a direct smear is made. Concentrated smears are prepared for examination in the lab when fluid cell counts are low.

A helpful tip for making high quality bone marrow smears, is to expel the contents of the aspirate into a Petri dish filled with citrate phosphate dextrose (CPD) anticoagulant (used in blood banking products) then swirl the material in the Petri dish. Then take a dropper, aspirate the particles that you can see while avoiding taking up blood in the process, place a small drop onto a slide, and perform a squash prep as previously directed. This method helps to prevent excess hemodilution, which is a common problem when making bone marrow smears. And remember that a bone marrow aspirate should always be interpreted along with a CBC collected at the same day or at least within a day.

When shipping cytology slides, it is best to place them in a protective plastic container that will help prevent them from breaking during transit. Slides should not be refrigerated or allowed to come into contact with ice. In addition, do not ship cytology slides along with surgical biopsy samples, as the formalin fumes that leach out of containers (even when screwed on tight) will alter the staining properties of cells.

Hopefully these tips will prove useful for you as you return to work! Also remember, when in doubt, call the laboratory that you are submitting your sample to if you have questions about submission requirements.
Practice is busy – I totally understand! There’s not enough hours in the day to see your appointments, fit in those emergency walk-ins, and of course write-up your records, let alone make and look at blood smears. However, by the end of this, I hope I can convince that it is worth your while, especially in the work-up of a sick patient.

First on when and how to do it. It is important to make a blood smear on freshly collected blood (see proceedings on “How to get the most out of your clinical pathology samples…”), so make it a part of your routine at the time blood is collected. Ask your technicians to grab a few glass slides and microhematocrit tubes, along with the EDTA tube and venipuncture +/- catheter supplies. After blood is placed into an EDTA tube for a CBC, cap the tube, and gently invert it several times to ensure thorough mixing of the anticoagulant with the blood. Once the catheter is taped (if you’re placing one) or you’re done filling up the other necessary tubes, open and gently tip the EDTA tube to the side and fill your microhematocrit tubes. If you’re performing a PCV, fill an extra microhematocrit tube for the purposes of the blood smear. From now on, it’s important to move quickly, otherwise the blood will dry. Take your glass slide and your microhematocrit tube – tap the tube just in front of the frosted edge, in the center of the slide (width-wise). Hold on to that slide with your non-dominant hand pointer finger.

Take another glass slide in your dominant hand – hold it with your thumb on the side and your pointer finger halfway done the length of the slide in the center. Place the edge of this spreader slide towards the opposite end of the slide from where your blood drop is at about a 45 degree angle. Then swiftly pull this slide back into the blood droplet, pause for a second or two while the blood travels across the edge of the slide, and then push the spreader slide across the original slide. Your goal is to create a nice curved feathered edge (arrow) and monolayer (*). See image below:

Practice makes perfect when it comes to making blood smears, so keep at it! If you find your smear too short, decrease the angle of your spreader slide, whereas if the smear is too long and the blood is going off the edge of the slide, increase the angle of your spreader slide. Also, slow and shaky hands don’t make for good smears – so aim to be swift and confident!

Once you have your blood smear(s) made, let them air dry or you can place a blow dryer on the low setting underneath the slide to promote rapid drying. It’s important not to stain them before the smear is completely dry. In fact, submission of unstained smears is preferred when sending to a diagnostic laboratory. You can Diff-Quik the slides later and look at them when you get a chance. Speaking of – why bother looking at a blood smear? I’m glad you asked!

If you have a sick patient and your clinic doesn’t have a CBC analyzer, then a blood smear gives you preliminary answers while you’re awaiting results. If a PCV reveals an anemia, then a blood smear can be used to evaluate RBC morphology for evidence of regeneration and other changes to help determine the mechanism of anemia. With practice, you can also get a sense of a leukopenia or leukocytosis, evaluate for evidence of a left shift and/or toxic change to support inflammation, and spot abnormal circulating cells that may signal underlying neoplasia. A blood smear can also provide a platelet estimate, which can provide valuable information in the work-up of a bleeding patient. And every now and then, you may luck out and find an infectious agent, which can expedite a definitive diagnosis and therapy! Even if your clinic does have an automated analyzer, there are situations where nucleated cells can be misclassified such as nRBCs, blasts, or basophils.
As with a physical exam, I recommend examining a blood smear using a systematic, repeatable approach so you don’t miss anything. Starting at 10x, scan the feathered edge. You are looking for things like platelet clumps (which will falsely decrease an automated count), infectious agents (e.g. microfiliaria), or abnormal circulating cells (e.g. mast cells, histiocytes). Next, take about two turns of the microscope into the body of the smear in order to find the monolayer. The monolayer is where the RBCs have enough space to not be “smooshed” together (as in when you’re too far back) and are not distorted (or in “no man’s land” near the feathered edge). Here you can estimate the RBC and WBC density. In a normal animal, the RBCs should all be touching with minimal white space between them. Whereas with a mild anemia (Hct ~30s) there is a small amount of white space, a moderate anemia (Hct ~20s) has more white space, and in a severe anemia (Hct in the teens or below) there is a large amount of white space and RBCs are sparse. As for WBCs, think of your microscopic fields as a cookie with the WBCs as the chocolate chips. The more normal blood smears you look at, the better sense you’ll get for a normal WBC count. The goal here is not to guess the exact white count, but get the impression when there are increased chocolate chips (or a leukocytosis) or more of a sugar cookie (or a leukopenia). Next, go to 40x (high dry / no oil) or 50x (oil). Here is where you will evaluate WBCs. Don’t worry about performing an actual differential of 100 cells (that’s what we clinical pathologists are for) – you’re busy! Just scan around and get a sense of the proportions of cells. In domestic species, segmented neutrophils should predominate with fewer lymphocytes and monocytes, and rare eosinophils (if any) and no basophils. Look for band or further immature neutrophils, evidence of toxic change (cytoplasmic basophilia, Dohle bodies, frothy cytoplasmic vacuolization), and any cells you can’t identify / not sure what they are. The presence of immature neutrophils (a left shift) and toxic change indicates an inflammatory leukogram. If you’re seeing more bands than segmented neutrophils, this suggests a degenerative left shift, which indicates a more acute and severe inflammatory process. If you’re seeing cells you’re not sure what they are – then send the blood and smear to a clinical pathologist for review! They may be reactive or neoplastic. To get the best interpretive comment possible, be sure to include signalment and a clinical history on the request form. This allows the clinical pathologist to interpret the changes in the context of your patient.

After evaluating WBCs, next go to 100x (oil) to evaluate RBC morphology. (see proceedings for “You have an anemic patient, now what?”) and platelets. For a platelet estimate, you will get count the number of platelets in 10 different, adjacent fields, get an average number, and multiply that by 15,000/ul. And that’s that!
Infectious disease potpourri
Ashleigh Newman, VMD, DACVP

There are times when a blood smear review or cytology submission really pulls it out for the win! And by that I mean, gives you a definitive diagnosis and allows for early therapeutic intervention, particularly in cases with infectious etiologies. The following is a “potpourri” of infectious agents that can be identified on microscopic exam. I’ve mixed them up from the order, in which we’ll be discussing them – to keep it fun and the cases a mystery!

**Blastomyces dermatitidis**
When treatment with antibiotics isn’t resulting in improvement and/or a pyogranulomatous inflammatory pattern is observed, a fungal infection should be suspected. Cutaneous fungal infections with any of the agents discussed here are typically raised, proliferative, and ulcerative. *Blastomyces* can affect nearly any organ (e.g. lymph nodes, eyes, internal organs). The mnemonic “big, blue, broad-based budding” can help you remember the appearance of Blasto. The yeast form seen in cytologic specimens is spherical with a thick wall. They are larger than *Histoplasma*, smaller than *Coccidioides* spherules, and lack the negative staining capsule as is seen with *Cryptococcus*. *Blastomyces* is found primarily in the Great Lakes region and Mississippi and Ohio river valleys. In addition to cytology, the Blastomyces Antigen Test for dogs is useful for diagnosis, as well as monitoring during antifungal therapy.

**Mycobacterium**
*Mycobacterium* appear as short, thin, non-staining rods. They may be observed extracellularly or within macrophages. Cutaneous or subcutaneous nodular lesion(s) with a granulomatous inflammatory pattern and no obvious cause, should prompt careful searching at 100x (and submission to a clinical pathologist!). Acid-fast staining can be performed to highlight these organisms. These infections may spread to draining lymph nodes, as well as systemically, particularly in immunosuppressed animals. There are a variety of species, which are cytologically indistinguishable, and have variable zoonotic risks. PCR or MALDI-TOF may be performed for speciation.

**Filamentous beaded bacteria**
There are several species of bacteria that have a long, slender, filamentous and beaded appearance, including *Nocardia*, *Actinomyces*, and rarely strains of other bacterial species (e.g. *Fusobacterium*). The beaded appearance is due to the alternating pale blue and small pink or purple staining areas. These pathogens can cause cutaneous or subcutaneous lesions, as well as pyothorax. It is important to try to identify if the bacteria are *Nocardia* or *Actinomyces*, since these species often require very prolonged antibiotic treatment. Initial differentiation of *Nocardia* and *Actinomyces* from other filamentous bacteria can be performed through Gram staining. *Nocardia* and *Actinomyces* are both Gram positive, while other filamentous bacterial pathogens are Gram negative. Submission to a microbiology laboratory for aerobic and anaerobic culture is recommended, as well. It is important to give the lab a heads up of your differentials, as these pathogens require special culture conditions and are difficult to grow (potentially leading to a negative culture, despite infection).

**Cryptococcus**
Infections with *Cryptococcus* are more commonly observed in cats, particularly in subcutaneous, nodular lesions of the nose, as well as upper respiratory and central nervous systems. Organisms may be quite numerous, particularly in immunosuppressed animals, and can appear as many bubbles on low power view of a cytology smear. While the round yeast form with a non-staining capsule is most commonly seen, elongated (pseudohyphae or hyphae) forms may also be present. These are purple and the yeast display narrow-based budding, in contrast to *Blastomyces*.

**Ehrlichia / Anaplasma**
There are two species of ehrlichia that can be seen in granulocytes of dogs: *Anaplasma phagocytophilum* and *Ehrlichia ewingii*. The morulae from these species appear identical microscopically and are round to oval, purple, and located in the cytoplasm. While we see these mostly in thrombocytopenic horses in the northeast, they are occasionally identified in dogs.
Suspicious clinical signs include fever, thrombocytopenia, leukopenia, anemia, lymphadenopathy, splenomegaly, weight loss, and lameness. Keep in mind that a negative 4DX or other serology based tick-borne test does not rule out an infection, as these will be negative prior to seroconversion. In this acute period, these agents may be seen in circulation.

**Histoplasma capsulatum**

*Histoplasma* infections are primarily in the lung, however they can be systemic affecting multiple organ systems including skin, lymph nodes, CSF, and GI. Similar to the other fungal and higher bacterial agents described here, the cutaneous lesions are often raised, proliferative, and ulcerative. Organisms may be few or numerous. They are small, round, have a thin clear halo, and have an eccentric crescent-shaped, purple nucleus. This fungal infection is seen mostly in the Ohio River valley and lower Mississippi River. We rarely see it in the northeast.

**Sporothrix schenckii**

Fungal infections with this agent are rarely seen. They closely mimic the appearance of *Histoplasma* and are similar in size, however in contrast they can also be oval or cigar-shaped, in addition to round. In cats, organisms are typically numerous, whereas in dogs the organisms are rare / hard to find. The cutaneous lesions have a similar gross appearance to other fungal agents (raised, proliferative, ulcerative).
In the work-up of sick patients, a CBC will often reveal an anemia. Great, add one more problem to your problem list, right? In some cases, the anemia is a primary concern and warrants further diagnostics, in others, it may be secondary to the underlying disease process. In order to decide which it is, it helps to have a systemic approach to interpreting the mechanism(s) of an anemia, which will then help point towards potential causes. Blood smear examination is a helpful piece to this puzzle and in certain cases can provide clues as to the underlying mechanism or even provide a diagnosis. Note, that there may be (and often is) more than one mechanism contributing to an anemia.

First, it’s helpful to describe the anemia based on the severity (mild, moderate, severe) and the indices (i.e. MCV, MCHC). In small animals, a hematocrit in the 30s is typically considered a mild anemia, 20s is moderate, and teens or lower is severe. For example, is it a mild normocytic, normochromic anemia? Or a severe macrocytic, hypochromic anemia? Or a moderate microcytic, hypochromic anemia? This information can help prioritize mechanisms. For example, anemia of inflammatory / chronic disease alone would not explain a severe anemia (i.e. a hematocrit in the teens or below). And remember that an increased MCHC is 9.99 times out of 10 an artifact and signals a pre-analytical error or interference (see proceedings on Getting the most out of your clinical pathology testing…). A microcytic, hypochromic anemia usually points to one of two mechanisms: iron deficiency or a portosystemic shunt. Remember that young animals (< 2-3 months of age) can have a microcytic anemia due to age (attributed to a “physiologic” iron deficiency combined with rapid growth) and Japanese dog breeds (e.g. Akitas, Shiba) can normally have microcytic RBCs, but are not anemic.

Next after the indices, ask yourself – is the anemia regenerative or non-regenerative? A caveat to this is - if the history suggests an acute process (i.e. <3-5 days) then the bone marrow may not have mounted a regenerative response yet. Therefore, in this case if it appears non-regenerative, it’s best to re-check a CBC after 5 days to see if a regenerative response has manifested.

Regeneration in small animals is assessed by an increased degree of polychromasia (on a blood smear), which corresponds to the reticulocyte count on a CBC. Remember that we do not expect to see polychromasia in horses (or other equids). In this look for the presence of macrocytes (larger RBCs with a fully hemoglobinized cytoplasm) to suggest a bone marrow response. Polychromatophils are larger than mature RBCs and have a purple hue to them due to the combination of the red (from hemoglobin) and blue (from RNA content). This often results in a macrocytic hypochromic pattern. However, you should not solely rely on this pattern of indices to support regeneration, as this will also be seen with RBC swelling due to prolonged in vitro storage (as can be seen with mailed out samples).

On a CBC, a reticulocyte percentage, as well as an absolute reticulocyte count may be provided. The absolute reticulocyte count is a calculated value, which takes into account the severity of the anemia by multiplying the % retic by the RBC number. Therefore, the absolute reticulocyte count should be used to determine if an anemia is regenerative or not. There are situations in which the % retics may be increased above the reference interval, but the absolute reticulocyte count is not. This would be interpreted as a non-regenerative anemia. For example, 3% retics may be sufficient with a mild anemia and Hct 35%, but 3% retics would not be sufficient in the face of a severe anemia and Hct of 15%. The absolute reticulocyte count takes the severity of the anemia into account, whereas the % retics does not. Other changes that can be seen in the setting of a regenerative response are basophilic stippling, increased numbers of nucleated red blood cells, and Howell-Jolly bodies. Keep in mind, these morphologies can also be seen in different settings and signify pathology.

Mechanisms associated with regenerative anemias include the two H’s: hemorrhage and hemolysis. The best way to confirm hemorrhage as a mechanism for an anemia is by obtaining a thorough history (e.g. drugs, toxins, trauma, lack of parasite preventative) and finding a source of blood loss on physical exam (e.g. urinary, gastrointestinal, nasal tracts, or internal “loss” into a body cavity). With hemorrhage, the total protein is typically decreased characterized by a panhypoproteinemia, as proteins are lost as well. However, hemoconcentration (resulting in an increase in albumin) and inflammation/antigenic stimulation (resulting in an increase in globulins) may hide this. Also, the TP may not be low with internal hemorrhage. A severe thrombocytopenia (<30,000/ul) may be to blame due to spontaneous bleeding. In that case, look for petechiae and/or ecchymoses in hairless places on the skin.
Chronic external blood loss may lead to in iron deficiency, once iron stores in the body are depleted. Once this occurs, the low iron stores will dampen the regenerative response and it may even become non-regenerative. In addition to a low MCV and MCHC, visibly hypochromic RBCs on blood smear exam is further support of this.

Hemolytic anemias typically incite a stronger regenerative response than a hemorrhagic anemia. There are many causes of hemolysis. First classifying the hemolysis as intravascular and/or extravascular, helps to narrow down the list, as well as give prognostic information (intravascular carries a worse prognosis). Extravascular hemolysis is more common than intravascular hemolysis, and is the result of macrophages prematurely phagocytizing RBCs and presenting them to the liver for processing. This results in icteric plasma due to an increase in unconjugated bilirubin. Intravascular hemolysis is characterized by RBCs lysing within circulation releasing free hemoglobin. This results in hemoglobinemia (pink/red plasma) +/- hemoglobinuria (pink/red urine). Keep in mind, that if intravascular hemolysis is occurring, there is also extravascular hemolysis at play, therefore also hyperbilirubinemia.

Blood smear examination to evaluate RBC morphology is strongly recommended to further work-up a hemolytic anemia. Immune-mediated hemolytic anemia (IMHA) is certainly seen most commonly in the dog, and may be primary (idiopathic) or secondary (i.e. secondary to drugs, infectious agents, or neoplasia). Examples of infectious agents that result in hemolysis and can be seen on blood smear exam include Mycoplasma, Babesia, and Cytauxzoon. Hemophagocytic histiocytic sarcoma closely mimics extravascular IMHA and should be suspected in predisposed breeds (e.g. Bernese mountain dogs, Golden retrievers, Flat coated retrievers) with diffuse splenomegaly and concurrent hypoalbuminemia and hypocholesterolemia. Coombs’ testing is helpful in this case, as these dogs are usually negative.

Spherocytes can be confidently identified on blood smear exam in the dog, but not so in the cat due to their smaller MCV and lack of prominent central pallor. The presence of moderate to numerous spherocytes is supportive of IMHA due to extravascular hemolysis, however, keep in mind that spherocytes are not pathognomic for this. Low numbers of spherocytes, particularly when seen in a thrombocytopenic patient along with keratocytes, acanthocytes, and schistocytes, suggest fragmentation injury (a form of hemolytic anemia), typically secondary to DIC. Spherocytes also may be present as a result of a recent blood transfusion. Ghost cells suggest the concurrent presence of intravascular hemolysis. However, ghost cells can also be seen as a result of in vitro hemolysis (e.g. prolonged storage, collection-associated). In vitro hemolysis would also result in the plasma being assessed as hemolyzed. If there is doubt on the presence of in vivo intravascular hemolysis – evaluate the patient for hemoglobinuria. Certainly the presence of agglutination would signal immune-mediated targeting of RBCs. When not grossly visible, microscopic agglutination can be assessed for using the saline dispersion test. To do this, get an empty, plain tube and add one drop of anticoagulated blood, followed by four drops of saline. Mix well with an eyedropper, then place one drop of this mixture onto a slide, cover with a coverslip, and examine. Lower the condenser (as you would when evaluating an unstained urine sediment) and light to evaluate the cells in motion. Saline will disperse rouleaux resulting from hyperglobulinemia, but not agglutinated RBCs. You may need to dilute with more saline depending on the Hct and globulins.

Oxidative damage is another cause of hemolytic anemia, and blood smear exam is key for diagnosing this. The most well-known RBC morphology indicative of oxidative injury are Heinz bodies, which are the result of oxidized hemoglobin. The often-forgotten RBC morphology are eccentrocytes, which is due to oxidation of the RBC membrane. In the setting of these morphologic changes, keratocytes may also signal oxidative damage. It's important to note that non-anemic cats often have small ("endogenous") Heinz bodies, due to their hemoglobin's increased susceptibility to oxidant injury and their spleen’s inefficiency at removing them. Increased numbers of these small Heinz bodies are seen in cats with many diseases, particularly lymphoma, hyperthyroidism and diabetes mellitus. Although these cats are often not anemic, their RBC lifespan is reduced. This also brings up the concept that not all RBC changes indicate the cause of an anemia. An anemia in a cat should not be solely attributed to oxidant injury if only small endogenous Heinz bodies are seen.

Likewise, if only low numbers of Heinz bodies, eccentrocytes, and/or keratocytes are seen in an anemic animal, they are unlikely to be the cause of the anemia. All they mean is that there is oxidant injury to RBCs and, even though affected RBCs have decreased lifespan, it does not mean the animal has an oxidant-induced hemolytic anemia. It is more likely that the underlying disease responsible for the oxidative injury is also causing the anemia (which is often due to bone marrow suppression from inflammatory disease).
When we see moderate to high numbers of these RBC abnormalities (or very large or multiple Heinz bodies in cats) then we think about attributing an anemia solely to oxidative injury. Some examples of oxidants inciting a hemolytic anemia include acetaminophen, onions, garlic, red maple, kale, zinc (pennies post-1982), benzocaine, vitamin K, and even skunk musk.

These are certainly the most common causes of regenerative anemias. There are the zebras out there – so when you’re left scratching your head in a young animal, think about hereditary defects.

Moving on to non-regenerative anemias. This is a result of decreased RBC production, which can be due to a multitude of causes. Sorry, no quick mnemonic here! The major mechanisms for non-regenerative anemias include:

- Decreased erythropoietin
- Suppression of erythropoiesis
- Deficiency of minerals, vitamins, or nutrients
- Defective hemoglobin synthesis
- Defective DNA synthesis or nuclear maturation
- Destruction of hematopoietic cells in the bone marrow
- Myelophthisis (crowding out) of normal hematopoietic cells and altered marrow environment

While decreased production may be due to bone marrow disease (more likely with a severe anemia), it may be an extramedullary disease process resulting in suppression of erythropoiesis (more often the case with a mild to moderate anemia). Therefore, a chemistry panel is a crucial part of the work-up to evaluate for systemic or endocrine disease (e.g. chronic kidney disease, hypothyroidism, Addison’s). The presence of a bicytopenia or pancytopenia should raise the suspicion of primary bone marrow disease, and a bone marrow aspirate should be considered, particularly if abnormal cells are seen in circulation and there is no other explanation for the anemia. In this setting, the cytopenias are typically due to myelophthisis (“crowding out” of normal hematopoietic cells) due to primary bone marrow neoplasia (e.g. acute leukemia) or metastatic neoplasia (e.g. lymphoma infiltrate). Obtaining a thorough drug history is important, as some drugs can directly result in decreased RBC production (e.g. TMS, albendazole, griseofulvin) and may affect more than one cell line (e.g. phenobarbital)

The most common cause of a mild to moderate, normocytic, normochromic, non-regenerative anemia is anemia of inflammatory disease, which used to be called anemia of chronic disease. This is not to be confused with anemia of chronic kidney disease, which is mainly attributed to decreased erythropoietin. There are no tell-tale RBC morphologic changes to support anemia of inflammatory disease. Rather, the concurrent presence of an inflammatory leukogram, is supportive. This anemia is associated with various disorders (e.g. cancer, liver, gastrointestinal disease). The main mechanisms are inflammatory cytokine-mediated suppression of erythropoiesis and decreased RBC lifespan. The main mechanism of suppression is sequestration of iron, which usually doesn’t manifest until inflammation is chronic. However, decreased RBC lifespan due to inflammatory cytokines and oxidant injury can occur with acute inflammation, as a result of premature phagocytosis (i.e. extravascular hemolysis).

While IMHA is typically regenerative, immune-mediated destruction of early precursors in the bone marrow will result in a non-regenerative anemia. When the earliest of precursors is targeted, this results in what is termed “pure red cell aplasia” and a severe anemia. Therefore immune-mediated anemia can be thought of as a spectrum. As with a regenerative IMHA, precursor targeted immune-mediated anemias may be primary or secondary. In cats, FeLV is certainly on the list of potential underlying causes.

A macrocytic, non-regenerative anemia usually indicates a problem with DNA maturation and can occur with dietary deficiency (vitamin B12, folate, cobalt) or excess (e.g. molybdenum), drugs, infections (FeLV), neoplasia (e.g. erythroleukemia, myelodysplastic syndrome), and immune-mediated and inherited disorders. Some breeds of dogs, e.g. miniature and toy poodles, can normally have macrocytic RBCs, but are not anemic.

Microcytic anemias usually indicate a problem with hemoglobin synthesis. This is mostly due to iron deficiency from chronic external blood loss, but can also be caused by other deficiencies (copper, vitamin B6) or excess (e.g. zinc), drugs, portosystemic shunts (not all animals with shunts will be anemic but they are frequently microcytic), and, rarely, immune-mediated and inherited disorders.
While I can’t go into everything in depth, hopefully this approach will help you navigate your next anemic patient. Also, when in doubt, send out to a clinical pathologist for a blood smear review! And don’t forget to include a history and any pertinent diagnostic abnormalities. Depending on the lab, you will typically get a more helpful comment tailored to your case, if we have background information on the patient. I also recommend checking out Eclinpath.com, which is a great reference and a free website out of Cornell University!
Minimizing Antimicrobial Resistance in Dogs and Cats: Do We Have a Problem?
Dawn Merton Boothe, DVM, PhD, DACVIM, DACVCP
Auburn University, AL USA

Introduction

“It is unwise to underestimate an adversary that has had a three billion year evolutionary head start” (Sayers, 2004). Attentiveness to emerging antimicrobial resistance is supported by the World Health Organization’s statistics indicating the sobering percent of resistance to first choice antimicrobials (generally fluoroquinolones, 3rd generation cephalosporins or carbapenems) currently present to over 50% of selected coliforms or gram positive organisms in partner hospitals. The American Veterinary Medical Association has promulgated through its member associations judicial antimicrobial use guidelines and the American Association of Veterinary Medical Colleges has partnered with the American Association of Public Land Grant Universities to promulgate Antimicrobial Drug Resistance learning outcomes targeting highschool, undergraduate and veterinary students. Most sobering: the Center for Disease Control indicates that the greatest risk factor for antimicrobial resistance is use.

Antimicrobial Resistance Patterns

Gram Negative: The advent of antimicrobial resistance is increasingly limiting therapeutic options in human and resistance to an antimicrobial varies with the species and strain. Among the most adaptable organisms is E. coli. Discovered in 1885 by a the diatrician Theodore Escherich, it was originally discovered in neonatal fecal samples. Dr. Erich recognized E. coli was acquired at birth and remained with us till death, with strains coming and going. It is the most thoroughly understood microbe, and is critically important as a research tool. Through E. coli, we have come to understand such diverse activities as intermediary metabolism, DNA replication and RNA transcription, protein synthesis and genetic recombination. Indeed, recombinant products would not be possible without E. coli. Escherichia coli, a member of the family Enterobacteriaceae, is a lactose fermenter, causing a distinct color on diagnostic agar. It is the predominant facultative anaerobe (in the normal intestine of both humans and many warm-blooded animals), playing a major role as normal microflora.12 However, it also is ubiquitous in the environment, as is recognized by its appearance as contaminants in food stuffs. It has or acquires genes that encode for flagella, making it mobile. Its presence in the environment is used as a sentinel of environmental contamination. Referred to as the “cockroach” of microbes because of its adaptability, E. coli rapidly divides, potentially doubling its population every 20 minutes. Further, it is highly mutagenic, with spontaneous mutations occuring in of 1 per 100 thousand to 1 per billion new progeny (assume 1 gm of feces contains 100 million E coli) thus assuring opportunity for spontaneous mutation even in the absence of stimuli, such as drugs. Resistance. The gastrointestinal environment is conducive to development of resistance. Environmental microbes maintain an ecological niche by suppressing competition through secretion of antibiotics. As such, commensal organisms are constantly being exposed to antibiotics. However, the microbe producing the antibiotic, as well as surrounding normal flora, are resistant to the antibiotic. Thus, genes for resistance develop along with genes directing antibiotic production and organisms are “primed” to develop resistance. Microflora of the GI tract can serve as reservoir of resistance genes. Exposure to antimicrobials may facilitate survival of isolates that have either spontaneously mutated or acquired resistant through other means. Resistance may be easily conferred to other potentially more virulent organisms.

E. coli rapidly develops resistance, particularly that associated with multiple drug resistance (MDR) when exposed to selected antimicrobials. E. coli develops resistance both vertically and horizontally. Shared resistance reflects the ability of bacteria to incorporate extrachromosomal DNA carrying the information for
resistance from other (including non-self) organisms. Extrachromosomal DNA (including plasmids and bacteriophages) encode for resistance to multiple drugs and can be transmitted vertically (to progeny) or horizontally, across species and genera. In general, resistance carried by plasmids “comes and goes”, meaning the presence of the drug may increase the likelihood of the plasmid being present, in large copy numbers; removal of the drug may be associated with resolution of the resistance. More disconcerting, resistance is easily conferred to more pathogenic organisms. In human medicine, *E. coli* has developed resistance to the fluorinated quinolones, beta-lactams, or both: it is among the Gram negative organisms that secrete extended spectrum beta-lactamas (ESBL). Emergence of extended spectrum extended spectrum beta-lactamas (ESBL) is an example of the relentless adaptive nature of microbes toward designer drugs intended to preclude the advent of resistance. The ESBLs are encoded by large plasmids that can confer the information between strains as well as different species of organisms. The gene mutation confers resistance to newer cephalosporins including cefotaxime, ceftazidime, and ceftriaxone, as well as cefpodoxime, or 4th generation including cefepime (no longer marketed in the USA); cefpime has been cited as possibly being effective against ESBL. The impact on clavulanic acid and sulbactam is not clear, although their use in place of cephalosporins appears to reduce the emergence of ESBL and may reduce the emergence of other resistant pathogens such as *Clostridium difficile* and vancomycin-resistant enterococci. The ESBL are most commonly found in *Klebsiella* spp. *E. coli* or *Proteus mirabilis* (3.1-9.5%), but they also have been detected in other members of the Enterobacteriaceae and in *Pseudomonas aeruginosa*. Resistance to fluoroquinolones has also been well characterized. Normally associated with point mutations in topoisomerases (DNA Gyrase and Topoisomerase IV), such resistance is, like beta-lactamas, within class. However, in the presence of continued drug, efflux pumps appear to be induced. Such pumps serve to remove toxic compounds from the organism, including antimicrobials. At least 5 efflux pump systems have been characterized; they are associated with porins. They are characterized by broad substrate specificity, thus can impart multidrug resistance. The the culture (above) and the antibiogram below typify the patterns of resistance that can emerge in animals which have received fluoroquinolones and in which resistance has emerged. *E coli* resistance in isolates associated with urinary tract infections is particularly well described. One study demonstrated the gastrointestinal emergence of quinolone -resistant *E. coli* genetically distinct from infection-causing strains in patients treated with ciprofloxacin. Like virulence factors, transfer of resistance genes in isolates between animals in humans may present a public health risk, as was recognized by the FDA by 1999. We have demonstrated the impact of *E. coli* MDR through several of our studies. In healthy normal dogs, we have demonstrated the impact of 10 mg/kg amoxicillin bid and 5 mg/kg enrofloxacin once daily orally for 7 days on fecal E. coli. For either drug, 100% of E coli became resistant to the drug, expressing high level (more than 8 times the MIC breakpoint). For amoxicillin, this resistance was limited to beta lactams and occasionally tetracyclines or sulfonamides;

### Table 1. The percent of *E. coli* feline and canine uropathogens from throughout the US (2009-2012) resistant to antimicrobial drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Overall (n=1612)</th>
<th>95% CI</th>
<th>Species</th>
<th>Gender</th>
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<tr>
<td></td>
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<td>Canine</td>
<td>Feline</td>
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<td></td>
<td></td>
<td></td>
<td>Female</td>
<td>Male</td>
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<tr>
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<td>39.1-44.0</td>
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<td>46.6-61.6</td>
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<td>37.6</td>
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<td>7.7-11.0</td>
<td>10.5</td>
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<td>7.1-9.9</td>
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<td>11.1-14.5</td>
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<td>7.9</td>
<td>6.6-9.3</td>
<td>8.56</td>
<td>6.0</td>
</tr>
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<td>1.9</td>
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<td>17.5-21.4</td>
<td>20.79</td>
<td>15.3</td>
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<tr>
<td>TMS</td>
<td>8.9</td>
<td>7.4-10.3</td>
<td>9.69</td>
<td>6.3</td>
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resistance tended to resolve by the 3 week study end period. Enrofloxacin resistance however, not only was multidrug resistance, but tended to persist. The relationship between enrofloxacin resistance and multidrug resistance was also demonstrate din a pilot surveillance study of approximately 400 *E. coli* pathogens collected form dogs or cats. The pattern of resistance varied regionally, being as much as 50% to amoxicillin or amoxicillin clavulanic acid and in the south, approximately 30% to enrofloxacin. Although the number of isolates resistant to beta lactams only (expressing single drug resistance) was high, single drug resistance to enrofloxacin was rare. If resistance was expressed to enrofloxacin, it was multidrug in nature. We have an ongoing study involving 3000 isolates throughout the United States sponsored by the Morris Animal Foundation and Idexx laboratories. Currently, regional differences in resistance continue to persist. Overall resistance is greatest to cephalixin (9%) as is demonstrated in Table 1; 100% of isolates are also resistant to doxycycline using the new interpretive criteria established by CLSI. Further, 40% are resistant to amoxicillin (amoxicillin). This latter statistic suggests that for treatment of *E. coli*, Clavamox® may not have that much advantage over amoxicillin along and this would also be true if treating *Enterococcus*. However, if treating other organisms, protection against beta-lactamases may be helpful.

**Gram Positive Organisms:** Methicillin resistance (MRSA; S. aureus; MRSP; S. intermedium [pseudintermedius] Multidrug resistance is now considered the normal response to antibiotics for Gram positive cocci pneumococci, enterococci and staphylococci. Methicillin resistance (MRSA; S. aureus; MRSP; S. intermedium [pseudintermedius] is indicated by the presence of the meca gene, which encodes a mutation in penicillin binding protein (PBP) resulting in formation of PBP2a rather than PBP2. As such, affinity is reduced for the beta-lactam ring, rendering the organism resistant to all beta-lactams. Protectors such as clavulanic acid are ineffective. Detection of MRSA or MRSP on C&S generally is based on resistance to oxacillin, which is more stable than methicillin in disks used for testing. However, increasingly, laboratories are indicating MRS based on absence of susceptibility to any beta-lactam. In our hospital, approximately 25 to 30% of Staphylococcus pseudintermedius express methicillin resistance. Antibiotics and especially cephalosporins are associated with induction, selection, and propagation of MRSA. MRSA in human patients has evolved from a hospital-acquired (HA-MRSA; nosocomial), in which occurs most commonly in patients immunocompromised by disease, drugs, procedures and duration of hospitalization, to a community acquired infection (CA-MRSA), in which otherwise healthy persons are infected, usually in the skin or soft tissue. Crowded conditions, shared items and poor hygiene increase the risk of community acquired infection. Although it is community acquired MRSA strain USA300 that appears to be most commonly associated with increased colonization in dogs and cats, it is USA100, most commonly associated with hospital acquired-MRSA infections in humans, that most commonly is associated with infections in dogs and cats animals. The impact of MRSA in veterinary medicine is increasingly problematic, not only because of its impact on the patient, but the public health considerations. The meca gene has been detected in methicillin-resistant Staphylococcus aureus organisms infecting dogs and MRSA has been associated with infection in dogs. However, MRSA also has been found in up to 4% of healthy dogs, with identification complicated by the need for multiple sampling sites (nasal and rectal or perineal). Infections have been isolated in family members and pets in the same household, but this is likely to reflect transmission from humans to the pet. It is likely that colonization is transient in animals. However, healthy pets have been demonstrated to be potential reservoirs for transmission of MRSA to healthy handlers and a potential health risk to immune-compromised patients (human and presumably other animals in the household). Human colonization with MRSP is unusual. However, MRSP has been reported as a cause of infection in human patients and transmission from pets with pyoderma has been confirmed. It is the very immunocompromised patient that is at risk for MRSA infection acquired from an animal. In such cases, the carrier or infected animal should be removed from the environment until successfully treated for methicillin-resistant Staphylococcus.
Glycopeptides such as vancomycin are the initial drugs used to treat MRSA in humans, although increasingly vancomycin resistant staph infections (VRSA) have emerged.

**Reducing Resistance: The Three “D”s.**

Regardless of the organism, the most significant mechanism by which bacterial resistance is likely to be reduced is implementation of behaviors that are designed to reduce patient risk such as length of hospital stay, and design, implementation of and adherence to infection control practices. Consider the three DE-s:

1. **DESCALATE.** Because previous antimicrobial therapy is one of the most important factors associated with resistance, approaches which minimize indiscriminant antimicrobial use will be important. Examples of human strategies include improving appropriate antimicrobial use (eg, including less ideal strategies such as strict adherence to prescribed formularies, setting limits on the duration of antimicrobial therapy); potentially reasonable strategies such as requiring prior approval for use of certain antibiotics such that proper use can be verified; and more rationale strategies such as narrowing the spectrum of empiric antibiotics, and rotating the use of antimicrobial drugs on a regular schedule); primary prevention by decreasing length of hospital stay, decreased use of invasive devices, and newer approaches such as selective digestive decontamination and vaccine development. Probably the single most important first step in judicious antimicrobial use and avoiding resistance is questioning/confirming the need for therapy. This is no small task, being fraught with the lack of effective diagnostic aids. Probably the most common— and least correct mindset is failure to recognize that we are in conflict with our directive of “above all else do no harm” if we use antimicrobials in the absence of infection. De-escalation includes taking actions that stay the hand in reaching for drugs if they are not really necessary. For urinary tract infections, increasingly the need for treating asymptomatic bacteria is questioned. What constitutes an infection may not depend only on the inoculum size (e.g., 1000 CFU/ml) but the presence of clinical signs.

2. **DESIGN.** Dosing regimens should be designed to assure that adequate drug concentrations are reached at the site of infection to kill, not simply inhibit, microbial growth. DEAD BUGS DON’T MUTATE! Once the decision to use the antimicrobial is made, efforts should focus selecting a drug to which the bug is most susceptible. A practice-based antiogram (see above) may be helpful. If an animal has not been exposed to antimicrobials, the chances are improved that any infecting pathogens are among the susceptible isolates. A narrow spectrum is preferred to a broad spectrum drug. Once the drug is chosen, design focuses on the dosing regimen to assure that concentrations adequate to kill the infecting microbe are achieved at the site of infection. This involves not only selecting the drug to which the isolate is most susceptible (and the drug most likely to reach the target site), but also designing the dosing regimen based on the MIC, potentially the MPC and time or concentration dependency of the drug (see parts II and III if relevant). An antiogram (see figure; top number is % susceptible, bottom number of each cell is number tested) generated for each practice can support empirical selection of antimicrobial drugs although increasingly, culture is indicated in all but the antimicrobial naïve patient (this includes direct - or indirect through a household member - exposure). (Note that squares without information are drugs which should not be tested toward that bug).

3. **DECONTAMINATE:** Hospital strategies include: improving infection control (eg, selective decontamination procedures, prevention of horizontal transmission via handwashing, use of disinfectants, glove and gown use, alternatives to soap, and improving the workload and facilities for health care workers), and identification of specific areas for treatment of potentially infectious agents (ie, bandaging areas that can be easily cleaned). Increasingly “detergent” should be applied to the patient and its home. For example, recurrent infections might be reduced if successful initial therapy is coupled with cleansing of the environment in which the pet is located such that it is not continued to be exposed to the infecting bug. For UTI infections, this may become particularly important in that urine contaminates the environment.
MINIMIZING ANTIMICROBIAL RESISTANCE PART II: INTERPRETING CULTURE AND SUSCEPTIBILITY DATA
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Auburn University, AL USA boothdm@auburn.edu

Introduction

For all infections, but particularly complicated infections, or in patients at risk for therapeutic failure or developing resistance, antimicrobial decision making should be guided by the three D’s: de-escalate antimicrobial use, design a proper dosing regimen, and decontaminate such that the spread of infection is minimized. This session will focus on the design of the dosing regimen. Once the decision to treat with systemic antimicrobials is made, the design of the dosing regimen includes not only selecting the best drug to target the infecting pathogen, but designing the most effective dosing regimen that minimizes emergent resistance. Among the most effective tools to support design of the dosing regimen is culture and susceptibility data. Clearly, patient data is most useful, but population culture and susceptibility data can support empirical therapeutic decision making. Identification of the infecting organism most appropriately should be based on appropriately collected cultures with (ideally) tube dilution susceptibility testing to allow assessment of “how” susceptible the isolate is, in general, to other drugs, but specifically to the drug of choice, such that a dosing regimen can be designed for the bug in the patient. Basing antimicrobial selection on C&S data does not guarantee success, just as failing to use C&S as a basis for selection (or selecting a drug characterized by “R” on the data) does not guarantee failure. Further, there are many pitfalls to C&S testing, including mis or over interpretation.

1. The data is only as good as sample collection, handling and testing. Just as absence of growth does not indicate absence of infection, isolation of an organism is not necessarily evidence of infection, nor even if infection is present, does the isolated organism represent the infecting organism. One of the biggest challenges is making sure that the cultured organism is the pathogen causing harm to the patient. Pure, vibrant (meaning special media was not needed to coax the growth of the organism) of a large number of colonies are indicators of infection. The isolation of three or more different organisms (including more than one strain of the same organism) may indicated contamination and reculture should be considered. As such, the method of sample collection is critical and must minimize contamination while optimizing pathogen growth. Clearly, (properly collected) culture of an organism from a tissue that is normally sterile indicates infection. Organisms that do not make sense (eg, Bacillus; an anaerobe from the urinary tract, etc) might indicate questionable data. For urine, cystocentesis. For tissues, as convenient as swabs can be, they increase the risk of contamination with commensals if the site is not properly prepared, are inhibitory to microbe growth, and organisms in the depth of the swab may not be cultured. They also preclude colony counts which otherwise not only help confirm pathogenicity, but give an indication of extent of growth, which in turn, should impact the dosing regimen (more bugs=more drug molecules. The extent of growth should be strongly considered when deciding to treat. Generally, clinical signs of infection may require, depending on the site, >10⁶ CFU/ml (or g tissue). For C&S purposes, quantitative cultures can be helpful: the urinary tract is not considered infected until >10⁵ CFU /ml are present whereas only > 10³ is indicative of infection in the respiratory tract. Another reason that samples must be handled properly is exemplified with UTI: with an E. coli doubling time as short as 20 minutes, a small colony count indicative of no infection can rapidly become a high colony count (>10(5) CFU/ml) indicative of infection. Use of “urinary” paddles might be considered for samples that are being shipped in order to increase accuracy of identification and numbers. Laboratories may also indicate “heavy, moderate or light” growth; isolates with the greatest amount growth might be targeted if all cannot be. Regarding the laboratory, no mandated accreditation process assures the quality of
the data. A veterinary microbiologist should be available to answer questions; veterinary interpretive criteria (breakpoints) generated by the Clinical Laboratory Standards Institute (CLSI) must be followed. A call to the diagnostic lab might be prudent before marked financial commitment is put into treating an organism that is not causing infection.

2. To wait or not to wait? Frequently, antimicrobial therapy is begun before cultures are collected. This is particularly true and appropriate in critical patients or in patients for which clinical signs are evident and are detracting from quality of life (patient or parent). However, should therapy begin and the choice prove to be wrong once C&S data is received, the original C&S data collected before the drug was begun may no longer be relevant to the patient. The use of the drug may change the pattern of resistance versus susceptibility, or may result in higher MIC (see mutant prevention concentration). If the patient has responded, no change is indicated. If not, a reculture may be indicated at that time, if possible. Certainly, dosing regimens with the appropriate drug should take into account the possibility that some level of resistance has developed toward the indicated drug.

3. In vitro to In vivo: Some of the Pitfalls. The C&S procedures themselves are fraught with potential errors. For practices that provide in- house susceptibility testing, care must be taken to follow guidelines established and published by (or comparable to) the Clinical and Laboratory Standards Institute (CLSI) or comparable standard-setting agency. Materials, including interpretive standards, should be validated by the appropriate agency. As such, in-house susceptibility testing should be considered with caution. Minor changes in pH, temperature, humidity, etc can profoundly affect results. Personnel should be trained specifically in culture techniques and hospitals that provide this service (as do diagnostic labs) should maintain well designed and adequately collected quality control data to validate their procedures (CLSI indicates control organisms). Pitfalls of susceptibility testing also reflect the drugs selected for testing. Not all companies are interested in establishing interpretive criteria and as such, not all drugs are available for testing. Because automated systems can not accommodate and laboratories (nor clients) can not afford to test all potential drugs used to treat an infection, one drug often is tested as a model for other drugs in the class. For example, cephalothin models first generation cephalosporins, even though it is no longer used clinically. Note that it does not represent cefazolin well, the latter being more effective toward Gram negative (especially *E. coli*) isolates. No single cephalosporin can represent 2nd or beyond generations. Enrofloxacin often represents the fluoroquinolones. In general, cross resistance can be expected among the FQs, although differences in potency do exist (for example, ciprofloxacin is more potent toward *Pseudomonas* or *E. coli*, but less to Gram positives compared to enrofloxacin).

Culture does not take into account active metabolites of some drugs (eg, enrofloxacin converted to ciprofloxacin). Note that if an organism is R to any FQ, FQ should be used only cautiously even if another is “S”. Amikacin is often more effective than gentamicin toward many organisms, but less effective toward *Staphylococcus* sp. (hence both are often on a report). Note that CLSI interpretive criteria are generated for specific species, and often for specific organisms and specific infections. Human laboratories will use human interpretive criteria, which often are not relevant to animals (eg, ciprofloxacin).

Ciprofloxacin (CIP) oral bioavailability in dogs is 30 to 40% of that in humans, and despite its increased potency compared to enrofloxacin (ENR) toward Gram negative organisms, its potential efficacy
CIP: both $C_{\text{max}}$ and area under the curve (AUC) of bioactivity of ENR may increase up to 50% or more by CIP; as such, C&S data may underestimate efficacy. MIC$_{\text{BP}}$ generally are based on the highest labeled dose (eg. 20 mg/kg for enrofloxacin, 13 mg/kg q 8 hrs for amoxicillin for canine UTI), but higher doses might be safely administered for many antibiotics. If recommended doses change, the manufacturer should provide CLSI with updated pharmacokinetic information so that interpretive criteria may change. One of the disadvantages of current susceptibility testing is that the concentrations tested are close to the MIC$_{\text{BP}}$ and thus, does not allow identification of isolates that are very susceptible (that is, MIC are far away from the MIC$_{\text{BP}}$). A final concern relates to the 3rd and 4th generation (extended spectrum) cephaplosporins: they are susceptible to extended spectrum beta-lactamase (ESBL) that tend to be induced in vivo but often missed in vitro. If CLSI guidelines are followed, resistance to cefpodoxime indicates ESBL being produced, If CLSI guidelines are not followed, therapeutic failure may occur. Carbepenems and clavulanic acid (eg, amoxicillin-clavulanic acid) generally are not susceptible to these enzymes.

**BRIDGING PHARMACODYNAMIC (PD) AND PHARMACOKINETIC (PK) DATA**

Agar gel diffusion data, which is the only data available for organisms that do not grow rapidly enough, only provides a “yes” (S) or “no” (R) (and sometimes “maybe” [I]) which is helpful, but not nearly as much as tube dilution data. An example of population data is the 2016 Cumulative Antimicrobial Susceptible Report which is a simple yes vs no for agar gel and tube dilution data (the number of isolates tested is in the first column; the percent of those isolates susceptible to each drug is in the appropriate cell). On the other hand, although tube dilution data does provide S, I, or R information, along with quantitative data (the MIC, or Minimum, Inhibitory Concentration), it is difficult to interpret. Simplistically, the MIC

(MIC$_{\text{BP}}$) is equivalent to or less for many organisms. Susceptibility data also does not take into account active metabolites, again exemplified by ENR, which is metabolized to

<table>
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<tr>
<th>Drug</th>
<th>MIC BP S</th>
<th>MIC BP R</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Amoxicillin</td>
<td>16</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>25.5</td>
<td>≥ 0.5</td>
</tr>
<tr>
<td>*Amoxicillin with clavulanic acid</td>
<td>≤ 4/2</td>
<td>≥ 8/4</td>
</tr>
<tr>
<td>*Ampicillin</td>
<td>31.25</td>
<td>≥ 32/16</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Carbenicillin</td>
<td>16</td>
<td>≤ 64</td>
</tr>
<tr>
<td>*Cefazolin</td>
<td>8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Cefoxaxone</td>
<td>8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Cefotaxim</td>
<td>12.5</td>
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</tr>
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<td>Cefotaximil</td>
<td>2</td>
<td>≥ 8</td>
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<td>Ceftriaxone</td>
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<td>≥ 32</td>
</tr>
<tr>
<td>Cefuroxone</td>
<td>8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>*Cefapenil</td>
<td>8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Cephalaxin</td>
<td>8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>*Chloramphenicol</td>
<td>8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Ciprofloxacin (≤ 1)</td>
<td>8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Clarithromycin</td>
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<td>8</td>
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<tr>
<td>*Clindamycin</td>
<td>0.5</td>
<td>≥ 4</td>
</tr>
<tr>
<td>*Doxiflaxacin</td>
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<td>≥ 4</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>4</td>
<td>≥ 4</td>
</tr>
<tr>
<td>*Enrofloxacin</td>
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<td>≥ 4</td>
</tr>
<tr>
<td>Erythromycin</td>
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<td>8</td>
</tr>
<tr>
<td>*Fioronicol</td>
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<td>≥ 4</td>
</tr>
<tr>
<td>Gentamicin†</td>
<td>4</td>
<td>≥ 16</td>
</tr>
<tr>
<td>*Impenem/cilastatin</td>
<td>4</td>
<td>≥ 16</td>
</tr>
<tr>
<td>Kanamycin†</td>
<td>1</td>
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</tr>
<tr>
<td>*Lincomycin†</td>
<td>0.5</td>
<td>≥ 4</td>
</tr>
<tr>
<td>Marbofloxacin</td>
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<td>≥ 4</td>
</tr>
<tr>
<td>Meropenem</td>
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<tr>
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</tr>
<tr>
<td>Nitrofurantion</td>
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<td>≥ 128</td>
</tr>
<tr>
<td>*Orbifloxacin</td>
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<td>8</td>
</tr>
<tr>
<td>*Oxolinic</td>
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<td>≥ 4</td>
</tr>
<tr>
<td>Penicillin G</td>
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</tr>
<tr>
<td>Pyperacillin</td>
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<td>≥ 128</td>
</tr>
<tr>
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<td>≥ 4</td>
</tr>
<tr>
<td>Sulfadiazine</td>
<td>≤ 2</td>
<td>≥ 4</td>
</tr>
<tr>
<td>*Tetracycline</td>
<td>≤ 4</td>
<td>≥ 16</td>
</tr>
<tr>
<td>*Ticarcillin</td>
<td>64/128</td>
<td>≥ 128</td>
</tr>
<tr>
<td>*Ticarcillin with clavulanic acid</td>
<td>128/254</td>
<td>≥ 128/2</td>
</tr>
<tr>
<td>*Trimethoprim/</td>
<td>≤ 2/83</td>
<td>≥ 4/76</td>
</tr>
<tr>
<td>Sulfamethoxazole</td>
<td>≤ 0.5/9.5</td>
<td>≤ 32</td>
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*Wild type* enrofloxacin
MIC = 0.005 µg/mL

Table 1. CLSI Interpretive criteria for selected drugs (*criteria established for animals for specific conditions*)
indicates “what is needed” in the patient to facilitate antimicrobial efficacy. Care must be taken with this simplistic approach: susceptibility data is generated from in vitro methodologies, yet it is applied to in vivo (and abnormal) conditions. Note that population susceptibility data can be helpful with empirical selection of antimicrobials (see below).

**Pharmacodynamic (Microbiological) data: what you need.** In contrast to disk diffusion, tube dilution methods involve inoculation of a series of test tubes with a standard number of organisms. The test tubes contain increasing concentrations of the drug of interest in two-fold dilutions whose range varies with the drug, reflecting concentrations achieved in the patient for that drug at the recommended dose. Following a standard time, the tubes are evaluated for detectable growth. The test tube that contains the lowest concentration of drug and no visible growth contains the minimum amount of drug necessary to inhibit (not kill) the growth of the organism cultured from the patient (the MIC). Ideally, this concentration must be achieved at the site of infection. Adaptation to computerized/automated systems allow much more accurate testing in short time periods. For either method of susceptibility testing, simplistically, the likelihood of a drug being effective in the patient is based on whether or not the recommended dose on the label is likely to generate plasma drug concentrations (PDC) that equal or surpass the MIC of the infecting organism. Diagnostic laboratories indicate the likelihood of susceptibility by the “SIR” letter It also must be handled properly: with a doubling time as short as 20 minutes, a small colony count indicative of no infection can rapidly become a high colony count (>10(5) CFU/ml) indicative of infection. Understanding the basis of that designation will facilitate antimicrobial selection. The SIR designation reflects whether or not the MIC of the infecting organism is less than (“S”), close or equal to (“I”) or greater than (“R”) the breakpoint MIC (MIC BP) of the drug. CLSI determines the breakpoint, based in part, on peak plasma drug concentrations (C max) of the drug (population data). Because dose and C max varies for each drug (eg, at 20 mg/kg, C max of enrofloxacin is 4 mcg/ml; at 22 mg/kg, C max of amikacin is 65 mcg/ml), the concentrations of drugs tested by the laboratory vary, and the breakpoint will also vary. Thus, one should not compare an MIC for enrofloxacin (eg, 0.25 mcg/ml) to an MIC for amikacin (eg, 4 mcg/ml) and assume the former is better. Rather, “how far” that MIC is from the Cmax determines how susceptible the isolate is to each drug. Note also that the range of each drug tested is very narrow, leading to “≤” on reports. For example, for the culture report below and amikacin, ≤ 4 means no growth occurred in the test tube containing 8 mcg/ml, which was the lowest concentration tested by the lab, so the MIC must be lower than
8 or ≤ 4 mcg/ml, (the next lowest concentration). The isolate is susceptible. An MIC of > X is accompanied by an “R” because the organism was not susceptible to the highest concentration tested. CLSI updates MIC_{BP} (generally yearly) particularly as new data is provided regarding organism susceptibility. Increasing resistance to organisms may lead to changes in the MIC_{BP} such as has recently occurred for amoxicillin and doxycycline. For older antibiotics approved decades ago, originally labeled doses may be inappropriate for all except very sensitive organisms. Again, a good laboratory will follow CLSI guidelines.

Not all “S’s” are alike. The selection of an antimicrobial should be based on the likelihood that therapeutic (effective) concentrations will be achieved at the tissue site. What is needed for therapeutic efficacy for infections is determined largely by the susceptibility (pharmacodynamic data) of the organism. If you have a C&S from your patient with MIC, the MIC for the drug of interest is how much you need. (For populations of microbes, the MIC_{90} provides an indication of what is needed.) Efficacy of an antibiotic is most likely to occur when the pharmacodynamic data is coupled with what is achieved in the patient. For the clinician seeking to improve antimicrobial efficacy, the further the MIC of the infecting organism is from the C_{max} (or MIC_{BP}) of the drug, the more likely effective concentrations will be reached at the site of infection. If a number of drugs are designated as “S”, the selection of which “S” is best might be narrowed by focusing on those drugs for which the MIC is furthest from the MIC_{BP} or C_{max}. The most susceptible, lowest tier drug should be selected.

Just because a drug is “S” does not mean that resistance is not developing. This is probably one of the most important points to understand. The “S” on a culture report indicates that CLSI anticipates that if the dose used for breakpoint determination is used in the patient, therapeutic success is more likely. As indicated above, the further the MIC is from the Cmax, the more likely this is to be true. However, an isolate may have developed some level of resistance. This is demonstrated in the culture report above: resistance to ticarcillin, an extended spectrum penicillin has emerged in this E. coli yet it remains

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susceptible to amoxicillin-clavulanic acid, indicating it is producing beta-lactamases. An isolate that has not resistance to enrofloxacin (“wild type”) would have an MIC approximating 0.004 mcg/ml; an isolate with an MIC of 0.25 to 0.5 mcg/ml has already undergone 1 to 2 steps of mutations (in topoisomerases) yet is still considered “Susceptible”. Unfortunately, most C&S methods do not test very low below the breakpoint (the limitation is in the number of wells that can be tested using commercial test kits) and as such, how low the MIC actually is often cannot be determined. **Bottom line: Selecting a drug: patient data:** Compare the MIC (what is needed) to the peak drug concentration achieved at the recommended (or modified) dose: the higher the Cmax is compared to the MIC, the greater the chance of therapeutic success and the less the chance of resistance. Once the “best” drugs are identified based on C&S, then the list can be narrowed down based on other factors. The same approach can be used for population data. Note that for the data above, when the MIC is compared to the Cmax for those “S” drugs, the following can be decided: the organism is most susceptible to the aminoglycosides and of them, gentamicin; followed by the fluorinated quinolones, with enro (at 20 mg/kg) being “more effective” compared to ciprofloxacin. Amoxi has a ratio of less than 1, even though it is “S”. **Why?** Because amoxicillin is only tested now for *E. coli* when infecting the urinary tract (effective concentrations to treat soft tissue infections can not be achieved, let alone maintained for amoxi with or without clavulanic acid, or ampicillin). This points out that for some drugs, CLSI does have different breakpoints for different tissues, particularly those that are effective only in urine (nitrofurantoin is another example).

**Population data:** Population data can be used for empirical antimicrobial selection. For example, using an antibiogram which indicates the proportion of isolates resistant vs susceptible to a drug. Similarly, theTarget® Antimicrobial Handbook indicates not only the most likely organisms cultured (but not necessarily pathogenic) from selected sites, but also provides a “scoring” system of susceptibility. For antibiograms (see Auburn University Canine Cumulative Antimicrobial Susceptibility Report) drugs to which >75% or more of isolates are susceptible might be wiser selections. A patient that has not been previously exposed to antimicrobials is more likely to be represented by the “susceptible” isolates whereas an “at risk” patient (eg, previously exposed to antimicrobials, immunosuppressed) may be better represented by the resistant proportion. Likewise, package inserts for newer antimicrobials include susceptibility data (MIC) and as such, can guide not only the selection of a drug, but the design of a dosing regimen. The MIC data represents isolates of the same organism collected from many different patients. A population distribution (which should be robust, ideally 100 or more isolates) found on a label may include: 1. the range of MIC for susceptible organisms; 2. the mode of MIC (the most frequently cited MIC); 3. or the MIC$_{50}$ and the MIC$_{90}$. The data are population statistics; the latter two reflect, respectively, the MIC below which 50% and 90% of the isolates (by genus and species) are inhibited (not killed). However, the MIC$_{50}$ and MIC$_{90}$ should be based on a large number of microorganisms to assure ac curate sample representation of the population (ideally >300). Organisms with MIC$_{90}$ that are low are more susceptible than organisms with higher MIC$_{90}$, organisms whose MIC$_{90}$ is approaching the C$_{max}$ of the drug (also on a package insert) prudently should not be treated with that drug. See also concentration and time dependency. An example of population MIC data is demonstrated from a fluorinated quinolone package insert. Those organisms most susceptible to the drug have the lowest MIC whereas organisms with higher MIC are less likely to respond.
Infection is (usually) in the tissue, not the blood. A reminder that C&S data is based on plasma not tissue concentrations. Further, other considerations must be made when basing drug selection and doses on the MIC. The MIC is the key, not the sole consideration. Design of dosing regimens are equally important to drug selection.

Cumulative Antimicrobial Susceptibility Report
Feline Isolates from January 2015 to December 2016

PERCENT SUSCEPTIBLE
(No. ISOLATES TESTED) *

| Organism                  | No. of isolates | Amikacin | Amoxicillin/Clavul | Ampicillin | Ceftazidime | Cefuroxime | Cefoxitin | Chloramphenicol | Erythromycin | Gentamicin | Linezolid | Metronidazole | Nitrofurantoin | Doxycycline | Pantocillin | Rifampin | Tobramycin/Tm-G + |
|---------------------------|-----------------|----------|--------------------|------------|-------------|------------|------------|----------------|--------------|------------|-----------|--------------|---------------|-------------|-------------|-----------|---------|------------------|
| Enterococcus faecalis     | 30              | 100 (8)  | 100 (17)           | 63         | 47          | 12 (17)    | 40         | 100 (12)       | 100 (17)     | 15 (13)    | 38 (29)   |              |               |             |             |           |         |                  |
| Enterococcus faecium      | 6               | 17       | 100                | 0          | 0           | 0 (4)      | 0          | 0 (1)          | 0 (3)        | 0 (4)      | 20 (6)    |              |               |             |             |           |         |                  |
| Escherichia coli          | 54              | 100 (52) | 44 (38)            | 77 (35)    | 77 (32)     | 75 (35)    | 75 (35)    | 70 (3)         | 100 (37)     | 72 (50)    | 82 (51)   |              |               |             |             |           |         |                  |
| Klebsiella pneumoniae     | 4               | 100      | 0                  | 0          | 0           | 100        | 0          | 0 (4)          | 0            | 75 (0)     | 0         |              |               |             |             |           |         |                  |
| Pseudomonas aeruginosa    | 14              | 71       |                    |            |             | 39         | 76         | 71 (5)         |              |           |           |              |               |             |             |           |         |                  |
| Staphylococcus aureus     | 8               | 100 (12) | 43 (17)            | 50 (10)    | 56 (12)     | 75 (18)    | 75 (18)    | 74 (9)         | 58 (5)       | 12 (12)    | 89 (17)   | 100        |               |             |             |           |         |                  |
| Staphylococcus intermedius| 7               | 100      | 71                 | 71         | 100        | 67         | 71         | 71 (12)        | 71 (12)      | 71 (12)    | 14 (14)   | 100        |               |             |             |           |         |                  |
| Group G Beta Streptococci | 6               | 100 (8)  | 100 (8)            | 68         | 70 (9)      | 75 (6)     | 56         | 81 (6)         | 100 (10)     | 100 (8)    |           |           |              |               |             |             |           |         |                  |

* Numbers in parentheses represent actual number tested different from total.
* Represents what is generally known as D. intermediate group and includes D. intermedius, D. pseudointermedius, and D. shahii.
MINIMIZING ANTIMICROBIAL RESISTANCE PART III: DESIGNING THE DOSING REGIMEN

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INTRODUCTION

Antimicrobial resistance is globally recognized. The Center for Disease Control indicates that antimicrobial use is the greatest risk factor for resistance and as such, even the most judicious antimicrobial use contributes to resistance. Designing the dosing regimen is the second “D” in the task of reducing antimicrobial resistance (De-escalate, De-sign and De-contaminate). The more “at risk” a patient is for therapeutic failure, the more critical the design of the dosing regimen. As such, once the decision to use an antimicrobial is made, the dosing regimen should be based on the best drug and the most appropriate dosing regimen. The best drug should be based, in part, on proximity of the MIC (or, using population data, the MIC 90) of the target organism to the drug concentration (Cmax) achieved in the patient at the dose to be used. Alternate sessions demonstrated that susceptibility data can be used to determine to which of the “S” drugs a target (or cultured) isolate is most susceptible. It is for this reason that the Minimum Inhibitory Concentration is the key and thus should be the basis for the design because of the following considerations.

Paramount to the use of population data in the guidance of drug selection is confidence in the isolate being treated. While population information regarding the most likely pathogens infecting body systems exist, historical data (eg such as that presented in the Target Antimicrobial Handbook®) has not (cannot?) discriminate between commensals and commensals that have become pathogenic (opportunists). This is likely to often lead to unnecessary or inappropriate antimicrobial use. Skin (S. pseudintermedius) and less commonly, urinary tract infections (E. coli) are probably the two infections for which infecting pathogens are most predictable. However, even for UTI, E. coli may represent only 50% or so of UTI pathogens (see Target Handbook); less, if the urine sample was not properly handled (ie, refrigerated) since the rapidity with which E. coli multiplies will rapidly overgrow other pathogens. Urine pH (particularly if coupled with gram stain) may provide some indication: if alkaline, a urease producer (eg, Proteus [gram negative rod], or Staphylococcus [gram positive cocci] may be present. Anaerobic infections can be predicted by odor and pus, although other facultative anaerobes may be present. Critical to judicious antimicrobial use is recognition that even if the pathogen. Failure to correctly identify an infecting pathogen becomes less important if the true pathogens is susceptible to the chosen drug. However, much of the data upon which literature is based is decades old, with organisms no longer as susceptible. The more complex the infection being treated, the less likely susceptibility patterns can be predicted because of the advent of antimicrobial resistance.

CONTEMPORARY PHARMACODYNAMIC DATA: CHOOSING A DRUG

Susceptibility testing: Population culture and susceptibility data can profoundly support empirical therapeutic decision making. MIC is an inhibitory and not a killing concentration. Whether or not the MIC determined renders a “Susceptible” vs “Resistant” designation depends on the breakpoint MIC that CLSI has determined: if the MIC of the isolate is at or lower than the susceptible breakpoint, the isolate is considered susceptible; if the MIC is at or higher than the resistant breakpoint, then CLSI
has determined it will not be possible to achieve effective concentrations at the recommended dose, and the isolate is given an R designation.

**Population data:** From either or both methods of susceptibility testing, two types of population data can be used to support empiric therapeutic decision making: proportion of susceptible versus resistant, and MIC population statistics. The advantage of the latter is that it also provides support for dosing regimens. The Target Antimicrobial Handbook® which, although limited to one diagnostic laboratory, is national in content. Although the dosing regimens are often inappropriate, it does offer guidance as to the organisms (not necessarily pathogens) most commonly isolated from various body tissues. Some points that can influence empirical decision making: Enterococcus and Pseudomonas are generally multidrug resistant. Enterococcus is resistant to all cephalosporins. However, the more common Enterococcus that we treat, *sp* *faecalis* remains very susceptible to penicillins, and as such amoxicillin is a good empiric choice if it is an assumed pathogen. Enterococci generally do not produce betalactamases, so clavulanic acid is not needed. For E coli, most first tier drugs are only fair in susceptibility. Note at our hospital, the addition of clavulanic acid to amoxicillin is prudent (although this may not be true nationally) suggesting beta lactamases are the most common mechanism of resistance. For staphylococci, note that the percent of isolates that are methicillin resistant is 43% (disconcertingly, this was only 22% in 2010). Note also that MRS is more prevalent in *S. pseudintermedius* compared to St. aureus (but also note the number of isolates tested). Although this data indicates that most staphylococci (and thus, most MRS) are susceptible to chloramphenicol, clinically, we do not see efficacy. Use of a static drug to treat an organism that has developed such resistance is not prudent.
The second type of population pharmacodynamic data that can be used is MIC data such as that on package inserts of animal antimicrobials approved in the United States after about 1990 or in current literature discussing antimicrobial use in the context of population MIC data (eg. Stegemann 2006 regarding data collected during approval of cefovecin). The latter is more likely to be current since package inserts cannot be easily changed once a drug is approved. Data supporting drug approval most commonly is collected from animals not previously exposed to antimicrobials. To show how population MIC data is collected and its application to a patient, assume that a dog has a skin infection caused by *Staphylococcus pseudintermedius*. Amoxicillin clavulanic acid might be a good choice, but patient MIC data is not available. How might population data be used? The MIC range includes the lowest (most susceptible isolate) and highest (most resistant isolate), respectively. The former, 0.03 mcg/ml, should not be used to represent that patient because it is lower than 99% of isolates infecting dogs and it is likely our patient’s isolate MIC is higher. The high end of the range, 8 mcg/ml, may be an outlier and thus may not effectively represent the true population or our patient. If the data is normally distributed, the most common MIC in the population – the mode - is also the median or the MIC50, or the 50th percentile. In this example, it is 0.5 mcg/ml. While this has a good chance of representing our patient, there is a 50% chance that the isolate MIC from our patient is higher. The MIC 90 or 90th percentile represents 90% of isolates in the population but avoids the outliers. If this target, 2 mcg/ml in this example is used, there is a 90% chance that the patient MIC is at or below this target. It can be compared to plasma drug concentrations achieved in the target population when given at the recommended dose (or the concentration can be “adjusted” proportionately if increased). In this instance, if we wanted to use amoxicillin clavulanic acid, we would want the dose to achieve 2 mcg/ml in the plasma.
The data can be applied to cefovecin, cephalaxin and amoxicillin-clavulanic acid based on the study by by Stegemann et al during the approval of cefovecin. Plasma drug concentrations are available on the package inserts (check Daily Med: http://dailymed.nlm.nih.gov/dailymed/) for cefovecin ($C_{\text{max}}$ of 3.5 mcg/ml); and cephalaxin (Rilexin®: $C_{\text{max}}$ of 22 mcg/ml at 22 mg/kg) but the package insert for amoxicillin-clavulanic is too old so we will have to rely on other literature (http://www.aavpt.org/general/custom.asp?page=48) which indicates a $C_{\text{max}}$ of 4 to 6 mcg/ml at a dose of 13 mg/kg. For MIC$_{90}$ data, for Staphylococcus pseudintermedius isolates (n=231) in the US: the MIC 90 for cefovecin, cephalaxin and amoxicillin-clavulanic acid are 0.25,2 and 0.25 (≤ indicates that the lowest MIC tested was 0.5 amoxicillin/0.25 clavulanic acid). Although the MIC cannot be directly ≤ 0.05/0.25 compared between the drugs, they can be compared to the plasma drug concentration for each drug and the recommended dose. Note that the $C_{\text{max}}$ of each drug far surpasses the MIC $_{90}$ for S. pseudintermedius, indicating each might be used (we have to apply time dependency next). The MIC$_{90}$ for S. aureus for cefovecin, cephalaxin and amoxiclav are: 2, 4 and ≤ 0.05/0.25 (n=89). S. aureus is not nearly as susceptible to any of the drugs although they apparently should still be effective. Now let’s do the same for E. coli. (n=223) The MIC$_{90}$ for cefovecin, cephalaxin and amoxiclav are 1, 16 and 8/4. Cefovecin might still be effective but cephalaxin probably should not be used (remember, this data is an average) and amoxic/Clav plasma drug concentrations are well below the MIC 90. Each of these beta-lactams is a time dependent drug, meaning drug concentrations should be above the MIC for all (amoxicillin, cephalaxin) to at least 50% of the dosing interval to minimize the advent of resistance. The drug with the shortest half-life is amoxicillin at 1 hr: 90% of any dose will be eliminated in plasma within 3 hrs. What are the implications for treatment of soft tissue infections? Not surprisingly, CLSI has recently determined that effective concentrations can not be achieved for E coli (and presumably many other coliforms) for treatment of non-urinary tract infections with amoxicillin clavulanic (but presumably gram positive such as Streptococcus and Enterococcus can still be targeted.

We can use package insert for marbofloxacin in dogs and cats to demonstrate how population MIC data can be used. The MIC data for each organisms includes the MIC range, the MIC$_{50}$ or median, and the MIC$_{90}$. The data for Staphylococcus pseudintermedius (135 isolates) is more robust than that of E. coli (61 isolates). The data for Staphylococcus aureus (with 12 isolates) should be applied to patients cautiously. Which organism is most susceptible to marbofloxacin? (Pasteurella because it has the lowest MIC$_{90}$). Least (Enterococcus). The Cmax of marbofloxacin at 2.5 mg/kg is 2.0 mcg/ml and at 5 mg/kg, 4.2 mcg/ml (in the dog). The MIC 90 for St. pseudintermedius and E coli are 0.25 and 0.06 mcg/ml, respectively. E coli is more susceptible. Which dose should be used? To answer that, remember that fluoroquinolones are concentration dependent drugs, and plasma drug concentrations (or concentrations at the site) should be 10X the infecting MIC. Now which dose should be sued for which organism? (High dose would be better for St. pseudintermedius).

**DESIGNING A DOSING REGIMEN**

Time has demonstrated that our approach to designing dosing regimens based on patient or population susceptibility data, while potentially useful for efficacy, has not necessarily been good for minimizing resistance. These may be for the following reasons:

1. **Bactericidal versus bacteriostatic drug**: Although reaching for a “cidal” drug is appropriate, the more important concern is to make sure that the dose must be designed to assure cidal concentrations are reached. This is much easier for a cidal drug. On the other hand, selected bacteriostatic drugs are capable of killing particularly if accumulated [eg, macrolides and lincosamides in phagocytes; urine concentration. Care must be taken to not apply this point to adamantly.
2. The mutant prevention concentration (MPC). An isolate upon which an MIC is based generally reflects 1 to 3 CFU isolated during the culture process. However, the infecting inoculum generally reflects greater than $10^5$ CFU. The larger the infecting population, the greater the challenge: the more drug molecules are needed to target the CFU, the greater the number of destructive enzymes, and the greater the risk of spontaneous mutation. The MPC helps exemplify this latter point. Because each isolate in an infecting inoculum has an MIC, the infecting population is actually characterized by an MIC population distribution for the drug of interest. The MIC yielded from C&S is likely to be the median (50th percentile), with those isolates at the low end most susceptible. Those at the high end represent mutant variants and are least susceptible to the drug. Indeed if the infecting inoculum reaches $10^6$ CFU, spontaneous mutations will allow at least one isolate develop resistance to any drug that might be used. The highest MIC of any of the infecting isolates in the inoculum is the MPC, and this is the concentration that must be reached in order to kill the mutants and avoid emergence of a resistant population. Should drug concentrations at the site of infection reach the mutant selection window (the concentration between the MIC from the susceptibility report and the MPC), because the most susceptible of the isolates are removed, a more resistant population will fill the resultant void. Once the new population reaches a sufficient size, infection may remerge. The new population will be characterized by a higher MIC compared to the original population. A normal, healthy patient may be able to overcome this infection, but a patient at risk may not be. The MPC cannot be predicted by the MIC but in general will be 10 to 100 or more fold higher than the MIC. Accordingly, dosing regimens should be designed to well exceed the cultured MIC (eg, target MIC90).

3. Postantibiotic Effect (PAE) and relationship between MIC and PDC: The PAE is the continued inhibition of microbial growth after a short exposure of the organisms to the drug. The impact of PAE on efficacy can be profound, particularly for concentration-dependent drugs. It is the PAE that allows some of these drugs to be administered at long intervals. The PAE may be absent for some organisms or some patients (e.g., some immunocompromised patients). In general, concentration-dependent drugs appear to exhibit longer PAE. The duration varies with the peak PDC (ie, higher = longer); the PAE is enhanced by combination antimicrobial therapy. 4. Concentration vs Time Dependency. The relationship between MIC and the magnitude and time course of PDC allows drugs to be fall into two categories. Efficacy of concentration dependent drugs, best represented by FQs and aminoglycosides, is best predicted by the ratio of peak plasma drug concentration (Cmax) compared MIC of the infecting organism (Cmax:MIC). For such drugs, the magnitude of the IQ generally should be 8-10 or higher for more difficult infections (eg, Pseudomonas aeruginos) or infections caused by multiple organisms. The duration that PDC is above the MIC is less important; in fact, efficacy may be enhanced with longer intervals. For such drugs, a low dose is is particularly detrimental. Thus, the highest dose should be used; this is particularly important for fluorinated quinolones (FQ), because resistance is always associated with multidrug resistance. For example, and MIC of 0.25 means 2.5 mcg/ml should be targeted at the site of infection. This necessitates a dose of 15 to 20 mg/kg. However, FQ efficacy is also related to total exposure; as such, twice daily administration of the same high dose might be indicated for organisms already characterized by low level resistance (see MPC below). For time-dependent drugs (eg, beta-lactams), efficacy is enhanced if PDC remain above the MIC [$T>\text{MIC}$] for the majority (60 to 70% or more) of the dosing interval. For such drugs, simply achieving the MIC is insufficient because PDC (and certainly tissue concentrations) fall below the MIC immediately. With time-dependent drugs, increasing the Cmax:MIC is likely to be beneficial, but choosing a drug with a longer half-life is important: since drug concentrations decrease by 50% every drug half-life, a Cmax:MIC ratio of two will result in PDC below the MIC in one half-life. The dosing interval can be two half-lives. To increase the dosing interval by two more half-lives, the dose would need to be doubled; to add another two half-lives, the dose would have to be quadrupled. For example, for a Staphylococcus pseudintermedius with an MIC for cephalxin of 2 mcg/ml (half-life approximates 3 hr), PDC achieve approximately 20 mcg/ml when given at a dose of 25 mg/kg. In one half-life (3 hr), PDC have dropped to 10; in 6 hr, to 5. Thus, a 12 hr dosing

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interval might be acceptable. However, this assumes all drug in plasma makes it to the tissue and the half-life is 3 hrs. In contrast, for amoxicillin, a dose of 13 mg/kg results in 4 mcg/ml in the plasma; with a half-life of about 1.5 hr, 90% of the drug is eliminated in 4.5 hours (a 9 hr dosing interval) and only isolates with very low MIC could be treated at this dose every 8 hrs. Indeed, CLSI (standard setting organization for C&S) has recently determined that amoxicillin with or without clavulanic acid should not be used to treat soft tissue gram negative infections; care should be taken even for gram positive infections in at risk patients. We recommend 25 mg/kg every 8 hours and only for very susceptible isolates. However, the shorter interval is more important than is the higher dose. Choosing time dependent drugs with long half-lives is prudent. Using another example, note that the MIC 90 of cefovecin for E. coli is 1 mcg/ml; the MIC 90 of Staphylococcus aureus is 2 mcg/ml. Based on the cefovecin package insert, while sufficient unbound drug will be achieved with an 8 mg/kg SC dose to treat “approved” organisms Streptococcus and Staphylococcus pseudintermedius, if treating the former organisms, the MIC 90 will be surpassed (below) in less than 1 day for Staphylococcus aureus and 2 to 3 days for E. coli. As such, a second dose may be necessary in some patients in 2 to 3 days.

4. Infections rarely are in plasma: Penetrating the site of infection. Water soluble drugs (beta lactams, aminoglycosides) may not reach concentrations in tissues that equal those in plasma. Even in non-sanctuary tissues, assume that only 30 to 50% water soluble drugs (beta-lactams, aminoglycosides) distribution into extracellular fluid. Thus, doses automatically should be doubled if based on MIC in plasma. Dose must be increased even higher for tissues characterized by non-fenestrated capillaries. For example, distribution of amoxicillin (but not imipenem) to bronchial secretions may be only 30%. For lipid soluble drugs, distribution into tissues tends to be excellent but for “static” drugs, killing concentrations may not be achieved. An exception may be drugs that are accumulated in WBC (such as macrolides and clindamycin as well as “cidal” fluorquinolones). In general, “I” drugs should be avoided because of this concern. Topical therapy will allow high concentrations to be reached with minimal impact host systemic toxicity or host microbiota. For UTI, infection is in the bladder wall covered by biofilm; additionally, not all patients concentration urine. Further, organisms in biofilm are frequently quiescent and thus not very susceptible to even bactericidal drugs. Most “bacteriostatic” drugs are eliminated in via the liver/bile and do not achieve high concentrations in urine. Thus assuming high concentrations in urine precludes the need for higher doses is not prudent if any complication exists.

A marked host inflammatory response mandates the need for cleansing the site; using drugs accumulated in white blood cells might be prudent. Most infections are associated with biofilm with is a formidable barrier to drug penetration. Foreign bodies should be removed whenever possible to help decrease the impact of biofilm.
DURATION OF THERAPY: A FOCUS ON URINARY TRACT INFECTIONS

The duration for successful treatment of uncomplicated lower UTIs might be as short as 3 to 5 days, such an approach is more likely to be successful if high doses and appropriate intervals are chosen. Treatment may need to be longer, however, if infection occurs anywhere other than the uroepithelium. In general, a 10- to 14-day therapeutic regimen has been recommended for the first episode of therapy. The "test for cure" can be based on a second culture 3 to 5 days into therapy, although this should be done only if clinical signs persist (otherwise, the temptation will be to continue to treat an asymptomatic bacteruria). Increasingly, evidence is emerging that in uncomplicated cases, 3 to 5 days of therapy may be most appropriate. Drugs that have been used successfully by humans for short-term dosing include trimethoprim/sulfonamide combinations, aminoglycosides, selected cephalosporins, and fluorinated quinolones. Limited information is available regarding duration of therapy in dogs. However, Westropp et al (JVIM 2012) have demonstrated that 3 days of dosing of enrofloxacin at 18-20 mg/kg/day for 3 days was not inferior to 13 to 25 mg/kg amoxicillin bid for 14 days in dogs with uncomplicated UTI. Factors that should preclude single-dose antimicrobial therapy for a lower UTI include recurrence, historical poor response to single-dose therapy, underlying predisposing factors to a UTI (including structural abnormalities of metabolic disorders such as diabetes mellitus, and hyperadrenocorticism), and either pyelonephritis or symptoms of a UTI that have occurred for more than 7 days.

For infections that reflect a relapse, the duration of therapy should be at least 2 weeks; however, for human patients suffering from a relapse, a higher cure rate occurred with a 6-week course of therapy. For animals, a duration of 4 to 6 weeks is recommended. Because relapse is likely to occur shortly after antimicrobial therapy is discontinued, cultures should be collected 7 to 10 days after cessation of therapy. The presence of relapse should lead to a longer course of therapy, perhaps at a higher dose. A new antibiotic should be selected if infection occurs more than 10 days after cessation of therapy; as more time elapses between cessation of therapy and the presence of bacteriuria, the more likely that reinfection is the cause of recurrence. In the event of relapse after 6 weeks of therapy, 6 months of therapy or more may be necessary. However, if the patient is asymptomatic, strong consideration should be given to no treatment unless mitigating circumstances indicate the need for therapy. Unless the underlying cause of infection can be removed, however, it is likely that resistance will emerge. An attempt should be made to assure that the infecting inoculum is killed with each therapeutic intervention such that the returning infection is characterized by lack of resistance. Greater care should be taken, however, in the selection of antibiotics for longer term therapy, with special consideration to toxicity. Drugs that are used for long-term therapy for human patients include amoxicillin, cephalixin, trimethoprim/sulfonamide combination, or a fluorinated quinolone. Cultures should be repeated monthly, and, as long as significant bacteria are not present, the drug need not be changed. Should relapse occur after a drug is discontinued, the same drug or a new drug should be administered for a longer course of therapy. Long-term therapy may be particularly important for animals in which renal parenchymal damage is a risk.
NON-OPIOID ANALGESIC ALTERNATIVES IN DOGS AND CATS
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INTRODUCTION

Drugs used to control pain are often chosen based on the type / phase of pain. This approach is largely rationale, based on the underlying cause of pain. Among the most effective and potent drugs used for controlling pain in animals, particularly acute pain, are the centrally and peripherally-acting opioid analgesics. These drugs are discussed in depth in an alternative manuscript in these proceedings. However, the past decade has been characterized by marked opioid use in humans, leading to an epidemic associated with massive economic and personal negative impacts. The Commission of the Food and Drug Administration has addressed all medical communities regarding appropriate opioid analgesic stewardship. This, and re-assignment of limited resources of opioid drugs has caused many practitioners to seek alternatives to opioid analgesic. Further, the limited role of opioids in controlling chronic pain has been generally accepted, but there is a need to understand analgesic alternatives for chronic pain in animals. Paramount to the use of opioid alternatives is the need to continue to provide effective analgesia in our veterinary patients, that is, patients should not suffer needlessly as the medical communities come to grips with opioid abuse. However, veterinarians can contribute to the solution by minimizing opioid sue when appropriate. The goal of this paper is to describe alternative analgesics for control of acute as well as chronic pain in dogs and cats. Pain management guidelines can also be found @https://www.aaha.org/public_documents/professional/guidelines/2015_aaha_aafp_pain_management_guidelines_for_dogs_and_cats.pdf), The Veterinary Anesthesia and Analgesia support group also provide guidance: http://www.vasg.org/ and it is this site that dose recommendations can be found. . Note that this manuscript focuses on drugs, but a host of non drug/non supplement therapies are also indicated for control of pain.

PATHOPHYSIOLOGY OF PAIN: A FOCUS ON TRANSMISSION

Using tissue injury (e.g., surgery) as a prototypical cause, the tree phases of pain are acute, subacute and chronic. Each is characterized by different tissue responses oriented toward healing. Acute pain reflects the inflammatory response (heat, edema, redness, loss of function, pain) that generally lasts about 72 hours. In regards to pain, the injured site is characterized by an exaggerated response to pain (primary hyperalgesia), originating from the injured tissue. However, sensitivity may extend into surrounding, non-injured tissues (allodynia also referred to as secondary hyperalgesia). During the subacute phase, the initial inflammatory response resolves as healing progresses. Lasting about 2 to 3 weeks, the area is characterized by epithelialization, angiogenesis, and fibrous and collagen deposition. During the final, chronic phase, remodeling (involving fibrous tissue and collagen) occurs, with the duration being several weeks up to months. The exact duration varies with each tissue. Mediators associated with each phase define to some degree the pain response, contributing to complexity. In addition to the mediators themselves, their source and location as well as receptors with which they interact define the pain response.

Pain signals during the acute phase are transmitted from nociceptors that transmit signals via unmyelinated (C type) and small myelinated (A type) nerves; kinases contribute to on going excitability and sensitivity. C type fibers transmitting to the dorsal horn contribute to the “wind-up" phenomena in 2nd order neurons. In the brain, excitatory mediators released by astrocytes descend into the dorsal horn neurons and through collateral projections into the secondary neurons, contribute to the emergence of secondary hyperpathia. Increased outflow to the brain leads to the behavioral aspects of pain. Persistent postsurgical pain (pain chronification) can occur if acute pain does not resolve. The transition to chronic pain probably reflects either persistent inflammation at the site of injury leading to hyperinnervation of that site, or injury to the nerve trunk themselves (e.g., neuropathic pain), with the pain referred to those sites. The latter pathophysiology probably reflects a neuroinflammatory reaction involving influx of macrophages at the dorsal root ganglia, as well as increased expression of pro-excitatory receptors or channels.
Among the receptors are N-methyl-D-aspartate (NMDA) receptors whose role in the transmission of pain (including neuropathic) involves glutamate and aspartate, major excitatory neurotransmitters in the CNS that bind to NMDA receptors. These receptors, located in the dorsal horn of the spinal cord, interact with opioid receptors and increasingly are recognized for their role in modulating response to pain. For example, activation of these receptors has been associated with the wind-up phenomena of hyperalgesia associated with chronic (and particularly neuralgic?) pain. Hyperalgesia associated with uncontrolled pain (and opioid use) may involve NMDA receptors. NMDA receptors also appear to be responsible for the development of tolerance to opioids. Induced COX-2 PGE also has been associated with hyperalgesia in either the spinal cord (primary hyperalgesia) or at nociceptors in peripheral tissues (secondary hyperalgesia), suggesting the importance of combination analgesic therapy.

The most effective means of controlling pain is pre-emptive. Nociception (perception of pain) is not limited simply to transmission of acute (nociceptive) pain, but also contributes to neuropathic pain. Both acute, and especially unrelied chronic pain, can shift from nociceptive to neuropathic pain. Neuropathic pain includes hyperalgesia (overreaction or increased sensitivity to painful stimuli), allodynia (reaction to an innocuous stimulation), or other neuropathies. Failure to control development of acute pain and hyperalgesia can lead to chronic pain; progressive and prolonged stimulation can lead to a “wind-up” phenomena. Reflecting an increased excitation of neurons in the dorsal horn, it is manifested as a pain response outside the site of injury, and can persist beyond resolution of the inciting cause (pathologic pain).

Should drugs be considered paramount to controlling pain in animals, several categories of analgesics are available, each varying in their mechanism of action and duration. Included are the opioid analgesics, sedatives / tranquilizers (generally altering response to pain, not providing analgesic effects), and nonsteroidal anti-inflammatory agents. Few of these drugs are approved for use in animals. In general, the use of all of these drugs should be considered part of a multimodal approach in that none may be effective as sole agent. In some respects, whether or not any of the following drugs is used to control acute versus chronic pain may largely depend on the availability of oral administration, although side effects associated with longer term use may also weigh in.

NON-OPIOID OTHER CENTRALLY-ACTING DRUGS

Several drug classes will not be discussed in this manuscript. The role of NSAIDs (including acetaminophen and galliprant) and cannabinoids in the management of pain is discussed more in depth in an alternative manuscript in these proceedings. Also not discussed in this manuscript are those products intended to support tissue health and thus decrease disease and pain. Examples include those drugs or supplements targeting polysulfated glycosaminoglycans (Adequan®, Cartrophen®, glucosamine/chondroitin sulfate products, omega fatty acids). Their role is variably established and their absence here should not be interpreted as discouragement of use of products demonstrated to be of high quality and safe. Biphosphonates which might be used to decrease pain associated with bone cancer are not discussed. Although glucocorticoids might be useful for control of pain associated with inflammation, their metabolic / endocrine / immunomodulatory side effects limits their analgesic use.

Tramadol (Ultram®; Schedule IV); also available combined with acetaminophen (do not use this preparation in cats) is a synthetic analogue of codeine currently marketed as a racemic (1:1) mixture of ± enantiomers. Tramadol appears to have multiple mechanisms of analgesia, with interaction among the pathways perhaps contributing to its efficacy. Opioid analgesia reflects agonistic interaction with mu receptors; as such, it is a Schedule IV substance and might not be considered an alternative to opioids by some. Although tramadol has only limited interaction with mu receptors, its primary metabolite, ODT (M1), has a 200 fold greater affinity for mu receptors and as such, is largely responsible for tramadol opioid effects. Tramadol is much less effective in humans that do not make M1. For a similar reason, the analgesic efficacy of tramadol in dogs (not cats) has been questioned: although dogs form the metabolite (at a 7 fold faster rate than humans; cats about 4 fold faster than dogs), at least based on greyhounds, they also appear to clear it more rapidly (half-life 7 hrs in humans
vs 1 to 2 hrs in dogs). Further, the oral bioavailability of the parent compound appears to be less in dogs. As such, area under the curve in dogs for ODT is much smaller compared to the parent compound in dogs versus humans. However, the presumed lack of efficacy of tramadol in dogs because of limited ODT formation should not be taken as evidence of lack of efficacy as an analgesic. In addition to its opioid-mediated analgesia, tramadol enhances spinal pain inhibitory pathways through inhibition of neuronal re-uptake of serotonin (5-HT) and noradrenaline (NA), and release of 5-HT; additionally, it is an antagonist of NMDA receptors. As such, tramadol might be indicated for a broad array of conditions associated with pain, including chronic pain: studies suggest that the analgesia associated with 5-HT1A receptor agonists increases with chronic or repeat administration. Several studies have demonstrated antinociceptive efficacy of tramadol in dogs, particularly when used in combination versus placebo with other analgesics. Its short half-life in dogs should lead to frequent dosing: Simulated oral dosing regimens based on kinetics determined in the dog indicate that 5 mg/kg every 6 h or 2.5 mg/kg every 4 h should yield tramadol and M1 plasma concentrations associated with analgesia in humans. Care must be taken when combining with other drugs that impair serotonin re-uptake, such as most behavior modifying drugs, and many dietary supplements, including sAMe and St. John's wort. Side effects of tramadol are unusual and generally reflect either overzealous use or overdosage. The risk of overdosage is increased in both renal and hepatic disease due to prolonged elimination of both parent and metabolite.

**Amantadine.** According to its package insert, amantadine is an antiviral drug with an unknown mechanism of anti-viral replication action. Approved for use to treatment influenza A virus, it also is useful for treatment of Parkinson’s disease in humans and drug-induced extrapyramidal effects. Its mechanism of action in Parkinson’s disease is not known, but proposed mechanisms included increased extracellular concentrations of dopamine (increased release or decreased uptake) at presynaptic neurons, direct simulation of dopamine receptors, or increased sensitivity of the receptors. Additionally, at concentrations considered to be in the low range, amantadine inhibits NMDA receptor-mediated stimulation of acetylcholine release (rat striatum; probably at the MK-801 site). Amantadine A dose of 31.5 mg/kg in dogs (equivalent to an approximate human dose of 15.8 mg/kg based on body surface area conversions) is not associated with anticholinergic actions, it nonetheless does cause anticholinergic-like side effects, including dry mouth, urinary retention, and constipation. In greyhounds, the half-life of amantadine approximates 4 to 6 hrs.

The role of **ketamine** (1-2 mg/kg [IV for burns], 0.5-1.0 mg/kg IM; CRI probably most appropriate) has been cited for its analgesic effects particularly when used in combination with other analgesics. In practice, use is most commonly associated with general anesthesia; analgesic effects occur at subanesthetic doses. It’s efficacy appears to prevent activation of NMDA (n-methyl-D-aspartate) receptors. In humans, some analgesia is provided when used in combination with other analgesics, particularly peripherally. However, side effects (CNS) limit its use as an analgesic. Ketamine is reasonably used in combination with other analgesics for control of pain. For example, in cats, it has been combined with domitor (0.025 to 06 mg/kg; IM in lumbar musculature), ketamine (5 mg/kg or 10 to 20 mls/hr of a CRI prepared with 60 mg ketamine/liter fluid) and butorphanol (0.2 mg/kg) as a preanesthetic followed by buprenorphine (0.03 mg/kg or 30 µg/kg) or oxymorphine (0.05 mg/kg IM) immediately post-operatively. Another example is control of cancer pain in dogs: ketamine has been used preoperatively (0.5 mg/kg IV), intraoperatively (10 µg/kg/min) and postoperatively (2 µg/kg/min) in conjunction with fentanyl.

**Dextromethorphan**, a “non-narcotic” opioid sold in over-the-counter cough preparations, also is an NMDA-receptor antagonist. It has increased the analgesic effects of opiates and nonsteroidal anti-inflammatory drugs (Moiniche 2002). The efficacy, however, is probably enhanced by an active metabolite, dextrorphan, which dogs do not seem to make in an unconjugated (active) form. The elimination half-life of dextromethorphan in dogs is approximately 2 hrs. It is not clear if a metabolite provides the major NMDA receptor antagonistic action, nor if the dog or cat produce sufficient metabolite for the drug to be effective in combination analgesic therapy.
**Tranquilizers and sedatives:** Tranquilizers do not provide analgesic effects but alter the animal's response to pain and they are most commonly used in combination with opioid analgesics. Some also may provide muscle relaxation. Those most commonly used are the phenothiazine derivatives (which may also provide antiemetic effects) such as chlorpromazine, promazine and acetylpromazine, and benzodiazepine derivatives such as diazepam and midazolam. Phenothiazines should be used cautiously in hypotensive patients or in patients with cardiovascular disease. The benzodiazepines are particularly useful in geriatric and debilitated animals. Agents from either group can be combined with opioid analgesics.

**Alpha2 agonists:** Alpha2 agonists warrant special consideration because they are potent analgesics at doses which do not cause sedation. Xylazine is an older drug whose duration of analgesia is short (0.5 hrs) and it has profound cardiovascular effects. However, its CNS depressant effects can be reversed with yohimbine or tolazoline. In addition, xylazine can be used in combination with opioid agonist-antagonists such as butorphanol and as an epidural, just prior to surgery or surgical recovery. Newer alpha2 agonists are safer and because of the proximity of alpha 2 receptors to opioid receptors, should have similar opioid sparing effects demonstrated with xylazine. These include medetomidine (0.75 mg/m2 IV or 1.0 mg/m2 IM; dogs) are associated with fewer cardiovascular effects and longer duration of activity compared to xylazine, but they are still evident. Medetomidine and its more potent dexametomidine (thus half the dose) provide both sedation and analgesia and like ketamine, they are indicated for control of pain in association with general anesthesia. They are labeled for use in dogs for clinical procedures that require short term (their half-life is short) chemical restraint and analgesia. The major attribute of these drugs is that their effects can be reversed with atipamezole, an alpha 2 antagonist. Like xylazine, dexametomidine can cause vomiting and cardiovascular suppression. An initial hypertensive response may preclude use of anticholinergics for the first 30 minutes or so (if indicated). Dexametomidine has proven equal or better as an analgesic compared to buprenorphine for control of pain in dogs. However, it should not be used at the full label dose for control of pain. Dexametomidine is approved as a transmucosally administered gel for the treatment of noise phobias; the role of this preparation for control of pain has not be examined. Domitor has been used in cats (see below) but caution is recommended in cats with respiratory disease.

**Anticonvulsants** such as carbamezapine, phenytoin, valproic acid and clonazepam have been used in humans to control selected neuralgias. However, the gabapentinoids (gabapentin [Neurontin®], pregabalin [Lyrica®]) are approved for use in humans to treat neuropathic pain, for example that associated with diabetes mellitus. Some of these drugs may be helpful by virtue of their antianxiety effects (e.g., midazolam, gabapentenoids).

**Gabapentin (Schedule V)** is an anticonvulsant originally approved in 1994 for treatment of partial seizures with or without generalization in humans with epilepsy, and subsequently approved for selected neuralgias. It has been used in dogs and cats for control of pain, as well as (particularly in cats) for its sedative and other effects for treatment of anxiety. Its mechanism is not clear but may involve muting calcium fluxes by targeting the alpha delta receptor. However, the drug may require close to 24 hrs at the receptor before analgesia occurs. Although gabapentin is absorbed well after oral administration, its absorption appears to be dose dependent, relying on a saturable transport process. This process has been cited as the reason that AED (and presumably analgesic) effects last longer than anticipated based on drug half-life, allowing twice daily administration. The drug is eliminated in people entirely by renal elimination; however, in dogs, 34% of the dose was metabolized to the N-methyl form, thus avoiding some of the risks of hepatotoxicity and drug interactions. The drug is sufficiently safe that TDM is not necessary; rather, treatment in humans involves an up titration approach, as is increased as needed to control seizures or pain. Although case reports indicate efficacy of gabapentin for acute or chronic pain in cats, efficacy could not be demonstrated using a thermal model of pain nor did gabapentin reduce isoflurane alveolar concentrations. In cats with OA, cats exhibited less activity but had higher owner scores. Gabapentin may have increased analgesia provided by analgesia in cats. Mild dizziness, nausea, and vomiting have occurred in a small percentage of human patients. Sedation in animals is common. Note that some gabapentin products contain xylitol and are not recommended in dogs or cats.
Oral administration of either an immediate or slow release product in beagles resulted in similar release kinetics. Gabapentin is among the drugs for which status epilepticus may occur during withdrawal; how this relates to sudden withdrawal when used to control pain is not clear.

Pregabalin (Schedule V) is the S enantiomer of an analog of GABA. It is also structurally related to the amino acid leucine and gabapentin. Pregabalin has been developed for the treatment of neuropathic pain and as adjunctive therapy in the treatment of partial seizures. However, according to manufacturer generate approval research, its mechanism of action is not clear. It does not appear to involve gabaminergic transmission, does not alter binding or responses at GABA-A or GABA-B receptors, and is not a substrate or blocker of GABA transporter GABA transaminase. It decreases central neuronal excitability by binding to an auxiliary subunit (alpha 2 –delta protein) of a voltage-gated neuronal calcium channel on neurons. It also reduces the release of multiple neurotransmitters (in vitro concentration of 1.6 ng/mL), glutamate, norepinephrine, substance P, and calcitonin gene-related peptide. It serves as a substrate of L-amino acid transporter in neuronal cell membranes, which facilitates pregabalin transport into the cell. As with gabapentin, there is a component of metabolism in dogs, with approximately 45% of the dose excreted as the N-methyl metabolite.

Tricyclic antidepressants have also been used in humans for the treatment of chronic pain. Amitriptyline and imipramine are considered first line drugs, particularly for pain which is continuous and aching. Selective serotonin reuptake inhibitors have less commonly been recommended; however, the newer serotonin/noradrenergic reuptake inhibitors (duloxetine; also see tramadol above) may be more useful. Sedation and anticholinergic side effects of these drugs may be undesirable. Not all tricyclic antidepressants—and particularly the newer products—may have analgesic properties through NMDA receptors or others. Trazodone has proven to be very effective for short term control of anxiety. Neuropathic, myofascial and arthritic pain appear to be most conducive to control. These drugs are contraindicated in patients suffering from urinary retention, heart block or narrow angle glaucoma.

Neurokinin receptor antagonists: Among the newer drugs for which visceral analgesic effects might be of benefit in both dogs and cats are the neurokinin receptor antagonists, such as maripitant. Although part of its efficacy may simply reflect control of post-operative vomiting, the role of NK-1 receptors in mediating pain has been well established. Preanesthetically, maropitant demonstrated lower (not significant) physiologic parameters compared to morphine in dogs undergoing OHE (Marquez 2015). (Note that injection itself may be painful; refrigeration may reduce this). One of the obvious advantages of this use is avoidance of vomiting commonly associated with opioids.

LOCAL ANESTHETICS (INCLUDING SYSTEMIC ADMINISTRATION)

Drugs approved with the intent of providing local analgesia are also given transdermally, epidurally and systemically, each with a correspondingly greater circumference of pain relief. For local effects, the potency, onset of action and duration of local anesthetic actions are dependent on lipid solubility, pKa and protein binding, respectively. Highly lipid soluble molecules easily penetrate cell membranes. Bupivacaine, which is more lipid soluble than lidocaine, is 10 times more potent than lidocaine. Likewise, tetracaine, which is more lipid soluble than procaine (an aromatic ring is added), is 40 times more potent than procaine. Drug pKa determines the amount of non ionized drug, which is able to move through cell membranes. The local anesthetics are weak bases with pKas of 7.7 to 9.0. In pharmaceutical preparations, the pH of the solution tends to be acidic, thus most of the drugs are present in ionized form. The higher the pka, the more drug is present in ionized form and the longer the onset of action. Local anesthetics which are more highly protein bound tend to be attracted to receptors and remain within sodium channels longer. Thus, bupivacaine which is highly protein bound has a longer duration of activity compared to procaine. Duration of activity is also impacted by effect of the drugs on local vasculature. All local anesthetics cause vasodilation which decreases the duration of action, as well as prolongs onset of action. Lidocaine with epinephrine is designed to prolong the effects of the anesthetic. Recently, the use of lidocaine administered as a constant rate of IV infusion has been advocated as an analgesic adjunct in human patients to centrally enhance perioperative analgesia.
Bupivicaine administered locally (around 5 intercostal nerves) was equal to epidural morphine for control of pain associated with lateral thoracotomy in dogs. When administered intrapleural (1.5 mg/kg), control of pain was superior to buprenorphine (0.01 mg/kg) in dogs undergoing thoracostomy. However, bupivicaine is more likely than lidocaine to cause adverse drug reactions, most notably cardiac depression and seizures. Doses should be less than 4 mg/kg. Currently, the systemic use of local anesthetics IV to control pain is being investigated in several species. The use is controversial and is not yet well documented. A “cocktail” of 7.5 ml lidocaine, 0.8 ml morphine (15 mg/ml) and 0.3 ml ketamine (10 mg/ml) mixed in 500 ml of fluids and administered to dogs intraoperatively at 10 ml/kg/hr (or delivering lidocaine at 50 μg/kg/min; morphine at 4 μg/kg/min and ketamine at 10 μg/kg/min) and postoperatively at 2-5 ml/kg/hr has been recommended. Topical administration of lidocaine gel (4%; over the counter: OTC) approved for use in humans is effective for controlling pain associated with minor surgical procedures. The gel is commonly used for minor procedures in dogs or cats, including catheter placement. A transdermal lidocaine patch system approved as an OTC in humans for control of local pain has been studied in both dogs (2-5% patches ?) and cats (1 patch; size?). Both species tolerated the drug well.

The efficacy of systemically administered sodium-channel local anesthetics (including mexiletine, a congner of lidocaine, and the anticonvulsant, carbamezepine) for treatment of pain, and particularly neuropathic pain, has been recognized for decades. What is missing are effective doses and clinical trials establishing efficacy.
INTRODUCTION

Nonsteroidal anti-inflammatory drugs have been used for centuries for the control of fever, pain and inflammation. Their action reflects variable inhibition of the metabolites of the cell membrane fatty acid, arachidonic acid. Non-selective NSAIDs target formation of prostaglandins (PGs) produced by both isoforms of cyclooxygenases (COX), 1 & 2; preferential drugs target COX 2 more than COX 1 ("preferential": carprofen, meloxicam, deracoxib); and selective drugs minimally target COX-1 ("selective": firocoxib, robenicoxib). Dual inhibitors (tepoxalin) target both COX 1 and 2 as well as the formation of leukotrienes mediated by lipoxygenases (LOX). For all, inhibition of COX is dose and drug-dependent. Target pharmacologic effects include, in order of dose, antipyresis, analgesia, and control of inflammation. However, unintentional effects reflecting inhibition of homeostatic PGs target all tissues in the body. Simplistically, PGs influence normal physiology and might best be predicted to be protective in nature. Constitutive PGs are mediated by COX-1 and are generally measured by inhibition of platelet activity (thromboxane); they also are responsible for homeostasis in most tissues. Inducible PGs mediated by COX-2 are responsible for homeostasis or a return to homeostasis on an "as needed" basis; activity is generally measured following endotoxin stimulation of mononuclear phagocytic cells. The ratio of COX 1 to COX 2 describes the amount of drug necessary to inhibit the respective isoform of the cyclooxygenase enzyme. A COX 1 : COX 2 ratio of greater than 1 indicates that the drug is more potent toward COX-2, and as such the drug might be expected to be safer and more effective compared to a ratio of < 1. However, species differences, test methodologies, and the variable effects of PGs in the body COX-2, suggest that this in vitro test might best be relegated to a screening tool. Manufacture bias should be avoided. Acetaminophen deserves special address: its mechanism of action is prior to that of traditional NSAIDs, targeting intermediary metabolites. As such, its anti-inflammatory effects are more limited compared to antipyresis and analgesic effects. Grapiprant also warrants special consideration since it blocks prostaglandin (specifically EP4) receptors, which presumably increases safety.

COX SELECTION: IMPACT ON PHYSIOLOGY

In general, inhibition of COX-2 is responsible for both efficacy as an anti-inflammatory and avoidance of COX-1 is responsible for safety. However, this simplistic approach may lead to therapeutic failure and increasing morbidity. Both COX-1 and COX-2 are constitutively expressed in many tissues and COX-1 does appear to have some role in inflammation. Inflammation: Prostaglandins (PGs), and primarily PGE2, induces vasodilation, capillary permeability, and chemotaxis and as such, cause each of the cardinal and clinical signs of inflammation, including pain and fever. PG E also modifies both T-cell and B-cell function, in part by inhibition of interleukin-2 (IL-2) secretion. Newly recognized inflammatory effects of PGE include its regulation of the production of IL-6, macrophage colony-stimulating factor, and vascular endothelial growth factor. In general, research consistently indicates that it is COX-2 that mediates the formation of PGE associated with inflammation, pain and fever. For example, whereas COX-1 is largely absent in normal synovial cells, COX 2 is induced in most types of arthritis, particularly in inflammatory arthritis in animals and rheumatoid arthritis in humans. In cartilage, COX-2 is associated with IL-1 degradation of proteoglycan and apoptosis of synovial cells (Hinz 2004).

Table 1. Cox1:Cox2 Ratios in Dogs by Differing Authors (Each study sponsored by the manufacturer of the drug that performs best)

<table>
<thead>
<tr>
<th>Drug/Ref</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
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<tbody>
<tr>
<td>Carprofen</td>
<td>129</td>
<td>6.5</td>
<td>1.75</td>
<td>6</td>
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<tr>
<td>Celcoxib</td>
<td>12</td>
<td>12.75</td>
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<td>Deracoxib</td>
<td>3.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Etofiban</td>
<td>74</td>
<td>69</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Firocoxib</td>
<td>29</td>
<td>10</td>
<td>12.3</td>
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<tr>
<td>Meloxicam</td>
<td>0.33</td>
<td>0.6</td>
<td>0.36</td>
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<td></td>
</tr>
<tr>
<td>Ketoprofen</td>
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<td>0.6</td>
<td>&lt;1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenylbutazone</td>
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<td>0.6</td>
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<td></td>
</tr>
</tbody>
</table>

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However, not all of the inflammatory actions of COX-2 should be considered undesirable; COX-2 induction in response to inflammation may play a role in tissue healing. Impaired COX-2 formation impairs healing in ligaments, bone, the GI tract and other tissues. The importance of COX-2 to dermal healing is not yet known. Interestingly, inhibition of COX-2 also has been associated with exacerbation of inflammation in some animal models. **Central Nervous System:** Both COX-1 and COX-2 are constitutively expressed in the brain spinal cord. Constitutive COX is highly regulated by factors such as ischemia, immunomodulation, cytokines, toxins, brain damage and maturation processes. However, COX-2 is the predominant COX. Although PGs of the CNS play a major role in pain, increasingly their contribution to other maladies is being elucidated. Among those aggressively being research is the role of COX in the pathogenesis of Alzheimer’s Disease (AZD). The extracellular deposition of fibrillar amyloid β, intracellular accumulation of abnormally phosphorylated tau protein and subsequent formation of Aβ plaques that mediate neurodegeneration and dementia in AZD is associated with inflammation (Hoozemans 2003). Mediators of inflammation are present throughout all stages of the disease whereas COX-2 is absent in normal astrocytes or microglial cells coupled. Further, COX-2 is upregulated in acute brain injury and in animal models of AZD. Finally, the connection between AZD and COX is supported by a lower incidence of AZD in patient’s with rheumatoid arthritis, presumably because of the use of NSAIDs for its treatment. The protective effects of NSAIDs in the AZD patient may reflect the antiplatelet properties of NSAIDs; aspirin in particular may decrease the risk of ischemic damage induced by blocked capillaries of the brain. Decreased formation of amyloid β protein also has been proposed (Warner 2004), suggesting a possible role in other diseases associated with amyloid β protein deposition. NSAIDs also may inhibit NMDA-receptor mediated neuronal cell death by preventing an increase in extracellular glutamate concentrations released in response to increased PGs. Finally, COX-2 appears to be involved in the loss of glutamate induced apoptotic cell death. Although companion animal disease comparable to AZD have not been identified, these benefits may ultimately prove useful in other CNS disorders that do afflict animals. **Pain:** PGs have been implicated in causing increased pain perception (allodonia) in damaged compared to normal tissues. Induced COX-2 PGE2 has been associated with hyperalgesia (exaggerated response to pain) in either the spinal cord (primary hyperalgesia) or at nociceptors in peripheral tissues (secondary hyperalgesia). Induction of spinal COX-2 in the dorsal horn also has been associated with central sensitization, manifested as a change in excitability threshold. The ability of PGs to contribute to hyperalgesia and central sensitization because of their effects on other mediators of pain and inflammation continues to be investigated. Potential influences include chemical mediators (eg, histamine, bradykinin, Substance P, nitric oxide), neurotransmitters (eg, glycine inhibition or glutamate stimulation) or through modulation of other receptors (eg, NMDA). **Gastrointestinal:** Both COX-1 and COX-2 are constitutively expressed in the GI tract. However, it is the constitutive expression of COX-1 that appears to play the predominant major role in the protection of the GI tract. These PGs decrease hydrochloric acid secretion, increase mucosal bicarbonate and mucus production, and increase epithelial cell proliferation and mucosal blood flow. Drugs which spare COX-1 while targeting COX-2 generally have been proven safer than those which target both COX isoforms. However, COX-1 is not the only isoform of importance to the GI tract. Indeed, induction of COX-2 appears to be important for healing of GI damage, appearing within an hour of GI damage. Interestingly, in the pancreas, constitutive COX-2 expression dominates, although the clinical relevance of this is not yet known. Indeed, induction of COX-2 may be important to healing, regardless of the tissue, including healing bones. The application of PGE-2 facilitates bone healing in experimental animals; 4 weeks of ibuprofen (16%) or rofecoxib (Vioxx®; 87%) compared to placebo (0%) led to malunion in rats with experimentally-induced fractures. **Cardiovascular:** The role of PGs in the cardiovascular system is largely beneficial and the relationship between COX-1 and COX-2 and their respective PG endproducts exemplifies the complex “ying-yang” balances which characterizes this family of chemicals. Platelets contain thromboxane synthetase, which catalyzes the formation of thromboxane from arachidonic acid. Thrombosis reflects platelet aggregation and vasoconstriction. The formation of a thrombus is kept “in check” by the presence of prostacyclin synthetase in vascular
endothelial cells. This enzyme catalyzes metabolism of arachidonic acid to prostacyclin (PGI2), a vasodilatory and platelet inhibiting prostaglandin endproduct. However, whereas TXA2 is associated with COX-2, prostacyclin synthetase colocalizes with COX-1. Thus, whereas drugs which target both COX isoforms will potentially allow the balance to be maintained, drugs which preferentially target only one isoform risk disruption of the balance. Such may be the case with COX-2 selective NSAIDs. Their preferential inhibition of COX-2 may allow thrombus formation to go unchecked, increasing the risk of thromboembolic disorders. **Kidney:** In the kidney; both COX-1 and 2 are constitutively expressed. Both are formed in the macula densa of humans and animals, but COX-2 may have a more important role than COX-1. In (nonhuman) animals, inhibition of COX-2 causes sodium and potassium retention in salt depleted, but not normal, animals. However, in humans, COX-2 appears to influence renal vasculature and podocytes. The role of COX in the kidney needs to be further elucidated before safety can be assumed for any NSAID; sparing COX-1 and targeting COX-2 can be expected to alter renal function. For example, kidneys do not develop in the embryos of COX-2 null knockout mice. The role of COX in the kidney differs among tissues and species. **Cancer:** In the 1990’s, a reduced risk of colon cancer was associated with consistent aspirin use. Subsequent studies demonstrated a marked increases in COX-2 in a variety of soft-tissue tumors in humans. A similar situation has been documented in transitional cell carcinoma in dogs (Knapp 2004). These studies suggest that benefits of NSAIDs in cancer may reflect inhibited COX-2 (Warner 2004). Mechanisms by which COX-2 may facilitate cancer growth or spread include impaired apoptosis, transactivation of epidermal growth factors or receptors (thus promoting colon cancer), and promotion of angiogenesis. Impact of COX-2 inhibitors has generally supported the role of COX-2 in cancer growth and spread. Depending on the model, inhibition reduces cell proliferation, increases apoptosis, and reduces metastasis. Inhibitors may also enhance anti-tumor effects of radiation, although toxicity is increased. However, at least for NSAIDs, GI toxicity is also increased when combined with antimetabolite anticancer drugs, presumably reflecting a combined toxic effect on the GI tract. COX-2 increasingly is identified with progression of cancer, the role of COX-1 continues to be scrutinized. For example, colon cancer was decreased in knock out mice void of COX-1, suggesting its role in the prevention of cancer should not be ignored. **Comparative Safety:** The differential effect of NSAIDs on the isoforms of cyclooxygenase offers some insight as to the differential pharmacologic and toxic effect of NSAIDs. As a class, NSAIDs appear to inhibit both COX 1 and COX 2. However, some the amount of drug necessary to inhibit each of the two isoforms provides a basis for assessing relative safety and efficacy of each drug. The ratio of COX 2 to COX 1 describes the amount of drug necessary to inhibit the respective isoform of the cyclooxygenase enzyme. A COX 1:COX 2 ratio of greater than 1 indicates that the drug is more potent toward Cox-2, that is, inhibition of COX 1 requires more drug than inhibition of COX-2, thus suggesting a safer drug compared to a drug characterized by a ratio less than one. The importance of species differences in interpreting these studies is exemplified by a study in humans that found the C1:C2 ratio for etodolac >>>> carprofen, but a similar study implemented by Ricketts with canine cells found just the opposite (Table to left). Various methods complicate interpretation of different reports. Further, the sponsor of each the cited studies can be identified by that drug which demonstrates the best ratio. Cox-1:Cox2 ratio should be used for screening only. Those drugs that have a more favorable COX1:COX2 ratio might be clinically safer in regards to minimizing the risk of NSAID-induced GI ulceration. **Safety:** Gastrointestinal damage is the most common and serious side effect of the NSAIDs among species. Cats are likely to be more sensitive to the GI side effects of NSAIDs compared to people. Because COX-2 is important to the healing ulcer, any NSAID should be discontinued at the first indication of GI upset. GI ulceration should be anticipated in any animal receiving an NSAID. Treatment for GI toxicity should protect the damaged mucosa, and if necessary, control gastric acid secretion. Because (intestinal) ulceration is difficult without acid, the single most important treatment.
may be antisecretory drugs. In the face of severe ulceration, a proton pump blocker such as omeprazole may be most effective. However, its inhibitory effects on drug metabolizing should be avoided in patients receiving NSAIDs. The antihistaminergic antisecretory drugs have been used for decades in humans. However, at least in dogs, famotidine consistently has been demonstrated to be either ineffective or less effective in maintaining a high (target) gastric pH, particularly compared to omeprazole (pump or tablet, although effects of pump decreased after 12 hrs). The effects of famotidine also seem to decrease with chronic (2 week) administration. Proton-pumps have a delay in onset in maximum efficacy. As such, starting both famotidine and omeprazole in patients with gastrointestinal mucosal damage might be considered, although proton pump inhibitors may be sufficiently effective on day 1. Esomeprazole, the S-enantiomer of omeprazole, has been demonstrated in dogs to be effective after (1 mg/kg) IV or oral (enteric coated granules) administration. The half-life is only 1 hr, but the irreversible nature of proton pump inhibitors should allow q 24 administration with multiple administration. Pantoprazole (1 mg/kg IV q 24 hr). In humans, a recent concern has been raised regarding the incidence of chronic kidney disease associated with long term use of proton pump inhibitors. This concern persists, and has transitioned from an increased risk in patients with acute kidney injury to an effect in all individuals regardless of kidney health status. The mechanism remains unclear; a direct cause and effect relationship needs further elucidation in humans and animals.

The benefits of sucralfate include binding to and thus protecting damaged mucosa, as well as increased PGs synthesis, angiogenesis, and sulfhydryl (oxygen radical scavengers) production. However, sucralfate must bind to damaged tissue to be effective and thus is helpful in treating, but not preventing, ulceration. **Hemostasis:** All traditional NSAIDs are able to impair platelet activity due to impaired prostaglandin (thromboxane) synthesis. At pharmacologic doses, aspirin selectively and irreversibly acetylates a serine residue of a platelet cyclooxygenase and, accordingly, will always have very low Cox 1: Cox2 ratios. However, newer NSAIDs inhibit prostacyclin, which acts to impair platelet aggregation, while minimally affecting thromboxane synthetase. As a result, thrombosis can occur relatively unchecked, predisposing patients to thrombosis (eg, Vioxx®). Unfortunately, it is unclear which of these drugs target predominantly Cox-2 in cats, decreasing confidence in use. **Renal:** The cat may be predisposed to nephrotoxicity (compared to the dog or human), perhaps in part because of differences in renal function. In the kidney, vasodilatory PGs are protective, assuring that medullary vasodilation and urinary output continue during states of renal arterial vasoconstriction. The loss of this protective effect becomes important in patients with compromised renal function. Newer NSAIDs do not appear any less likely to be associated with this effect than the traditional NSAIDs in humans. In cats, a review of adverse drug events at the FDA’s CVM website reveals that the proportion of adverse events of cats that reflect the kidneys (eg, 45% of meloxicam and 24% of carprofen) is greater than that in dogs (9%), and is greater for meloxicam compared to carprofen. Unfortunately, this is the only comparative data available for NSAIDs and it has been removed from the website.

### Table 2. Package insert target animal safety data

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose X Dose</th>
<th>Duration (weeks)</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carprofen</td>
<td>5.7-10</td>
<td>52-14</td>
<td>No change, Mild histologic</td>
</tr>
<tr>
<td>Etodolac</td>
<td>5.3-10</td>
<td>36</td>
<td>6/8 deaths (3 wks-months)</td>
</tr>
<tr>
<td><strong>Deracoxib</strong></td>
<td>3-10</td>
<td>6</td>
<td>NSF, Jejunal erosions*</td>
</tr>
<tr>
<td>Deracoxib</td>
<td>8-10</td>
<td>2-26</td>
<td>V/D, melena</td>
</tr>
<tr>
<td>?Firocoxib</td>
<td>5-10</td>
<td>26</td>
<td>V/D, ulcer 1/8, death</td>
</tr>
<tr>
<td>Meloxicam</td>
<td>5-10</td>
<td>6</td>
<td>microscopic</td>
</tr>
<tr>
<td>Tepoxalin</td>
<td>10-26</td>
<td>Test</td>
<td>Ulceration</td>
</tr>
</tbody>
</table>

* Time to wait before changing NSAIDs would be at least 3 X

### Table 3. Adverse events associated with meloxicam or carprofen in dogs or cats (based on 2004 FDA-CVM cumulative ADE data)

<table>
<thead>
<tr>
<th></th>
<th>Dog</th>
<th>Cat</th>
<th>Dog</th>
<th>Cat</th>
<th>Dog</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carprofen</td>
<td>14826</td>
<td>489</td>
<td>1124</td>
<td>497</td>
<td>3658</td>
</tr>
<tr>
<td>Meloxicam</td>
<td>3658</td>
<td></td>
<td>3658</td>
<td></td>
<td>3658</td>
</tr>
<tr>
<td>Vomiting (%)</td>
<td>29.6</td>
<td>47.2</td>
<td>25.7</td>
<td>33.4</td>
<td>34.0</td>
</tr>
<tr>
<td>Diarrhea (%)</td>
<td>9.9</td>
<td>5.7</td>
<td>11.5</td>
<td>0.0</td>
<td>13.7</td>
</tr>
<tr>
<td>Anorexia (%)</td>
<td>26.3</td>
<td>31.3</td>
<td>20.2</td>
<td>45.5</td>
<td>22.3</td>
</tr>
<tr>
<td>Bun (%)</td>
<td>9.6</td>
<td>24.1</td>
<td>9.3</td>
<td>45.1</td>
<td>17.1</td>
</tr>
<tr>
<td>Creatinine (%)</td>
<td>0.0</td>
<td>22.3</td>
<td>0.0</td>
<td>46.3</td>
<td>14.9</td>
</tr>
<tr>
<td>Kidney failure (%)</td>
<td>0.0</td>
<td>5.7</td>
<td>0.0</td>
<td>23.3</td>
<td>0.0</td>
</tr>
<tr>
<td>Azotemia</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>26.1</td>
<td>0.0</td>
</tr>
<tr>
<td>Death</td>
<td>11.4</td>
<td>13.7</td>
<td>8.0</td>
<td>13.5</td>
<td>9.5</td>
</tr>
</tbody>
</table>
Again, acetaminophen warrants special consideration: because it does not target end
prostaglandins, its safety concerns do not include those traditionally associated with NSAID. Indeed, it
can be used in combination with other NSAIDS. Its toxicity reflects relative overdose (which occurs
essentially with any dose in cats); the drug is generally removed by glucuronidation. If insufficient, the
drug is shunted back to phase I metabolism which results in the production of toxic metabolites
(oxygen radicals). If sufficient, glutathione can remove these radicals before they cause tissue damage.
Cathemoglobin, which contains more sulfhydryl groups than most species, is particularly susceptible to
oxidation. Treatment of acute toxicity or supplementation with n-acetylcysteine (or SAMe), a precursor
to glutathione, helps protect the liver and treat methemoglobinemia. Inhibition of cytochrome P450
metabolism helps prevent the formation of toxic metabolites. Package Insert Information: Limited post-
market clinical trials and are often sponsored by drug companies and potentially biased. Among the
most important sources of data is post market surveillance Table 3. The FDA web site for adverse
event reporting (www.fda.gov/cvm/) can be reviewed. Novartis reported (JAVMA 224; 2005:1112) that, of 29 dogs that developed perforated ulcers while receiving deracoxib, 90% received
either an overdose or another NSAID or glucocorticoid within the last 24 hr of presentation. GI damage
is the most common and serious side effect of the NSAIDs. Dogs are described as “exquisitely
sensitive” to nonsteroidal anti-inflammatory induced GI ulceration. All NSAIDs used in the dog have
been reported to cause GI ulceration. Further, because COX-2 is important to the healing ulcer, any
NSAID should be discontinued at the first indication of GI upset. GI ulceration should be anticipated in
dogs receiving these drugs and clients counseled regarding the side effects and potential treatments
for ulcerative injury. Unfortunately, there is no sensitive indicator of GI bleeding in dogs and damage
may be quite extensive before signs are evident. Treatment for GI toxicity should replace the missing
PGs (misoprostol), protect the damaged mucosa (sucralfate), and if necessary, control gastric acid
secretion. Because (intestinal) ulceration is difficult without acid, the single most important treatment
may be antisecretory drugs. In the face of severe ulceration, a proton pump blocker such as
omeprazole/ may be most effective; combination with an antihistaminergic drug such as famotidine
(bid dosing may be necessary) may be initially indicated. Patients which are predisposed to analgesic
nephropathy include geriatric patients, patients suffering from cardiac, renal or liver disease,
hypovolemic states including shock and dehydration, and patients receiving nephrotoxic (ie,
aminoglycosides, amphotericin B or other anti-prostaglandin drugs) or nephroactive (eg, diuretics)
drugs. Treatment or prevention includes administration of sodium containing fluids. Adverse event data
reveals that all NSAIDs will cause liver disease in dogs. Cats appear to a be predisposed to NSAID
induced renal disease (not just meloxicam). Table 3 delineates the percent of ADE that were kidney
associated in 2004. While the percent of renal ADE associated with meloxicam is higher in cats
(whether or not this is significant remains to be proven) compared to carprofen, the percent of renal
ADE in cats for either NSAID is greater than that in dogs. Caution is indicated in cats with high normal
creatinine; renal function should be intermittently monitored in cats. Because older animals may have
less protective ability against NSAIDS, hepatoprotectants such as SAMe, n-acetylcysteine (especially
for acute hepatopathy) or milk thistle should be considered. Glucosamine /chondroitin sulfate
combinations should be considered to help protect the GI tract, as well as help cartilage heal. Finally,
because NSAIDs are metabolized by the liver, other drugs which induce or inhibit drug metabolism
(imidazole antifungals, cimetidine, chloramphenicol) should be avoided. Client Information tear sheets
which accompany package in serts should be sent home with clients.
INTRODUCTION

The first portion of this discussion focused on the impact of NSAIDs on normal and abnormal physiology, including a focus on safety. Impacting safety are the pharmacokinetics of the drug and a special focus on the safety / use of NSAIDs in cats.

PHARMACOKINETICS.

Newer NSAIDs share a number of pharmacokinetic properties with older NSAIDs. The relevance of protein binding (which is > 90%) is questionable except in patients with altered hepatic clearance. Most, if not all, are metabolized by the liver, with marked differences among the species. Low bioavailability, which characterizes firocoxib (and possibly others) may contribute to variability in response. Note the half-life of the drugs is variable; the duration of effect may be longer than the half-life due to slow dissociation of the drug with receptors. As such, transitioning should take place after at least 3 to 5 half-lives or more. Saturation of drug metabolizing enzymes may be a problem for all with relative overdosing as has been shown for deracoxib. Many of the NSAIDs are present as racemic mixtures of the R and S isomer each of which is seen by the body as a different drug, with differing pharmacologic and pharmacodynamic effects as well as differences in clearance etc. Species differences are a given; although the cat is recognized for slower clearance of many NSAIDs, several drugs (those better tolerated) are cleared more rapidly by cats than dogs (see Table 1). Most NSAIDs are marketed as enantiomers; R and S stereoisomers are handled in the body as two different drugs, contributing to variability in species response. Age, gender and breed differences should be anticipated.

Poor metabolizers have been described for some drugs in humans (eg. Asian descent) and dogs. It is tempting to speculate that breed differences will also emerge in cats of Asian descent. Adding to the complex impact of disposition on NSAID safety is the impact of enantiomers. Enantiomers are molecules that are mirror images of one another (R- or S+). They occur for many drugs which contain a chiral carbon around which its bonds rotate. Although chemically similar, the body handles each enantiomer differently in regards to disposition (see carprofen in table). Further, the pharmacodynamic effects of enantiomers are likely to be different. In essence, the body treats each enantiomer as a different drug. Most NSAIDs contain a

<table>
<thead>
<tr>
<th>Drug</th>
<th>Species</th>
<th>Route</th>
<th>Dose (mg/kg)</th>
<th>Half-life (hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>FE</td>
<td>PO</td>
<td>2.5</td>
<td>27-45</td>
</tr>
<tr>
<td></td>
<td>CA</td>
<td>IV</td>
<td>44</td>
<td>8.6</td>
</tr>
<tr>
<td>Carprofen</td>
<td>CA</td>
<td>PO</td>
<td>2</td>
<td>4.95 + 1.32</td>
</tr>
<tr>
<td></td>
<td>SC</td>
<td></td>
<td>2</td>
<td>7.1 + 2.3</td>
</tr>
<tr>
<td></td>
<td>FE</td>
<td>IV</td>
<td>4</td>
<td>20.1 + 16.6</td>
</tr>
<tr>
<td></td>
<td>CA</td>
<td>IV</td>
<td>5</td>
<td>1.3 + 0.02</td>
</tr>
<tr>
<td></td>
<td>(Beagles)</td>
<td>IV</td>
<td>5</td>
<td>5.1 + 0.05</td>
</tr>
<tr>
<td>Deracoxib</td>
<td>CA</td>
<td>IV</td>
<td>2.35</td>
<td>3(5)</td>
</tr>
<tr>
<td></td>
<td>CA</td>
<td>IV</td>
<td>20</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>FE</td>
<td>PO</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Etodolac</td>
<td>CA</td>
<td></td>
<td>9.7 + 0.98</td>
<td></td>
</tr>
<tr>
<td>Firocoxib</td>
<td>CA</td>
<td>PO</td>
<td>5</td>
<td>7.8</td>
</tr>
<tr>
<td>Ketoprofen</td>
<td>CA</td>
<td>PO</td>
<td>5</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>FE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meloxicam</td>
<td>CA</td>
<td>PO</td>
<td>0.2</td>
<td>23.7 + 7.1</td>
</tr>
<tr>
<td></td>
<td>CA</td>
<td>SC</td>
<td>0.3</td>
<td>15.1 + 5</td>
</tr>
<tr>
<td>Piroxicam</td>
<td>CA</td>
<td>PO</td>
<td>40-50</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FE</td>
<td>PO</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FE (8)</td>
<td>PO</td>
<td>0.3</td>
<td>13</td>
</tr>
<tr>
<td>Robenicoxib</td>
<td>FE</td>
<td>SQ</td>
<td>2</td>
<td>1.9 (0.5-1)</td>
</tr>
<tr>
<td></td>
<td>CA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tepoxalin</td>
<td>CA</td>
<td>PO</td>
<td>20</td>
<td>2.0 + 1.2</td>
</tr>
<tr>
<td>metabolite</td>
<td>FE</td>
<td>PO</td>
<td>10</td>
<td>4.7 + 0.8</td>
</tr>
<tr>
<td>metabolite</td>
<td></td>
<td></td>
<td></td>
<td>3.5 + 0.4</td>
</tr>
</tbody>
</table>
chiral carbon, and are sold as racemic mixtures (50:50) of the R(-) or S(+) enantiomers. As such, extrapolation among species, genders, breeds, ages, etc becomes increasingly complex. Female cats already have been demonstrated to have a relative deficiency of CYP3A, the enzyme responsible for most drug metabolism.

**SPECIFIC DRUGS**

**Grapiprant:** The most recently approved drug that targets specific small molecules is the non-cox-inhibiting NSAID, grapiprant (Galliprant®). Indicated for treatment of osteoarthritis, this drug differs from nonsteroidal anti-inflammatories (which target cyclooxygenase, and newer ones, cyclooxygenase 2) by targeting (agonizing) the prostaglandin E-2 EP4 receptor. All prostaglandin receptors are G-protein coupled receptors which bind to one or more of 9 prostanoid receptors. PgE is the predominant PG interfaces with 4 receptors (hence, PgE receptors). The receptors is located in areas beyond the joint. In the gastrointestinal tract, they are highly expressed in small intestine and colon, providing an anti-inflammatory role (suggesting this drug might be avoided in patients with IBD). EP4 is important in stimulating bone growth, suggesting antagonisms might impair bone healing and it may be important in cancer growth, suggesting its antagonism might be helpful in cancer. EP4 may also protect the heart. Although this drug is categorized by an NSAID, its mechanism of action is clearly different than cyclooxygenase inhibitors because of its increased specificity. Based on assessment of the package insert, selective inhibition of prostaglandin receptor EP4 appears to be associated with better safety compared to co-inhibiting drugs. However, equally important, the EP4 receptor is located in those tissues in which PGE (constitutive or inducible) is located, indicating that the very physiologic responses discussed above are also influenced by this receptor. As such, its blockade must still be implemented with caution.

**Special Focus on Cats:** Drugs: **Robenacoxib:** Robenacoxib is approved for use in companion animals outside the United States and has been recently approved for use in cats and dogs in the US. In the author’s opinion, the major advantage of this drug compared to others is the short half-life, which (particularly in cats) decreases kidney NSAID exposure time. Isoform preference in studies supported by the manufacturer indicate a COX-1 to COX-2 ratio (95% inhibition) of 450 using whole blood assays in cats, indicated Cox-2 preference or selectivity. The disposition of robenacoxib in cats at 2 mg/kg intravenously has been reported, revealing an elimination half-life that is very short compared to meloxicam, also approved for use in the cat. Data from the ex vivo and pharmacokinetic studies were subsequently integrated with a model of inflammation in cats (n=10), resulting in a recommended dose 2 mg/kg dose every 12 hours. At 5 to 10 times the recommended dose (1-2 mg/kg), no significant changes occurred compared to placebo when dose for 28 days. When dose for 44 days, creatinine increased in all groups (including placebo), with the increase being significantly different from placebo only for the 2 mg/kg (but not 6 or 10 mg/kg) group (n=8/group). The authors concluded that robenacoxib was not associated with any biologically relevant toxicity even at 20 mg/kg for 42 days. In the FOI associated with Onsior® approval, cats (n=8; 8 mos old) receiving 4 or 10 X the recommended dose for 21 days or up to 5 X the dose for 6 months remained clinically normal, but had lower kidney weights compared to placebo. The apparent safety of robenacoxib may reflect not only is potency for Cox-2, but also is short plasma elimination half-life. Its efficacy appears to benefit from a longer presence at sites of inflammation, with control of pain (and inhibition of Cox-2 in inflammatory exudate) occurring for 24 hours. Robenacoxib has performed favorably (non inferior) to ketoprofen for treatment of musculoskeletal disorders in cats. The product has not been on the market sufficient long for post market surveillance to make an impact on safety assessment. **Meloxicam** Meloxicam, like piroxicam, is a member of the oxicam group of NSAIDs. The COX2:COX1 ratio for meloxicam, unlike that for piroxicam, favors selective COX2 inhibition in humans, suggesting that it has a wider margin of safety than most other. However, in cats, it is not clear if piroxicam is COX2 versus COX1-protective.
Both piroxicam and meloxicam are characterized by a shorter half-life in cats compared to dogs. Meloxicam is more potent (although not necessarily more efficacious) than aspirin, indomethacin, and piroxicam; hence, its dose is smaller. The disposition of meloxicam has been studied in cats. Meloxicam is among the NSAIDs characterized by a shorter half-life in cats compared to dogs, and is one of the few NSAIDS that appear to be well tolerated in cats. It’s safe use in Canada for several years predated its approval for use in cats in the US. Meloxicam is the only NSAID approved for use in cats in the United States. However, despite its apparent safety compared to other NSAIDs, the therapeutic margin of meloxicam is relatively narrow. Cats do not tolerate doses greater than or equal to 0.3 mg/kg gastric ulceration and death has occurred at 3X to 6X the normal for 10 days. Several studies support the efficacy of meloxicam in cats. In one study, the optimal dose of meloxicam to prevent endotoxin-induced fever in cats was 0.3 mg/kg, a dose also noted for its ability to cause toxicity. Meloxicam (0.2 mg/kg SC), carprofen (4 mg/kg SC), tolfenamic acid (4 mg/kg SC) and ketoprofen (2 mg/kg SC) did not differ in their ability to control post-operative pain following OHE in cats in one study. Meloxicam has been approved in Australia for chronic use at a dose of 0.05 mg/kg daily in cats. Genuw (J Fel Med Surg 2008) recently reported the safe used of meloxicam in adult (aged) cats using a case-control design (n=46; treated cats, age 12.9+4.2 yr) in Australia. Meloxicam was administered at 0.1 mg/cat (0.01-0.03 mg/kg (0.1 mg/cat) with food once daily following a 0.1 mg/kg once daily for 4 days oral loading dose. Mean duration of treatment was 5.8 months. However, there are some issues with the study. Since it was case controlled, no placebo of blinding took place and efficacy comparisons were based on comparison at baseline. Thus, it is difficult to assess efficacy. Illnesses in both the treatment and control group were diverse, with 25% of each group suffering from some disease. Ten pairs of cats were matched by disease. Cats with renal disease were included if disease was stable. One cat in each group died due to chronic renal insufficiency. Creatinine was measured only in the first 10 pairs of cats and only until 1 month of therapy. No statistical differences were found in creatinine between the treatment and control group (data not provided) at 1 month; however, the power of the study was not reported. One cat in each group died from chronic renal insufficiency; creatinine increased in cats with pre-existing disease treated with meloxicam numerically more than it did in control cats with the exception of 6 months (data not provided to evaluate variability). In addition, 4 cats in the meloxicam group and one in the control group developed vomiting. Caution is suggested when interpreting the results of this study as evidence of safety of meloxicam in regards to renal dysfunction associated with long term therapy. Caution is thus encouraged when using any NSAID, and perhaps meloxicam, in cats with pre-existing renal disease; this may include that population of cats whose serum creatinines are “high” normal.

Piroxicam is an oxicam NSAID approved for humans. It has received attention for its ability to reduce the size of tumors (transitional cell tumors and others) in dogs. Piroxicam may interact by an additive or synergistic action with anticancer drugs to cause tumor cell death. Piroxicam is a potent antiinflammatory in musculoskeletal conditions. The disposition of piroxicam has been studied in the cat. Notably, the half-life of the drug is much shorter in cats (12 hours) compared with dogs (40 to 50 hours). Little information ir available for this drug in cats. Tepoxalin [withdrawn in the US] is a potent antipyretic agent in cats at doses between 5 and 10 mg/kg and provides analgesia at least equivalent to butorphanol at 10 mg/kg for onychectomy (Personal Communication, Gerryll Hall, Technical Service Veterinarian, Schering Plough, April 2004). Tepoxalin is among the dual acting NSAIDs. It is a potent anti-inflammatory and analgesic pyrazole derivative that also inhibits production of IL-1, suppresses NFkB activation and dependent gene expression (Keier 2004). Tepoxalin also has been studied in cats. Whereas tepoxalin was well tolerated in cats when administered at 100 mg/kg once daily for 3 consecutive days, saturation kinetics occur when 60 mg/kg was administered as two doses four hours apart. Signs suggestive of CNS ADE occurred (drunken-like state), a response not recorded in any other species.) Carprofen is approved for use in cats in select countries outside the United States. The disposition of the drug has been studied in cats, including enantiomers at the dose associated with control of inflammation (4 mg/kg). A smaller clearance for carprofen in cats results in a elimination half-life that is at least twice as long in cats compared to dogs. The S isomer is cleared almost 3 times as rapidly as the R isomer resulting in
a shorter half-life for S compared to R in the cat. The relative COX2 selectivity of carprofen that occurs in dogs has not been well documented in cats, although Brideau’s data suggest relative selectivity similar to that in dogs. Clinically, however, this may not be true. The gastrointestinal effects of single dose carprofen (4 mg/kg IV) or aspirin (20 mg/kg IV) were studied in cats (n=5) using endoscopy and a randomized crossover design. Lesions in the stomach and duodenum 8 h postinjection were limited to minor pinpoint erosion in one cat. Clinical laboratory tests were not affected by either drug. However, duodenal perforation has been reported in cats using oral administration of carprofen (2.2 mg/kg twice daily for 7 days) following ovariohysterectomy. The ulceration was no doubt exacerbated by flunixin meglumine and dexamethasone treatment prior to referral. As a postoperative analgesic in cats, carprofen compares favorably with pethidine (meperidine), providing equal but longer analgesia (at least 24 hours) when administered at 4 mg/kg subcutaneously postoperatively (Balmer et al., 1998). In a clinical trial of cats undergoing OHE, no difference was found in the analgesic effects of carprofen, ketoprofen, meloxicam or tolfenamic acid. based on the visual analogue scale and a nociceptive threshold at the incision site. With 9/10 cats responding, all responses were described as good, although none prevented wound tenderness.) Ketoprofen. Although not firmly established, the efficacy of ketoprofen has also been attributed to its ability to inhibit some lipooxygenases and thus formation of leukotrienes. Ketoprofen is not approved for use in small animals in the United States but is approved for both dogs and cats in Europe. The half-life of the drug is short in cats (1.5 hrs) compared to dogs. Ketoprofen has been used as an analgesic in cats, particularly in Canada (1 mg/kg every 24 hours for 7 to 10 days). The antipyretic effect of ketoprofen (2 mg/kg subcutaneously followed by 1 mg/kg once daily orally) in febrile cats was rapid, being evident in 4 hours with temperatures normalized at that time. Temperatures did not change in the antibiotic-only treated cats. The use of ketoprofen as an analgesic is variable. In cats subjected to ovariohysterectomy, ketoprofen (2 mg/kg subcutaneously) compared favorably with buprenorphine (0.006 mg/kg or 6 mcg/kg intramuscularly) and meperidine as gas anesthesia was discontinued. Response better for both drugs compared with the control at both 4 and 8 hours but still present for buprenorphine only compared with control at 18 hours.
MEDICAL CANNABINOIDS: UNDERSTANDING THE TARGET?
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INTRODUCTION
The legalization of medical marijuana (Cannabis spp.) for treatment of human diseases has been accompanied by an increased use in animals. Much of this use is implemented without veterinary supervision by pet owners able to purchase animal dietary supplements derived from marijuana or its constituents. However, this use is largely accomplished by clients who access them through internet sites marketing products specifically for use in dogs and cats. Although such use is not currently evidence-based, support for medical use of marijuana-based products is increasing emerging in human medicine and most of the indications should extrapolate to animal diseases. The purpose of this manuscript is to describe the types of products being marketed, the regulations surrounding their use, and provide a scientific bases for their use based on presumed mechanisms of action. Finally, evidence supporting use for various indications will be summarized.

Marijuana refers to the dried leaves and tops of the hemp plant (Cannibis sativa) (Svienska 2008). It has been a part of recreational, religious and medical activities of a variety of cultures for over 5000 years (Krietzer 2009; Burns 2006) and was among the most commonly prescribed medications in the United States Pharmacopoeia until declared illegal in the 1930s. Cannibis sp is a pharmacologically (and toxicologically) diverse herb, containing at least 480 distinct compounds with their proportions varying between each subspecies, the part of the plant, and how that product is cured or prepared. Plant products include, in addition to marijuana, hashish and hashish oil, formed from the resin secreted by the plant. Hemp is commonly used to refer to the stem of the marijuana plant. However, marijuana is one of several varieties of hemp plants grown and harvested specifically for the stem which is used for a variety of products such as ropes, animal bedding. Unique to Cannibis are close to 70 different terpene phenolic compounds referred to as cannabinoids. Phytocannabinoids are unique to Cannibis. These lipophilic, low-molecular-weight compounds (300 Da) (Hosking 2008) are structurally similar to the eicosonoid arachidonic acid, the precursor of prostaglandins and leukotrienes. The most important of the phytocannabinoids are: Δ9–tetrahydrocannabinol (Δ9–THC), cannabidiol (CBD), cannabichromene (CBC), and cannabigerol (CBG) (Grotenhermen, 2003). In addition to phytocannabinoids, marijuana contains approximately 140 different terpenoids. These compounds are responsible for a variety of actions as well as its scent. The specific terpenoids yielded from a particular marijuana plant depend on the type of Cannibis (determining the fiber content), the part of the plant, its sex and age, whether or not it is cultivated in or outdoors, when it is harvested and the conditions at harvest, and how it is dried and stored. The serotoninergic effects of marijuana (5-HT1A and 2A) may reflect the impact of these essential oils, contributing to analgesia and mood modification. Other components in the plant include nitrogen containing compounds (n = 70: alkaloids, amines); carbohydrates, including common monosacharides (n=13: fructose, glucose, mannose), selected disaccharides (sucrose, maltose), and several polysaccharides (eg, cellulose, pectin) as well as several sugar alcohols (n = 12; mannitol, sorbitol, glycerol). A number of flavonoids also are present (n=23); among them, apigenin has a wide variety of effects, including interaction with benzodiazepine receptors, resulting in an anxiolytic effect. Other ingredients include fatty acids (n=33) and others.

PHARMACODYNAMICS: THE ENDOCANNABINOID SYSTEM

The Endocannabinoid System. Among the phytocannabinoids, Δ9–THC is the most understood as it is the main property of psychogenic producing behavior and pharmacological activity against pain (Grotenhermen, 2003, Di Marzo, 2007). Its discovery and elucidation of its role in the human body paralleled that of the discovery of the opioid receptors, leading to a description of the endocannabinoid system, including endogenous cannabinoid ligands (endocannabinoids) and their respective endocannabinoid (eCB) receptors (Hosking 2006; Di Marzo 2006; Di Marzo, 2007). The major cannabinoid receptors, CB1R and CB2R, are G-protein coupled receptors found within the cytoplasm of the cell. The CB1R receptor is ubiquitous and commonly found within the central and
peripheral nervous tissue as well as in peripheral tissues associated with the immune system (e.g., tonsils and spleen) (Burns 2006; Grotenhermen, 2003, Hosking 2008). In nervous tissue, the receptors predominate in the mitochondria of the neuron. These manufacturers of energy are negatively impacted by THC, but these effects of THC are largely blocked by CBD, demonstrating the yin/yang effects of these compounds. Similar to opioids, endogenous endocannabinoid ligands are capable of acting as agonists or antagonists on their corresponding receptors (Di Marzo 2006). CB2r are located principally on immune cells, but this includes microglia. The cannabinoid receptors are influenced by both endocannabinoids and phytocannabinoids. At least 5 endogenous cannabinoids have been described, with anandamide (CB1 and 2 agonist, but higher affinity for CB1) being the most thoroughly studied. It is synthesized by post-synaptic neurons, acting as a retrograde messenger to influence neurotransmitter, and particularly GABA, release. It is extremely unstable, being rapidly hydrolyzed to ethanolamine (an antimistamine) and arachidonic acid. Cannabinoids are able to disrupt short-term memory, impair cognition and time perception, alter mood while enhancing body awareness, discoordination, sleepiness, and reduce attention focus and the ability to “filter” irrelevant information. Although interaction with cannabinoid receptors is unique among plants to hemp, cannabinoids do not necessarily cause their effects by direct interaction with CBR. Other receptors are also targeted (e.g., benzodiazepines, serotonin, others). Cannabinoids can influence the release of other neurotransmitters.

Pharmacodynamic effects: The endocannabinoid system is a known contributor to physiology, but has been recognized for only about 25 years. In general, it contributes to homeostasis (Relax, Eat, Sleep, Forget and Protect; McParland 2014). Endocannabinoids appear to be important as neuroprotectants (e.g., antioxidants, inhibition of calcium influx and excessive glutamate production), for example, that associated with CNS ischemia or hypoxia, or the presence of neurotoxins. These effects appear to be mediated predominantly by CB1 (located particularly in the dorsal horn of the spinal cord) although CB2 also plays a role, depending on the tissue (Svizenska 2008). Cannabinoids also inhibit neuroinflammation (see therapeutic indications). Although not all effects of cannabinoids are mediated by CBR, their extensive distribution contributes to a variety of physiologic responses. The dopaminergic reward pathway is stimulated by CB1 receptors, motivating eating, smoking and substance abuse. A variety of clinical effects occur, including but not limited to inhibition of nociception (sensation and pain), decreased anxiety and emesis, manipulation of gastrointestinal and cardiovascular function, and stimulation of appetite (4). The CB2R receptors are principally found in cells and organs of the immune system, including leukocytes, monocytes, B and T cells, spleen, and tonsils. Activation of CB2R receptor receptors do not cause the effects on mentation that activation of CB1R produces, and has become a target for therapeutic use in human medicine by reducing inflammation, immune suppression, and as a chemotherapeutic (Burns 2006; Grotenhermen 2003; Hohmann 2006). CB2 receptors often modulate other signals. For example, they inhibit voltage-activated calcium channels, decreasing excitatory (acetylcholine) and increasing inhibitory (GABA) neurotransmitters. CB2 also is located on neurons where it may be associated with cell differentiation (Svizenska 2008). Untoward Pharmacologic Effects. As with many CNS active drugs, marijuana is associated with both tolerance (higher concentration needed to impart a similar pharmacologic effect) and withdrawal (a clinical syndrome of nervousness, tension, restlessness, sleep disturbance and anxiety). However, the long elimination half-life of the most active ingredient, THC (and others) appears to preclude a clear cut abstinence syndrome (Svizenska 2008). As with other addictive agents, laboratory rodents have been demonstrated to self medicate, suggesting an addictive component. Tolerance also should be expected: dogs exhibit a unique ataxic response to IV CBD. However, tolerance to this effect rapidly emerges within one week of repetitive treatment.

Specific cannabinoids: Cannabidiol (CBD) was the first isolated phytocannabinoid to be isolated from the Cannabis plant in 1930-1940s. In 1960s, CBD was employed as an anticonvulsant due to having similar pharmacologic effects as phenobarbital and diphenydantion (DPH) (Mechoulam 2002). CBD, has very low affinity for the cannabinoid receptors (often manifested as antagonistic), however, it serves as an antagonist for CB1R and CB2R agonists (Mechoulam 2007). The natural CBD (+) does not bind to CB1R, however, the synthetic (+) has been shown to bind to both CB1R and
CB2R (Mechoulam 2002). Besides anti-convulsant effects, CBD has been used for its anxiolytic, anti-psychotic, and anti-nausea effects (Mechoulam 2002). Interestingly, after oral administration of CBD in a murine model for rheumatoid arthritis, researchers saw a diminished interferon gamma (IFN-γ), decreased release of tumor necrosis factor alpha (TNF-α) and nitrous oxide (Mechoulam 2002; Malfait 2000). CBD also works as a potent anti-oxidative agent, showing greater protective nature against glutamate neurotoxicity than either ascorbate (Vitamin C) or α tocopherol (Vitamin E) (Mechoulam 2007). These anti-oxidative effects may have explained why CBD was successful in correcting hypermotility in mice, with no effects on the control population (Capasso 2008). On a smaller scale, CBD has been shown to stimulate mesenchymal stem cells responsible for bone formation and fracture healing, while also controlling bone resorption (Izzo 2009). Although CBD has very low toxicity on rhesus monkeys after IV administration, it has been reported to have very low oral bioavailability (9 h), which may be due to first pass metabolism (Mechoulam 2002). Cannabichromene, CBC, one of the non-psychotropic cannabinoids, has been shown to have strong anti-inflammatory properties through indirect activation of CB1R, through inhibition of the endocannabinoid inactivation (Shinjyo 2013). Most recently, CBC was determined to normalize the intestinal motility of an experimental model of intestinal inflammation in mice, but not alter the rate of transit in control animals (Izzo 2001; Izzo 2012). Cannabigerol, CBG, whose mechanism of action has not been completely elucidated, has a wide variety of therapeautic targets from antitumor activity as well as potent antibacterial effects towards selected microbes, including methicillin resistance staphylococci (MIC of 0.5 to 2 mcg/ml) (Appendino 2008; Rock 2011; Izzo 2009). Traditionally, cannibinol (CBN), the primary product of Δ9-THC breakdown, was been used to predict the age of the marijuana plant. CBN has recently been discovered to have an immunosuppressive effect by decreasing the production of interleukin -2 (IL-2) by decreasing T cell activation (Faubert 2000).

**Cannabinoids in Dogs or cats:** Cannabinoid receptors have been studied in a limited fashion in dogs. Initial studies focused on relevance to humans and provide evidence that dogs may react with unique behaviors. **Receptors:** In 1975, tritium-labeled Δ-9 THC (0.5 mg/kg IV) radioactivity was distributed throughout canine cerebellum and cerebral cortex, with increased concentrations in grey matter versus white matter noted; up to 50% of the signal reflected metabolites (28). Peripherally, radioactivity occurred in all organs save the vitreous humor. Peripheral tissues with the highest concentrations (relative) were bile (8), adrenal gland (3, 5), liver, auricle and ventricle of the heart, renal cortex, and pancreas (1), with the least concentration in the fat, trachea, and testis. The canine CB2R has been relatively cloned and characterized and shows 76% homology with other species (31). CB1R is located in the apical region of the striated cells of parotid and mandibular salivary glands (12). Both CB1R and CB2R were demonstrated in various cells of canine epidermis and dermis of dogs; both receptors increased in atopic dogs (8).

**Marijuana and pets** Legalization in states has yet to include veterinary medicine. Legalization of medical or recreation marijuana among the states is likely to be associated with an increased incidence of toxicity, with a 4 fold increase cited in one study although toxicity may reflect additional ingested foods (eg, chocolate) (Meola 2012). THC is among the compounds cited as a toxicologic hazard in detection (police) dogs (Llera 2008). It is the most common drug to which detection dogs are exposed. Both dogs and cats may become intoxicated with smoke inhalation as well as ingestion of food containing marijuana (or hashish). It is absorbed rapidly following either oral or inhalant administration with clinical signs evident within 30 to 60 minutes of ingestion, although one reference (Osweiler 2008) indicates onset as long as 12 hours after exposure. Cannabinoids of medical significance appear to undergo first pass metabolism and as such, the risk of toxicity with inhalant products is much greater compared to oral. The implication for medical use is that oral administration may not be cost effective. The drug is eliminated by hepatic metabolism and biliary excretion with elimination being complete in 5 days in dogs; duration of toxicity ranges from 30 minutes to 3 days, but 18-24 is the average. Enterohemepatic circulation may contribute to the prolonged half-life. The most common signs of toxicity following ingestion in dogs include tachycardia, hypotension, depression, ataxia, vomiting (inducing emesis is not recommended in clinically depressed dogs because of the risk of aspiration), altered behavior, bradycardia, hypersalivation, weakness, hypothermia and seizures.
Treatment is largely supportive, with sedation with benzodiazepines or phenothiazines as needed. Antiemetic therapy may be indicated. **Pharmacologic manipulation** The system can be manipulated by interfering with endogenous receptor ligands with cannabinoid or cannabinoid-like drugs, and enzymes responsible for endocannabinoid synthesis and degradation.

**REGULATORY CONSIDERATIONS**

Currently, at least 24 (PRO-CON) States have approved marijuana in some form. According to NORML (http://norml.org/states), a site dedicated to law reformation, 34 states have some type of conditional use, 15 states of decriminalized use, 14 states of medical marijuana laws. Several states have passed Industrial Hemp bills, with such plants being legal under some conditions as long as they contain less than 0.3% THC (DMW). However, cannabinoids themselves, included the oil CBD concentrate from these plants remains as a Class 1 Schedule substance, meaning it has a high risk of abuse potential and no recognized medical benefit. 10 different pharmaceutical cannabis products (including synthetic) have or are undergoing some level of approval (http://medicalmarijuana.procon.org/view.resource.php?resourceID=000883). This includes those predominantly cannabidiol (CBD) products derived from “industrial hemp”. Note that laws that legalize marijuana in selected states do not (and are likely not) to apply to veterinary medicine. Further, simply because marijuana has been legalized does not necessarily mean that they can be purchased in those states easily, nor that they can be transported across state lines. Until such laws are clear in what veterinarians can and cannot do, having clients purchase the products online is prudent.

In December, Congress passed the 2018 Farm Bill that specifically addressed industrial hemp. Some important points about the impact of this Bill on cannabinoid use in animals:

The Farm Bill of 2014 had already legalized industrial hemp (less than 0.3% THC DMW) for pilot programs; the 2018 bill removes the pilot distinction and now any one can grow it. But there are some important caveats: (LongLink @ www.brookings.edu...)

Regarding Industrial Hemp:

1. States must regulate the cultivation of IH. Each state's regulatory program must be approved by the Secretary of USDA. This includes verification that a plant being grown meets the definition of industrial hemp. This begins with the Secretary of USDA who will oversee states' regulatory plans for IH. These plans must be followed by the cultivators in the respective states. As such, although any person can grow IH, it will require a license and as such, not be as easy as growing tomatoes.

2. The protections for hemp (not necessarily CBD; see below [heavy sigh]) research established in 2014 have been extended.

Regarding CBD:

3. Hemp-derived products (I assume IH derived products) have been removed from Schedule 1 status, but CBD is NOT generally legalized. This is because the DEA and FDA have not dropped CBD from Schedule I status - yet. However, along with the DEA, some exceptions have been made: any cannabinoid (THC, CBD, etc) derived from hemp will be legal if produced in a manner consistent with the Farm Bill, that is, it is from IH that has been grown according to state and thus Federal regulations.

Some (Boothe's take): this suggests that any CBD product, regardless of CBD content, is legal as long as it is derived from appropriately regulated IH (meaning all the above restrictions are met). However,
4. the Farm Bill does not change the fact the Federal Government's still considers all state programs that have legalized cannabis products to be illegal. So while there is a potential for a broad expansion of commercially available CBD products because of the bill, CBD is not in general "legal". Each producer is going to have to demonstrate that the CBD used in their products is derived from approved IH. Those current commercial producers that obtain their product from crops meeting the 2014 Farm Bill (presumably?) may have a jump on the industry.

Hopefully, a movement toward change in this aspect (more general legalization of CBD) can occur when the democrats come to power in the House.

4. Unfortunately (and ironically), under currently law, any research involving CBD must be obtained from research grade cannabis which currently can be obtained only from University of Mississippi School of Pharmacy. Before (presumably) any more research goes forward, the dEA and FDA are going to have to provide guidance. (Heavy sigh).

5. Still to be worked out is the impact of pharmaceutical grade CBD such as that found in Epidiolex (which remains regulated by DEA as a Schedule V product) on CBD in IH. The approval of Epidiolex means that an active pharmaceutical ingredient that is in an approved drug is being sold commercially as supplements (which reflect either concentrated CBD oil or supplements which have been modified with the addition of CBD oil). Even if the CBD oil in these products has been derived from appropriately grown IH, the pharmaceutical manufacturers are not likely to take lightly the widespread availability of products containing an active pharmaceutical ingredient available in an approved drug. Expect some regulatory push back as the FDA tries to make Pharma and Dietary supplement manufacturers happy.

Summary:
The Farm Bill continues what the 2014 Bill began. The next step is putting into play Federal regulations for IH which will include approving the plants, and state regulatory programs. No CBD will be federally legal until the manufacturer can demonstrate that the product being sold is derived from CBD obtained from IH demonstrated to be as such. This will take some time (unless some of our current manufacturers, as I indicated above, already did that to be in compliance with the 2014 Bill).

Congress generally understands the safety of CBD; whether and when it can find a path to deregulate CBD without impacting THC is not clear. There is likely to be a lot of continued discussion as the implications of the Bill are discussed and the regulations surrounding the bill are promulgated and implemented.

MEDICAL USES

The proposed indications for medical marijuana have included, but are not limited to behavioral, sleep and gastrointestinal disorders, neuroprotection, antispasmodic but prokinetic, anorexia, nausea, glaucoma, diabetes, immunosuppression, malaria, anti-inflammatory and, of course, pain (Table 1, Izzo 2009). A proposed advantage of medical marijuana compared to a single drug (e.g., dronabinol, a synthetic THC [Marinol®]), is the multiple compounds contained in the plant. Two advantages are offered: 1. The compounds might act synergistically (a “synergistic” shotgun or entourage effect) to provide an enhanced desired pharmacologic effect while 2. at the same time, mitigating (one compound acting on another) undesirable effects. However, evidence for a synergistic benefit is lacking based on the lack of differences when THC is consumed as marijuana, versus Marinol® (humans). (Brenneisen 200X). Presumably, because marijuana contains so much THC, it may not be the most effective portion of the plant and it may contribute to more side effects. Cannabinoid deficiency has been linked as an etiology of a variety of illnesses: ("eCB deficiency syndrome" )as an
etiology in migraine, fibromyalgia, irritable bowel syndrome, psychological disorders, and others (McParland 2014). However, finding evidence to support either the negative or positive effects of cannabis can be difficult because such information is often tainted with emotionally-mediated opinion. PRO-CON (http://medicalmarijuana.procon.org/view.resource.php?resourceID=000881) is a useful site that provides links to evidence using a categorical approach, as well as information on approval status among the states.

**Pain Management:** Cannabinoid use has been best prescribed for its use in controlling neuropathic pain. Peripherally, CB2R receptors, and to a much lesser extent CB1R, have been effective in modulating the inflammatory response as well as tissue and nerve injury (19,33). CB2R, present on both mast cells and leukocytes, play multiple key roles in the modulation of the local inflammatory response including preventing mast cell degranulation, diminishing neutrophil migration, and decreasing the release of nitric oxide from macrophages (33). Cannabinoids have an analgesic effect on neuropathic pain in rodent models (thought to be mediated via the CB1 and CB2 receptors) in addition to other receptors, such as transient receptor potential vanilloid type 1 (TRPV1). Secondary to nerve injury, cannabinoid induced antinociception is more effective in alleviating pain than opioid drugs by suppressing wind up and noxious stimulus induced central sensitization (Hohmann 2006). Recently, studies of the interactions between the cannabinoid and the opioid systems indicates that co-administration of two agents may produce favorable synergistic effects, and may offer a new treatment strategy for multi-modal analgesia (MacPherson 2000; Ripamonti 2001). Recent findings have also suggested that NSAIDs may also owe some of their therapeutic success to their interaction with the endocannabinoid system either by inactivation of proteins or by encouraging biosynthesis. Rofenicoxib, a cyclooxygenase 2 (COX-2) selective nonsteroidal anti-inflammatory, synergizes with anandamide (the endogenous agonist of CB1R and CB2R), in a positive feedback loop to further elevate levels of anandamide as well as other analgesic fatty acid ethanolamide levels (Di Marzo 2007).

**Neurologic:** Suppression of convulsions/seizures: While the exact mechanisms resulting in suppression of epileptic seizures by cannabinoids are unknown, there are many receptors for cannabinoids (particularly CB1) in areas of the brain known to be sites where partial seizures originate. Experimentally, CBD attenuates experimentally-induced seizures in animals; this may reflect reduced calcium fluxes (Izzo 2009). THCV also has been associated with some anticonvulsant effects by virtue of its inhibitory effects on CB1. Anxiolytic: These effects have been demonstrated in healthy human volunteers (Izzo 2009). CBD exerts benzodiazepine independent effects, possibly by activating post synaptic 5-HT1A receptors. Neuroprotection: CBD is an antioxidant and as such has been proposed for treatment of Alzheimer’s disease, Parkinson’s disease and Huntington’s disease. Restoration of calcium homeostasis may prevent apoptosis (Izzo 2009). In rodents, CBD reverses brain damage associated with ischemia. Emesis and Appetite: Control of emesis and approved appetite are among the approved indications for FDA-approved cannabinoids. Emesis involves, among other signals, release of serotonin and subsequent stimulation of 5HT that activate neurons in the area postrema. CB1 receptors in the cerebrum, vestibular nuclei, and other brainstem nuclei involved in emesis suppress vestibular nuclei signals associated with nausea. Among the mechanisms of improved appetite is facilitated olfaction. Appetite is an approved indication for FDA-approved cannabinoid products.

**Other:** Cancer: In addition to control of adverse clinical signs associated with cancer and its treatment, a number of the cannabinoids have antiproliferative-anti apoptotic effects in a number of tumor cell lines. The National Cancer Institute has a link describing ongoing studies. http://www.cancer.gov/about-cancer/treatment/cam/patient/cannabis-pdq. Diabetes mellitus: CBD inhibits development of diabetes in non-obese diabetic mice, including ameliorating clinical signs of disease. This appears to reflect, in part, control of pancreatic inflammation, but also reduction of oxidative stress in target tissues (eg, retina). Bone formation: A number of cannabinoids (essentially all in Table 1) stimulate mesenchymal stem cells responsible for bone formation and fracture healing. CBD also controls bone resorption, reducing bone loss (Izzo, 2009). Antimicrobial: CBC and CBG have
demonstrated potent antibacterial effects towards selected microbes, including methicillin resistance staphylococci (MIC of 0.5 to 2 mcg/ml).

STUDIES IN ANIMALS:
One study has demonstrated efficacy of cannabidiol in animals: one for control of pain associated with chronic osteoarthritis (https://www.ncbi.nlm.nih.gov/pubmed/30083539) when used orally at 2 mg/kg q 12 hrs. Treatments for epilepsy are currently ongoing.
Medical Cannabis for Veterinarians
Robert J. Silver DVM, MS

Introduction
The use of cannabis for animal species is an area of growing interest, largely due to the therapeutic benefits being observed for humans and animals in the era of cannabis legalization. The close relationship humans have with their pets and other veterinary species has led to a renewed interest in the possibility and promise of cannabis to treat health issues similar to those already being treated in humans, in our animal community.

*Cannabis sativa* L., more popularly known as: Hemp, Marijuana, Mary Jane, Pot, Weed, Ganja, Bhang, Reefer, Dope, or Grass, has been a part of human history since before the written word. Archeological and anthropological evidence supports the fact that cannabis was cultivated by humans since before the beginnings of agriculture more than 10,000 years ago. During the Neolithic period ancient peoples used every part of the plant: The stems and stalks for fiber for cordage and cloth; the seeds which are high in protein and omega 3 fatty acids, for nourishment, and the roots, leaves and flowers for medicinal and ritual applications.

Veterinary medicine has not seen the same advances compared to human medicine for objective, non-biased scientific evidence for the use of medical cannabis in veterinary species. This is due, in part, to the fact that the legalization statutes, state by state, do not provide for similar legal privileges for veterinarians and their patients as physicians have for recommending cannabis for their human patients. However, with the passage of the Farm Bill of 2013, the cultivation and commercialization of hemp (legal low THC cannabis) on a state by state basis began, and with the passage of the Farm Bill of 2018 with the McConnell amendment, the Controlled Substance Act (CSA) Schedule One categorization of the resin derivatives of the hemp plant have been removed, thus opening the door to increased investigation with controlled studies of the benefits and potential risks of veterinary phytocannabinoid therapies.

With the removal of the CSA scheduling of hemp resins, veterinarians will be able to recommend, prescribe, or dispense legally grown low-THC cannabis, which by legal definition is called: “Hemp”. The use of high-THC cannabis, which is called “marihuana” or just “cannabis”, still remains a Schedule One substance, according to the Drug Enforcement agency (DEA) and is only permitted to be recommended by physicians who are certified as medical marihuana physicians. Two states, California and New York have had bills introduced in their state legislatures to give veterinarians the same privileges as human physicians to recommend medical marihuana to their patients. Hopefully this trend will extend to all of the states offering medical marihuana legislation.

Cannabis: The Plant and its Botany
The Latin binomial name for hemp, marihuana or just cannabis in general is *Cannabis sativa* L. It is in the Family Cannabaceae, and shares this Family with hops (*Humulus lupulus*) and common hackberry (*Celtis spp.*). Cannabis is dioecious in that plants can be either male or female, although rarely the plant will contain both male and female reproductive parts. These plants are called hermaphrodites. For resin and seed production, female plants are preferred. When fertilized by the male plant’s pollen, seeds will result, which in seed-oil cultivars is used as a source of omega 3 and 6 oils, and high quality protein. When the female plants are isolated from the male plant’s fertilization, it results in what has been called: “Sinsemilla” or “without seeds”. The unfertilized female plant directs its reproductive “energies” that would be used to make seeds in the fertilized female plant toward the production of increased amounts of the cannabinoid/terpene resins. For production of medical or recreational cannabis, the unfertilized female plant is preferred. (1)

Strains: In “Cannabis Culture” the use of the designation, “strains”, denotes both the psychotropic effects of cannabis on the human user and certain botanical characteristics of the plant, such as the shape of the leaves or how the plant grows. Since the “legalization” of medical cannabis in the US and the rise of the industry, breeding practices have interbred strains to create hybrids which blur the lines of distinction between these chemovars.

Strains are subsets of the *Cannabis sativa* L. genome, which contain different distributions of phytocannabinoids, terpenes and flavonoids. The number of possible combinations among these cannabis phytoconstituents is close to infinite. These strains are much like breeds of dogs. All are *Canis familiaris*, but there are definite differences between a Chihuahua and a Saint Bernard, in
spite of the similarity of 99% of their shared genome.

**Chemovars**: The two primary strains that are described for cannabis are “sativa” and “indica”. Each traditionally has had specific characteristics, both physical for the plant, and psychotropic impact for the user. A better term than strain would be “chemovar”, which describes the specific chemical makeup (fingerprint) of a given cultivar of the cannabis plant.

**Sativa** is known to be uplifting and cerebral, and in its negative manifestation, speedy and anxiety-causing, with tachycardia and emotional paranoia or nervousness.

**Indica** is known to be physical, a “body high”, but also can be calming, pain relieving and sedating and/or soporific depending on its profile of terpenes and phytocannabinoids.

**Terpenes** will be described in greater depth later in this paper and are responsible for the majority of these effects for each strain. To a lesser extent the THC:CBD ratio and presence of other phytocannabinoids will also play a role in the biological impact of cannabis.

**Cultivars**: This is a general term that is based more on the phenotype of the plant, but also can take into account the chemical constituents of the plant as is more appropriately defined with the term chemovar. Hemp and “marihuana” are considered cultivars of the cannabis plant, based on the legal definition of hemp which involves a reduced, non-psychotropic amount of THC in the plant (<0.3%). The actual limits of THC in hemp are determined legally by each sovereign nation.

There are three general categories of cultivars of cannabis:
- Plants that produce high amounts of seed oil and protein (food),
- Plants that produce fiber (building materials, biofuels, paper and cloth and cordage)
- Plants that produce large amounts medical resins such as THC and CBD, etc.

**Cannabis sativa** L: The Phytochemical constituents of a Complex Plant

There are several plant constituents in cannabis of medicinal value. Of most interest are the phytocannabinoids, which consist of more than 100 terpenophilic compounds, found mainly in cannabis, but recently have been described in several other plants in the family Linaceae (Flax), and Asteraceae (Echinacea (2) and Helichrysum(3)). Other phytocconstituents such as terpenes, terpenoids, and flavonoids also contribute to the medicinal profile of cannabis.

**Phytocannabinoids** exist in the plant as carboxylic acids and in the acidic form are non-psychotropic. The acidic form is converted to neutral molecular analogs by light, heat and combustion. (1) The phytocannabinoid that has gotten the most attention in this plant is Δ-9 Tetrahydrocannabinol (THC), which provides its psychotropic and some of its medicinal qualities.

THC has resulted in cannabis’ value, notoriety and illegality. The other phytocannabinoids, which are divided into multiple classes based on chemical structure, are not psychotropic, but contain the majority of the medicinal properties of this plant.

### TABLE SUMMARIZING BIO-ACTIVITY of the MAJOR and MINOR CANNABINOIDS (1)

<table>
<thead>
<tr>
<th>Compound</th>
<th>Bio-Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ-9-tetrahydrocannabinol (Δ-9 THC)</td>
<td>Analgesic (reduces pain), anti-inflammatory, antioxidant, bronchodilatory, improves symptoms of Alzheimer’s disease, benefit duodenal ulcers, muscle relaxant, anti-itch, cholestatic jaundice.</td>
</tr>
<tr>
<td>Δ-9 Tetrahydrocannabinolic acid (Δ-9 THCA)</td>
<td>THCA is the acidic or carboxylated form of THC. It is the predominant cannabinoid in psychoactive strains. It is non-psychotropic until activated or decarboxylated, smoked or cooked at temperatures greater than 245°F. Also has medicinal benefits, similar but also separate and different than THC.</td>
</tr>
<tr>
<td>Δ-9 Tetrahydrocannabivarin (Δ-9 THCV)</td>
<td>Anti-inflammatory, anti-convulsant, analgesic properties, anti-oxidant, neuroprotective in model of Parkinsons’s in one study, improved glucose tolerance and insulin sensitivity in vivo</td>
</tr>
<tr>
<td>Δ-8 Tetrahydrocannabinol (Δ-8 THC)</td>
<td>Stable in air, much less psychotropic than Δ-9 THC; At low doses, Δ-8 THC (0.001 mg/kg PO was found to induce appetite stimulation without psychotropic effects.</td>
</tr>
<tr>
<td>Δ-8 Tetrahydrocannabinolic acid (Δ-8 THCA)</td>
<td>The carboxylated (acidic) form of Δ-8 THC</td>
</tr>
</tbody>
</table>
Cannabidiol (CBD)  Anti-anxiety, anticonvulsant, Parkinson’s disease, Huntington’s disease, psychosis, MS, Alzheimers’s, cytotoxic for breast cancer, effective against MRSA, reduces oily skin, treatment of addiction

Cannabidiolic acid (CBDA)  Acidic form of CBD, carboxylated form of CBD. Has medicinal properties but not well studied at this point in time.

Cannabichromene (CBC)  Anti-inflammatory, analgesic, antifungal, anti-depressant, Anandamide reuptake inhibitor

Cannabigerol (CBG)  Anti-fungal, GABA uptake inhibitor (calming), antidepressant, analgesic, anti-inflammatory, reduces scales in psoriasis, effective against MRSA

Cannabidivarin (CBDV)  Anti-convulsant

Cannabinol (CBN)  Sedative, effective versus MERSA, helps with burns, reduces scales in psoriasis, helps with breast cancer. May be a degradation product of THC or CBD.

Terpenes/Terpenoids are equally important phytoconstituents of cannabis. These organic compounds are produced by a variety of plants. It is thought they serve a protective function for these plants. They are a significant component in plant essential oils. These molecules are responsible for the aroma of cannabis, and because they, like cannabinoids, are lipophilic, they also cross the blood-brain barrier and contribute to the medicinal benefits of cannabis. The US FDA considers terpenes and terpenoids to be Generally Recognized as Safe (GRAS), as they are flavor and fragrance components common to human and pet diets. Cannabinoids, terpenes and terpenoids are all produced in the same glandular structure on the cannabis plant, the trichome, from the same chemical precursor, geranyl pyrophosphate. Hops (Humulus lupus) is a member of the same Cannabaceae Family as cannabis, and they shares many of the same terpenes such as β-myrcene, β-pinene, humulone, and β-caryophyllene. Cannabinoids found in the plant are virtually odorless, emitting only a slight pitch-pine scent.

Flavonoids provide additional anti-inflammatory and anti-oxidant properties to cannabis. There are 21 flavonoids identified in cannabis that are in three different categories: 1) Flavones, such as vitexin, apigenin, isovitexin, luteolin and orientin; 2) Flavonols such as quercitin and kaempferol; and 3) Prenylated aglycone flavanones, which are unique to cannabis: Canniflavins A, B & C, which are similar to the prenylnaringenin from hops (Humulus lupulus). These are potent inhibitors of COX2 enzymes, affecting PGE2 production, reducing inflammation through that pathway. (4)

The Biological Impact of Cannabis Phyto-Constituents: The Entourage Effect

The biological effects of cannabis are due to interactions among the three main phytoconstituents: Phytocannabinoids, terpenes and flavonoids. This phytochemical interaction has been termed the “Entourage Effect”, and helps to explain the multiple biological activities of the cannabis plant, and the differences that are seen in bioactivity of the different strains of the cannabis plant. The Entourage Effect states that the potency of the whole plant extract is the sum total of the interaction of all of the plant constituents involved, and is different than the effect of any individual plant component alone. An additional aspect of the Entourage Effect is that there are multiple receptor mediated and non-receptor mediated pathways that these phytochemicals can influence the biomedical activity of the plant as a whole. (5)

The Endocannabinoid System: Ligands, Receptors and Enzymes

Following the determination of the structure of the first cannabinoid, Δ-9 THC in 1964 by Mechoulam, researchers started looking for the membrane receptors that could mediate the activity of the phytocannabinoids and the endogenous ligands to these membrane receptors. In 1988, the first cannabinoid receptor was discovered in the rat brain using a radioactive-labeled THC derivative. This receptor, termed Cannabinoid Receptor 1 (CB1), was determined to be a G-protein coupled receptor with the highest density in the rat cerebral cortex, hippocampus, hypothalamus, cerebellum, basal ganglia, brain stem, spinal cord and amygdala. This receptor is present in all vertebrate species and many invertebrates, indicating that that the endocannabinoid
system has been in existence for over 500 million years. The Endocannabinoid system consists of:

- The endocannabinoid ligand, which binds to the cannabinoid receptor
- The receptor itself
- The enzymes that synthesize and degrade the ligands.

The endocannabinoid system (ECS) has been identified in nearly all animals, from complex mammals like primates, to phylogenetically primitive animals such as members of the Phylum Cnidaria (which used to be called the coelenterates and includes jelly fish). The near universal presence and early emergence of the ECS, evolutionarily, is a strong indicator of its biological importance. Cannabinoid receptors are expressed in most animals, including: Vertebrates: mammals, birds, reptiles, and fish; Invertebrates: Sea urchins, leeches, mussels, nematodes and others. The most primitive animal an ECS has been observed in is the Hydra (*H. vulgaris*), a Cnidarian in the Class Hydrozoa, which is the first animal to develop a neural network. De Petrocellis et al. determined the major function of the ECS in the Hydra is to control the feeding response (6). Insects do not have an ECS, and it is thought that is due to the fact that the precursor for the synthesis of endocannabinoids is from arachidonic acid which is lacking in the insect. (7) It is evident from this information, that all veterinary species contain an ECS.

Therefore, an understanding of the ECS in these species is critical to the development of clinical applications for endocannabinoids and the phytocannabinoids derived primarily from *Cannabis sativa* L.

**LIGANDS**

**Endocannabinoids**

Mechoulam, who discovered THC, also discovered the first endocannabinoid, which he called “Anandamide” after the Sanskrit word for bliss. Anandamide binds to the CB1 receptor and creates similar effects as the phytocannabinoids naturally occurring in cannabis. A second endocannabinoid was subsequently discovered, 2-arachidonoyl glycerol (2-AG) which Mechoulam characterized from canine gut tissue. (8) There are several other compounds currently under investigation as additional endocannabinoids.

Endocannabinoids are long-chain polyunsaturated fatty acids that are derivatives of arachidonic acid, and have varying degrees of selectivity for either one or both of the cannabinoid receptors. Endocannabinoids are unlike other neurotransmitters in that they are lipophilic versus aqueous in nature. They also are not stored, but are manufactured *ad hoc* from precursors in the cellular membrane.

Endocannabinoids are released as calcium levels increase inside the neuron or when G-coupled protein receptors are activated. Endocannabinoids function as neuroprotectants by virtue of their antioxidant activity and by inhibiting calcium influx and excessive glutamate production. There are both cannabinoid receptor-dependent and cannabinoid receptor-independent actions of endocannabinoids.

Activities that are cannabinoid receptor-dependent include cognition, memory, appetite control, emesis, motor behavior, sensory, anxiety, and autonomic and neuroendocrine processes. Endocannabinoids can induce hypotension and bradycardia, inhibit cell growth, affect energy metabolism and modulate immune responses, as well as being involved in fat accumulation, glucose and lipid metabolism. Endocannabinoids can also exert pro-inflammatory actions such as enhancing the cellular migration of eosinophils, neutrophils and natural killer T cells (9) Endocannabinoids use a previously undiscovered form of neuronal communication: “Retrograde signaling”, which is the opposite to the normal direction of neurotransmitter release from presynaptic neuron to reception on the postsynaptic neuron. Endocannabinoids released from the postsynaptic neuron actually bind at CB1 receptors on the presynaptic GABA neurons to modulate neuronal activity. This novel discovery of retrograde signaling was termed: Depolarization-Induced Suppression of Inhibition or DSI.

DSI helps to explain a number of previously unexplained aspects of brain activity. When you temporarily dampen inhibition, a form of learning termed, “long-term potentiation” occurs, which is a process by which information is stored through the strengthening of synapses. It was also found that CB1 receptors can, in some cases, block presynaptic cells from releasing excitatory neurotransmitters. This is true in the cerebellum where endocannabinoids located on excitatory synapses help to regulate neurons involved with motor and proprioceptive control of movement. (10) This helps to explain, in part, the “static ataxia” uniquely observed in dogs only. The canine
species have the highest density of CB1 receptors in the cerebellum of any other species studied to date.

**Phyto-Cannabinoids**
Cannabinoids that are plant-based, known as phytocannabinoids, can bind or interact with the G-Protein Coupled receptors of the ECS. THC is the only phytocannabinoid that directly binds with the cannabinoid receptor as a partial agonist. (5)

**Terpenes**
Terpenes and terpenoids exert strong biological effects by themselves, and have been found to interact synergistically with phytocannabinoids in the treatment of pain, inflammation, depression, anxiety, addiction, epilepsy, cancer, fungal and bacterial infections (including MERSA)

**RECEPTORS**

**Cannabinoid receptors: CB1 & CB2**
The CB1 receptor is found in its highest concentrations on neurons that release gamma amino butyric acid (GABA), the main inhibitory neurotransmitter. It is located near the synapse. The discovery of this endocannabinoid receptor was a water-shed moment in neurophysiology in that it led to the discovery of the body’s own endogenous cannabinoid molecules (endocannabinoids). The endocannabinoid receptors evolved along with the endocannabinoids to constitute a naturally-occurring cellular communication system, which is the endocannabinoid system. It is sheer coincidence that the phytocannabinoids found in the cannabis plant resemble the endocannabinoids enough to activate the cannabinoid receptors or influence the ECS through non-receptor mediated activity.
The cannabinoid receptor CB1 is the most abundant G protein-coupled receptor expressed in the brain, with particularly dense expression in (rank order): the substantia nigra, globus pallidus, hippocampus, cerebral cortex, putamen, caudate, cerebellum and amygdala. This distribution has been determined for the human brain. Immunohistochemical studies in the dog have localized the CB receptors, especially in the skin. (11)
The cannabinoid receptor, CB2, is a second, G-protein coupled receptor for endocannabinoids. These receptors have been found to be strongly expressed in cells of the immune system, including the microglia, the peripheral nervous system and the organs. CB2 immunoreactivity was found in the B cell zones of lymphoid follicles in the dog, as well as in structures of the skin including mast cells, and hair follicles. (11) CB2 receptors are up-regulated during the early phases of inflammation in cells of the CNS and peripheral tissues, suggesting a role for cannabinoids in the management of inflammatory conditions of those tissues.
The endocannabinoid system’s major homeostatic functions were summarized by DiMarzo as: “Relax, Eat, Sleep, Forget and Protect.” (12)
The Endocannabinoid system has an effect on embryological development, neural plasticity, neuroprotection, immunity and inflammation, apoptosis and carcinogenesis, pain and emotional memory, hunger, feeding and metabolism. (13)

**Non-CB receptors**
In addition to the receptor-dependent mechanism of action of the cannabinoids, terpenes and terpenoids, their activity can also be mediated through non-receptor dependent interactions. The endocannabinoids exert multiple pharmacological effects through a number of different mechanisms not restricted to modulation of the endocannabinoid system through receptor-ligand binding. A partial list of these non-receptor dependent actions include: (14)
- Transient receptor potential (TRPV1) channel activation
  - Also activates: TRPV2, 3, 4, TRPA1, TRPM8
- Peroxisome proliferator-activated receptor λ (PPAR λ)
- GPR55, GPR18 = Atypical Cannabinoid receptors
- GPR3, GPR6, GPR12
- Serotonin Receptors:
  - 5-HT1A
  - 5-HT2A
  - 5-HT3A
- Glycine receptors
- GABA-A
- Opioid receptors
- Adenosine membrane transporter phospholipase A1
- Lipoxygenase (LOX) and cyclooxygenase-2 (COX-2) enzymes
- Calcium modulation
- Inhibition of anandamide inactivation by CBD, CBG and CBC

Terpenes and terpenoids exert strong biological effects by themselves, and have been found to interact synergistically with phytocannabinoids in the treatment of pain, inflammation, depression, anxiety, addiction, epilepsy, cancer, fungal and bacterial infections (including MERSA) (5)

**Enzymes to Recycle Endogenous Ligands**

Fatty acid amide hydrolase (FAAH) and monoacylglycerol lipase (MAGL) which are enzymes of the endocannabinoid system, deactivate the endocannabinoids AEA and 2AG and their congeners, respectively. Systemic endocannabinoid levels which are referred to as the “endocannabinoid tone”, are tissue dependent and are regulated by these two enzymes. It’s important to note here, that cannabidiol and phytocannabinoids other than THC inhibit these enzymes which prolongs systemic tissue exposure to the endocannabinoids.

**CANNABIS AND CANCER?**

First a little history:

A 2500 year old mummy was unearthed in Siberia in 1993. Known as the “Siberian Ice Maiden”, her burial chamber contained, among other things, a pouch of cannabis. MRI imaging revealed that the princess had a primary tumor in her right breast with enlarged lymph nodes and metastatic disease. It has been speculated that the cannabis was used to manage her pain and other symptoms, or even had been used as a treatment for her breast cancer. (15)

What if cannabis did cure cancer? Cannabis has had the popular “reputation” that it can cause cancer to go into long term remission. and that claim is repeated frequently on the internet. You have on the internet the phenomenon of “Rick Simpson Oil” (RSO), in which Mr. Simpson has cured his own cancer taking a concentrated oil extract he made himself from the cannabis plant. This recipe is available on the internet and there are claims from a number of people who had cancer who tried this approach and reportedly cured themselves.

Since the mid-1970’s, researchers have been studying the effects that both endogenous and exogenous cannabinoids have on cancer, in vitro and in vivo. With the on-going legalization of medical cannabis, state by state in over 50% of the US, this topic has now become open to increased investigation. The cannabis plant, the source of plant-based cannabinoids, has been illegal for 70+ years in the United States and most of the world. The two plant-based cannabinoids, cannabidiol (CBD) and Δ9-Tetrahydrocannabinol (THC) are the cannabis plant compounds at the center of these research efforts. Another factor that has been an impediment to veterinary research, specifically, is the lack of state medical cannabis legislation that would allow veterinarians to work with medical cannabis with their patients. This legislation would give veterinarians the same privileges that human physicians have with regard to recommending medical cannabis treatments to their patients.

**The Endocannabinoid System And Cancer**

The endocannabinoid system (ECS) is a recently discovered signaling system made up of: 1) Endocannabinoid receptors, which are G-protein coupled receptors distributed widely in the central nervous system and immune systems, as well as other bodily systems; 2) Intrinsic lipid ligands, the endocannabinoids AEA (N-arachidonoylethanolamide or “anandamide”) and 2-AG (2-arachidonoylglycerol); and 3) the transporters and biosynthetic and degradative enzymes. The endogenous ligands will also bind to other receptors such as the TPRV1 (vanilloid or capsaicin) receptor, the GPR55 “orphan” receptor, the peroxisome proliferator-activated receptor (PPAR), and the 5-HT1A receptor as well as others.

The endocannabinoid system can produce anti-neoplastic effects in a number of other types of cancers, such as cancer of the breast, prostate, bone, skin, brain (gliomas), and lung. (16) Studies have found that by removing the activity of the CB1 and CB2 receptors experimentally, a higher incidence of cancer will result. In mouse models of cancer, the genetic ablation of CB1 and CB2 receptors increases ultraviolet light-induced skin carcinogenesis. CB2 receptor (found mainly in the immune system) over-expression enhances predisposition to viral leukemia. CB2 receptors
can be found on tumor cells. The pharmacological activation of cannabinoid receptors can reduce tumor growth. Upregulated endocannabinoid-degrading enzymes have been observed in aggressive human tumors and cancer cell lines, indicating that the presence and signaling action of endocannabinoids, and perhaps phytocannabinoids, can have a tumor suppressive role. When CB1 receptors have been deleted in a genetic mouse model of colon cancer, it was found that tumor growth was increased. Precancerous lesions in the mouse colon, induced by the chemical azoxymethane, could be reduced with increases in endocannabinoid levels. With reduced expression of the endocannabinoid-degrading enzyme, monoacylglycerol (MAGL), prolonging elevated serum levels of endocannabinoids, tumor growth was inhibited in xenografted mice. The precise signaling mechanisms that regulates cannabinoid-induced cell death or cell proliferation continue to be under investigation. We are still discovering the many details that will help to clarify the role the endocannabinoid system plays in tumorigenesis and tumor suppression.

**Phytocannabinoids And Cancer**

The anti-proliferative properties of cannabis were first reported 40 years ago when it was shown that THC inhibited lung adenocarcinoma growth *in vitro* and *in vivo* in mice. No other research resulted from this study for well over 20 years, due to the prohibition around the cannabis plant. In the last 20 years, though, we have seen an emerging body of investigation, mainly using *in vitro* models of different cancers, to further elucidate the mechanisms whereby the ECS has an impact on cancer cell proliferation, angiogenesis and metastasis. Since the late 1990s, following the discovery of the ECS, these studies have shown that the cannabinoids have an anti-tumor effect in a wide variety of experimental models of cancer. These studies have found that the pharmacological stimulation of CB receptors is anti-tumorigenic. A number of cannabinoids (endo-, phyto- and synthetic) have been shown to have this activity, including: 1) THC; 2) CBD; 2) 2-AG and anandamide; 3) Synthetic cannabinoid receptor agonists with equal affinity for both CB1 and CB2, such as WIN 55, 212-2, and HU-210; 4) Synthetic cannabinoid receptor agonists with a higher affinity for CB1 such as methandamide; and, 5) Synthetic cannabinoid receptor agonists with a higher affinity for CB2, such as JWH-133. The anti-neoplastic mechanisms of action of cannabinoids have been established through examination of the pharmacological impact of cannabinoid receptor agonists on tumor growth.

**Induction of Cancer Cell Death and Anti-Proliferative Effects**

The cannabinoids have been found to induce apoptosis by means of CB1 and CB2 stimulation of the synthesis of the pro-apoptotic sphingolipid, ceramide. In studies performed in vitro with THC-resistant and THC-sensitive glioma cells, it was found to up-regulate the expression of the stress-regulated protein P8 (AKA: NUPR1). This protein is a transcription regulator and involved in the control of tumorigenesis and tumor progression. In these studies THC also impacted the endoplasmic reticulum (ER) stress-related transcription factors ATF4, CHOP (AKA: DDIT3) and TRIB3. ER stress response is an attempt by the endoplasmic reticulum to re-establish homeostasis. The stress response becomes activated in response to Ca\(^{2+}\) depletion, oxidative injury, a high fat diet, hypoglycemia, viral infections and exposure to certain anti-cancer agents. ER stress reduces the protein load on the endoplasmic reticulum by shutting down protein translation and gene transcription with the goal of increasing the ER protein-folding capability. When this stress response fails to restore homeostasis, cell death can ensue, usually through intrinsic apoptosis, but through a different pathway can result in autophagy, which is another cause for cancer cell death. Autophagy is an attempt of the cell to correct its imbalance, and if successful, the cell will continue to live. So autophagy is not always an effective path to cancer cell death. Interestingly, it has been found that autophagy is "upstream" to apoptosis in the mechanism of cannabinoid-induced cancer cell death. Blocking autophagy prevents cannabinoid-stimulated apoptosis, but
apoptotic blockade prevents apoptotic cell death but not autophagy. In addition to inducing cancer cell death through autophagy or apoptosis, cannabinoids also have been found to have an anti-proliferative effect by inducing cell cycle arrest. The effect of cannabinoids on hormone-dependent tumors may be due to their interference with activation of growth factor receptors. Cannabinoids can also down-regulate other cancer cell growth factors such as: PIGF, BFGF, SDF-1, Ang-2, leptin, interferon-γ, and thrombopoietin. Cannabidiol, or CBD, is a cannabinoid that does not bind to CB1 or CB2 receptors directly, yet uses many alternate pathways to influence the endocannabinoid system.

CBD has been observed to promote the apoptotic death of cancer cells, independent of CB1 and CB2 receptors. Its mechanism of action, which has not been completely worked out, promotes the production of reactive oxidative species in cancer cells as well as an increase in the other endocannabinoids through inhibition of FAAH and MAGL.

Inhibition of Angiogenesis, Tissue Invasion and Metastasis (17)
The activation of the vascular endothelial growth factor (VEGF) pathway in cancer cells, is known to induce angiogenesis. It has been found that cannabinoids down-regulate the two main VEGF receptor (VEGFR1 & VEGFR2) pathways through reduced production of VEGF. VEGFR activation is decreased as a result of the reduced amount of its ligand, VEGF. Activation of the CB receptors in vascular endothelial tissue inhibits proliferation and migration, and induces apoptosis in addition to activating endothelial cells. Thus cannabinoid activity results in a more normalized tumor vasculature with smaller and/or fewer vessels that are less "leaky", therefore less likely to result in metastasis.

Phytocannabinoids have been found to reduce metastasis in animal models for glioma, breast, lung and cervical cancers grown in tissue culture. Tissue invasion, which is required for metastasis, is regulated by the extracellular proteases (MMP2) and their inhibitors (TIMP1), which are modulated by cannabinoids.

Clinical Use Of Phytocannabinoids In Cancer Patients
Although historical and anecdotal evidence suggests that cannabis can be used to treat clinical neoplastic disease, carefully-controlled clinical trials of cannabis as a cancer treatment are rare to non-existent. Currently in the United States, THC and CBD are both considered to be Schedule One controlled substances. Researchers need a special Schedule One registration with the Drug Enforcement Agency of the United States to conduct research using these cannabis resins, and the only legal source of cannabis for clinical trials is the National Institute for Drug Abuse (NIDA), which makes acquisition of these plant extracts difficult to obtain. These factors all have made it difficult to conduct clinical studies into the effectiveness of cannabis for treating cancer.

Ladin (22) in his review article describes a number of in vitro and in vivo studies using cannabinoids in human patients with glioblastoma. Here they are summarized:

- Phase 1 clinical trial of THC in GBM patients indicated a good safety profile
- Intra-tumor injection of THC in nine patients with actively growing recurrent GBM decreased tumor cell proliferation and induced apoptosis
- Cannabinoids promoted the survival of healthy oligodendrocytes, astrocytes and neurons
- The anti-neoplastic effect of THC was enhanced when combined with CBD
- THC:CBD in combination with standard GBM alkylating chemotherapy drug Temozolomide (TMZ) was more effective in reducing tumor size than any one of these agents alone.
- THC:CBD treatment of GBM tumors in mice enhanced the cytotoxicity of ionizing radiation

GW Pharmaceuticals’ sublingual drug Sativex contains a 1:1 ratio of CBD:THC. In a study including 21 adult patients with histopathologically-confirmed GBM, subjects were receiving TMX chemotherapy and also received 27 mg THC and 25 mg CBD daily. The control group only received the TMZ. They had a 44% 1 year survival rate. The cohort of subjects receiving the TMZ and Sativex showed an 83% 1 year survival rate with a median survival of over 662 days as compared to 369 days in the control group. (23)

Cannabinoids can express anti-neoplastic activity through binding with the CB1 receptor (This is for THC, anandamide, 2-AG). Phytocannabinoids other than THC (CBD, CBG, CBC, and others) do not bind to the CB1 or CB2 receptors but inhibit the enzymes (FAAH, MAGL) that degrade
anandamide and 2-AG, thus causing prolonged binding of endocannabinoids to the cannabinoid receptors, which also has an anti-neoplastic effect. The brain has the highest density of CB1 receptors in the body. Numerous studies in vitro and in animal models suggest that cannabinoids can inhibit gliomas. (17) It has been found that other cancer cell lines are also inhibited by cannabinoids in vitro, such as adenocarcinomas of the lung, breast, colon and pancreas, and also myeloma, lymphoma, and melanoma. Cannabinoids can enhance the activity of certain chemotherapeutic agents, and through the down-regulation of p-glycoprotein may also be able to reduce chemotherapy resistance. The majority of studies evaluating cannabis in cancer patients have been to evaluate its ability to address symptoms of cancer or cancer therapies, such as pain, nausea, or anorexia. These studies have found that cannabis can work well for cancer pain. Cannabinoids have been found to work synergistically with concurrent opiate medication for a better pain response in the cancer patient. (24) One of the highest callings for medical cannabis in benefiting the cancer patient is its ability to control nausea, often better than the existing anti-emetic armamentarium. The synthetic cannabinoid, dronabinol (Marinol™) was originally designed for this purpose, and can work well. Marinol™ is a 100% synthetic THC analog, and some persons taking it report hallucinations. This side-effect is due to the lack of modulating phytocannabinoids such as CBD or CBG to temper the psychoactivity of the Marinol™. There is good evidence that plant-based cannabinoids work better to relieve nausea without the potential for adverse events. This is due to the tempering effect that a full spectrum cannabis extract with its full complement of phytocannabinoids has on the psychotropic effect of THC. (25) Appetite stimulation is the third reason that cannabis has value to the cancer patient. Famous for creating “The Munchies”, THC and CBD can help to relieve nausea and also increase appetite better than many of the existing aperient formulations. Cannabis is the only anti-emetic that is also an appetite stimulant, although for anorexia-cachexia one study found it to be no better than placebo. This RCT was placebo-controlled to compare cannabis with the synthetic cannabinoid, dronabinol (Marinol™) in 243 human patients with cancer-related anorexia-cachexia syndrome. It was found that there was no difference among any of the two treatments and the placebo with respect to affecting appetite or quality of life. (26) Anecdotally, veterinary oncologists are observing that THC does not stimulate appetite in more than 50% of dogs, although this may be dose-dependent. Cats have been observed to exhibit a better appetite response to THC than dogs. Terpenes, which make up 10% of the total production of active molecules by the cannabis plant, are an integral part of the “Entourage Effect” that contributes to the clinical efficacy of cannabis. Certain terpenes, such as limonene has been shown to cause apoptosis of breast cancer cells. Additionally, for cancer pain, terpenes contribute substantially. Both phytocannabinoids and terpenes reduce inflammation and pain via inhibition of COX-2 and PGE2α as well as a number of other mechanisms of action such as the TRPV1 pathway.

Veterinary Cancer Patient Cannabis Considerations

For the veterinary cancer patient, legal constraints to the use of THC must be accounted for in a reasonable and safe fashion. Veterinarians cannot prescribe or dispense THC. However, pet owners will come to a veterinarian asking for help using cannabinoids to treat their pet’s cancer who have full access to state-legal dispensaries and the THC products they sell. The veterinarian should explain the risks and the problems with the current legal landscape, and the potential to send a patient to the ER with high doses of THC. The vet should explain that they cannot legally recommend or prescribe these Schedule One controlled substances. If the owner persists, then the veterinarian can give advice that will help to create a successful outcome free of unwanted side effects.

Studies in the 1970s discovered that dogs have a very high density of CB1 receptors in their hind brain that govern balance and cardiovascular function. (27)(28) This is why a few dogs who are naïve to THC, when exposed to a sufficiently large amount of THC, especially when in combination with chocolate, as is commonly found in human “edibles”, have had reported deaths. (29) These early studies found that dogs, can develop tolerance to THC’s adverse effects in about a week when THC is introduced in small amounts initially and gradually increased over time. Once tolerance is achieved, dogs were able to handle escalated doses a 100 times higher than the original dose that had caused the adverse response. Once tolerance has been developed, the
canine patient can then tolerate larger doses if their condition warranted dose escalation. Studies (30) and reports from human and veterinary oncologists, veterinarians, medical cannabis physicians, pet owners, and human cancer survivors, although anecdotal, indicate that the most successful approach to the use of cannabinoids in the cancer patient involves the oral use of a combined formula containing both THC and CBD, usually in a 1:1 ratio. This combination enhances the anticancer activity of the formula as these two cannabinoids have similar but different mechanisms of action against cancer, and work together synergistically. Using a “ratio approach” to the blending of CBD with THC can help to reduce the dose of THC that is needed to inhibit tumor growth. The use of CBD also reduces the unwanted side-effects of THC, such as psychoactivity, convulsions, discoordination, and psychotic effects in humans and dogs, and in the dog, specifically, static ataxia.

The best approach to the blended use of THC and CBD from cannabis in a cancer patient is to use products that have THC and CBD formulated into a specific ratio of CBD:THC. Ratios can help to reduce the side-effects from THC and maximize the anti-neoplastic activity of the treatment. The protocol is to first start with cannabis that is hemp, which would have a ratio of CBD to THC of about 25:1. The use of this small amount of THC present in the hemp will help to “tolerize” your patient to the larger doses of THC that will be used for anti-neoplastic effects in a 1:1 ratio of CBD:THC. CBD-dominant strains of cannabis with very low THC, but higher than hemp levels of THC (>0.3%) can be used to increase the patient’s tolerance to the higher doses of THC that are needed for cancer.

In several oncology cases reported to this author by a board certified veterinary oncologist who helps clients who are giving their pets THC and CBD for cancer to do that safely and reduce harm, found that by starting low, going slow, and staying as low as possible with the THC dosage, that tumor remission was achieved in each case with less than 1.0 mg/kg BID of THC. (31) Once the dog has been on the hemp for a week at a dose of 0.5-1.0 mg/kg BID of CBD, it should be tolerant of increases in its THC dosage, which will better address the anti-neoplastic effects of cannabis. Starting dosages for the THC are also in the 0.1-0.5 mg/kg BID range, with the lower doses being less likely to cause adverse events. In this author’s experience, patients who were taking 100% CBD for their tumors, with zero THC, would show an observed reduction in the size of the mass in about 6 weeks for that reduction to be substantial, as documented with photographs. The dose used in these successful cases was 0.5-1.0 mg/kg BID of CBD. Not all tumors respond to this dosage, but in those that do respond, it is remarkable to watch.

**SUMMARY**

The available literature suggests that the endocannabinoid system can be targeted to suppress the evolution and progression of certain types of cancer and the pain, emesis and appetite syndromes associated with these diagnoses and treatments. In spite of the fact that there are no controlled clinical trials of cannabinoids in cancer patients as anti-neoplastic agents, there still is sufficient evidence to support the use of cannabinoids for veterinary patients with cancer in general, although specific tumor types may be more or less resistant to their beneficial effects. The biggest problem to date is the legal landscape, since cannabinoids have been shown to be quite safe when given in controlled amounts. Large population studies in humans have found no correlation between smoking cannabis and increased risk of respiratory symptoms/chronic obstructive pulmonary disease or lung cancer. (32) The same has been found to be true with bladder cancer prevention and cannabis. (33)

Phytocannabinoids can be used concurrently with chemotherapy and have been found to not interfere with chemotherapy efficacy in the tumors and chemotherapy agents measured. (34) Determining the effective dosage for an individual patient and tumor type will improve outcomes, and is work that still needs to be done. Cannabinoids can induce autophagy, apoptosis, cell cycle arrest, reduce angiogenesis, tissue invasion and metastasis, without affecting normal cells. Cannabinoids can reduce pain, nausea and improve appetite. All of these actions make the use of cannabinoids for cancer very attractive to both the practitioner and the pet owner.

**BIOMEDICAL ACTIONS AND POTENTIAL VETERINARY APPLICATIONS** (43)

**Pain, Inflammation and Immunomodulation**

- Effective for both acute and chronic pain by centrally and peripherally modulating nociception
- CBD affects T-cells resulting in a mild generalized immunosuppressive effect
CBD has been found to have potential benefit for arthritis and psoriasis in humans.

**Epilepsy**
- CBD attenuates seizures in experimental models of epilepsy in animals
- THCV inhibits CB1 receptor activity resulting some anticonvulsant activity

**Anxiolytic**
- CBD exerts benzodiazepam-independent activity, postulated to be via post-synaptic 5-HT1A receptors

**Neuroprotection**
- CBD acts as an antioxidant and as such has been suggested for Alzheimer’s, Parkinson’s and Huntington’s diseases.

**Anti-emesis**
- CBD in animal models has been found to be effective for the control of vomiting that is unresponsive to 5-HT-3 agonists such as metoclopramide or ondansetron

**Diabetes Mellitus**

**Bone formation**
- Cannabinoids stimulate the stem cells responsible for fracture healing and bone formation, as well as reducing bone loss by controlling bone reabsorption

**Cancer**
- Many of the cannabinoids have anti-apoptotic effects and reduce neoplastic proliferation in selected tumor cell lines
- Anecdotal reports from both human and veterinary patients indicate the potential for complete remission and possibly even cure of a number of different neoplastic diseases

**Anti-microbial**
- Both CBC and CBG have potent anti-bacterial effects including against MERSA (MIC of 0.5-2 mcg/ml)

**VETERINARY EVIDENCE-BASED APPLICATIONS**

**SURVEY-STUDIES**
1. Pet Owner Experiences with Hemp Products (Kogan1)
   This was an on-line survey of pet owners who visited a CBD for pets website.
   **Results:**
   Most common uses for hemp products reported by pet owners on the survey: (D=dog; C=cat)
   Listed in descending order of frequency of survey response
   - Pain management and arthritis (D)
   - Anxiety (D&C)
   - Cancer (D)
   - Inflammation (C)
   - Disturbed sleep (D&C)

2. Demographics and Dog Owner Perceptions of Cannabis (Kogan 2)
   This second study was a follow-up on-line survey of pet owners using social media (1068 respondents)
   **Results:**
   Most common uses for hemp products reported by pet owners on the survey: (NOTE: Dog owners only were included in this study)
   Listed in descending order of frequency of survey response:
   - Pain relief
   - Anxiety
   - Reduction of inflammation
   - Epilepsy
   - Cancer
   - Arthritis
   - Allergies
Compilation of Veterinarian Uses of Cannabis in their patients
Although this is not controlled data, this author has personally spoken with or corresponded with hundreds of veterinarians who have been using industrial hemp extracts in their practices over the past 2 years. Over 40,000 bottles have been distributed to veterinarians in this time period. These uses parallel those detailed by the two previous surveys described above.

- Pain management
- Anxiety and behavior problems
- Epilepsy
- Cancer treatment
- Cancer treatment side-effects
- Inflammatory bowel disease

As the clinical use of cannabis becomes more common place we are seeing more detailed studies emerging to help fill in the many blanks that are still lacking in our comprehensive understanding of the safety and efficacy of this very popular and quite effective emerging veterinary plant-based therapeutic. The following studies are some of the few published studies in the dog. An article in the British Medical Journal by Walter Dixon MD an early pharmacologist, is the first published evaluation of cannabis in dogs and cats, written just before the turn of the 20th century in 1899. (35) It is the first publication where static ataxia is described. Since then, studies have been few and far between due to the controlled substance status of cannabis, and the negative effect that has on research study funding.

EARLY STUDIES IN THE DOG
Research performed in the 1970’s by the Department of Defense, explored whether marijuana could be “weaponized”. Research animals were administered radioactive-labeled THC intravenously at escalating dosages. As a result, researchers found that dogs, as compared to pigeons, monkeys, guinea pigs, rats and mice, had the highest concentration of THC (now known to be bound to CB1 receptors) in the cerebellum. The canine THC detected was more dense than in any of the other species studied (27).

Dogs, as compared to other species studied developed a pathognomonic neurologic condition termed “Static Ataxia” which results from the THC binding to CB1 receptors in the dog’s cerebellum. Previous studies had found that the minimum dose of THC administered IV to create static ataxia was 0.5 mg/kg IV. (28)

Tolerance to the “behavioral” effects of THC in the dog developed after daily injections were given. McMillan found that a dose of 2 mg/kg IV produced marked static ataxia, evidenced by “swaying movements, hypersensitivity to moving objects and prance-like foot placement.” Most of the dogs in this study group developed tolerance to these adverse neurological effects rapidly after the first administration of 2 mg/kg of THC. Subsequent injections continued to increase the degree of tolerance to THC. The magnitude of tolerance developed in these canine studies was in excess of 100 fold. (36)

VETERINARY UNIVERSITY STUDIES
COLORADO STATE UNIVERSITY

1. Pet Owner Experiences with Hemp Products (37)
This was an on-line survey of pet owners who visited a CBD for pets website. Results:
Most common uses for hemp products reported by pet owners on the survey:
(D=dog; C=cat)
Listed in descending order of frequency of survey response
- Pain management and arthritis (D)
- Anxiety (D& C)
- Cancer (D)
- Inflammation (C)
- Disturbed sleep (D&C)

2. Demographics and Dog Owner Perceptions of Cannabis (38)
This second study was a follow-up on-line survey of pet owners using social media (1068 respondents)
Results:
Most common uses for hemp products reported by pet owners on the survey:
Listed in descending order of frequency of survey response:

- Pain relief
- Anxiety
- Reduction of inflammation
- Epilepsy
- Cancer
- Arthritis
- Allergies

3. Pharmacokinetics of cannabidiol administered by 3 delivery methods at 2 different dosages to healthy dogs.

This study was designed to determine the pharmacokinetics of CBD in healthy dogs. A sample population of 30 healthy research dogs were assigned to receive 1 of 3 different formulation at a dose of 75 or 150 mg q12 h for 6 weeks. The dosage formats were: 1) Liquid oil infusion administered to the oral mucosa; 2) oral capsules with microencapsulated oil beads; 3) transdermal application. Serial CBD plasma concentrations were measured over the first 12 hours and repeated at 2, 4, and 6 weeks. Greater plasma concentrations were measured with the oral CBD-oil infused formulation. The plasma half-life of CBD administered via this route after the 75 mg and 150 mg doses respectively were 199.7 +/- 55.0 and 127.5 +/- 32.2 min. This study found that blood levels are dose proportional, as expected and the oral liquid CBD absorbed transmucosally was the superior formulation of the three formulations tested, with orally administered microencapsulated beads the second-best formulation in terms of pharmacokinetic profile. The CBD had a peak at 2 hours post ingestion and a half life of 4-6 hours. (39).

4. A report of adverse effects associated with the administration of cannabidiol in healthy dogs.

A study that is currently in press for the Fall of 2018 was performed at Colorado State University’s College of Veterinary Medicine, Neurology Department. The principal investigator, Stephanie McGrath, who is a veterinary neurologist and Assistant Professor at CSU’s Veterinary Teaching Hospital, conducted a 6-week high dose evaluation of the tolerability of two high doses of CBD in healthy beagle dogs. A sample population of 30 healthy Beagle dogs were randomly assigned to receive one of three formulations: Microencapsulated oil beads, CBD infused oil or CBD infused transdermal cream for 6 weeks. Two dosage tiers were evaluated in this study, 10 mg/kg/day and 20 mg/kg/day. These dosages far exceed the dosages used in the two efficacy studies that followed this by a factor of 2X and 4X greater. The two efficacy studies evaluated the use of CBD for refractory epilepsy and osteoarthritis in the dog. Complete blood counts, chemistry panels, urinalysis and pre- and post-prandial bile acids were performed at 0, 2, 4 and 6 weeks. Elevations in alkaline phosphatase double the high end of the reference range (140 IU/L) were observed in some dogs (11/30:36%) after being on the CBD for 4 weeks, although it did elevate in some dogs at 2 weeks, especially at the higher dosing tier. Long term liver toxicity was not evaluated in this study, although bile acids and liver enzymes remained normal for all dogs throughout the study. None of the dogs receiving the transdermal formulation developed elevated alkaline phosphatase values. All dogs experienced mild diarrhea, although there was no correlation with formulation or dose. 6 out of the 30 dogs developed vomiting, but there was no significant difference between the occurrence of vomiting and CBD dose or formulation.

Erythematous pinnae were the next most commonly reported clinical sign in this study. These otic changes were seen in 36% of dogs with the otic changes becoming more severe after 2 weeks in the 10 mg/kg/day dosage group for all three formulations. The transdermal crème had more incidences of otic changes than either the transmucosal or oral routes of administration, which is understandable since the transdermal crème was applied to the inside of the pinna. Less common findings included ocular discharge in 10/30 dogs (33%) and nasal discharge in 10/30 dogs (33%). 5 dogs (17%) had salivary staining on their feet and occasionally ventral abdomen. Two dogs had spontaneous prolapsed glands of the nictitans. One dog had a transient elevated body temperature (104.2°F). It was also observed that some dogs would salivate following administration of the CBD-infused oil formulations at both doses. The study concludes that CBD seems to be well tolerated in the dog at these high dosages, but emphasize that a larger and longer in duration safety study is needed to evaluate the very long-term effect of CBD on the liver
and its association with diarrhea (40) (41).
5. Efficacy Studies: Pilot Trials for Osteoarthritis and Refractory Epilepsy (in press)

Osteoarthritis

The osteoarthritis (OA) wing of this efficacy study consisted of 24 client-owned dogs with clinical evidence of OA radiographically and who had an identifiable lameness. A double-blind, randomized, placebo-controlled, study design was utilized, with each study group receiving medication for 6 weeks and a placebo for 6 weeks. The treatment group received 2.5 mg of CBD oil q 12 hours. Gait analysis and an activity monitor were used to gain objective data, and a behavioral questionnaire was given to the dog owners which provided subjective information. The study results for OA were not yet available at the time of this publication. (40)

Refractory Epilepsy

The epilepsy segment of the study consisted of 16 client-owned dogs who were diagnosed with idiopathic refractory epilepsy, having 2 or more breakthrough seizures per month while receiving conventional anti-convulsant therapies. Inclusion criteria included a normal neurologic exam and a normal epilepsy workup with an MRI and CSF analysis. Nine (9) dogs were randomly assigned to the treatment group and 7 to the control (placebo) group. The treatment group received 2.5 mg/kg CBD oil q 12 hours by mouth. The control group received placebo oil for 12 weeks. Study subjects were required to stay on their standard anticonvulsant drugs (AED). Routine blood work and CBD levels were determined every four weeks. AED levels were measured at the conclusion of the trial.

67% (6/9) of the dogs in the treatment group experienced a greater than 40% reduction in average monthly seizures during the study; whereas only 29% (2/7) of the dogs in the control group had a greater than 40% reduction in average monthly seizures.

Elevations in alkaline phosphatase (ALP) were recorded for the treatment group, and one dog in the control group. The single control dog had previously measured elevations in ALP so this elevation was not considered to be relevant to the study. 6 dogs (67%) in the treatment group had elevations in ALP measured at the end of the study. The mean ALP value was 619 IU/L (range 15-140 IU/L).

AED concentrations in the treatment group for phenobarbital decreased in 2/7 dogs (29%) and increased in 5/7 dogs (71%). In the control group phenobarbital levels decreased in 3/5 dogs (60%) and increased in 2/5 dogs (40%); there was no significant change in either group. This is an interesting finding to note, because there has been a concern that CBD, which is metabolized through the P450 group of cytochromes, might interfere with the drug disposition of pharmaceuticals that also are metabolized through that pathway. From the results of this pilot study, that effect is not apparent, at least with respect to phenobarbital levels.

Potassium bromide (KBR) levels in the treatment group decreased in 2/3 dogs (67%) and increased in 1/3 dogs (33%). In the control group KBR levels decreased in 1 out of 2 dogs (50%) and increased in 1 out of 2 dogs (50%). There was no significant change in either group, although the total number of study subjects was low in this pilot study. This research and the osteoarthritis section of this study have not yet been published, pending the results of the plasma analysis of cannabinoid levels that were measured at 0, 4, 8, 12 weeks and the completion of the efficacy study of the effects of CBD on osteoarthritis. (40)

6. AKC Canine Health Foundation study of cannabidiol use for refractory epilepsy in dogs

The American Kennel Club Canine Health Foundation has granted nearly $400,000 in funding to this research group at CSU for a larger, expanded study with 60 dogs, as a result of the positive results of this pilot work, with respect to the use of CBD oil to address refractory epilepsy. This study will also be looking at uncontrolled epileptics having 2 or more seizures per month while receiving standard therapy. In this expanded study, which will use a cross-over design, each subject will receive 12 weeks of treatment or placebo with a 4-week washout period between treatments. This study began in January 2018, and is currently enrolling patients. (40)

CORNELL UNIVERSITY

The objectives of this recent Cornell study were to determine the oral pharmacokinetics and safety, as well as analgesic efficacy of using CBD in dogs with osteoarthritis (OA). Single-dose pharmacokinetics were performed using two different doses of 2mg/kg and 8 mg/kg of CBD in a carrier oil. From this data, a prospective, randomized, placebo-controlled, double-blind crossover study was conducted using 16 client-owned dogs with radiographically confirmed evidence of osteoarthritis who were enrolled and who completed this study. Dogs were randomized to receive
either 2 mg/kg q12h orally of CBD oil, or a placebo consisting of olive oil with a benign herbal extract at a similar volume q 12 h for 4 weeks. Subjects were given a 2-week washout period and then the treatments were crossed-over and each subject received the other treatment twice daily for 4 weeks. Veterinary assessment of lameness, movement, and response to manipulation, owner questionnaires (Canine Brief Pain Inventory (CBPI), Hudson activity scale), objective kinetic analysis on a pressure-sensitive walkway, hematology and chemistry analysis were obtained at weeks, 0, 2, and 4 for both oils. Statistical analysis was performed on the results, with a p<0.05 considered to be significant. Pharmacokinetics showed a half-life of elimination of 4 - 6 hours at both doses and no observable side-effects. Median maximum concentration of CBD oil was 102 ng/ml (61 - 132 ng/ml) and this peak was reached at 90 minutes following administration of the single dose of 2 mg/kg. The investigators on this study decided that since the pharmacokinetics of the 2 mg and 8 mg doses were so similar they chose to use the lower of the two doses for the efficacy wing of this study. Assessment of pain and mobility showed a significant decrease in pain and increase in activity (p<0.001) at week 2 and 4 during CBD treatment as compared to baseline at each bi-weekly evaluation. It was found that the CBD oil resulted in reduced pain scores when compared to baseline on both bi-weekly examinations (p = 0.03). No side effects were reported by owners, but serum chemistry demonstrated an increase in serum alkaline phosphatase (9/16 dogs: 56%) while receiving the CBD oil, which reached significance at week 4 (p<0.005). The authors of this study conclude that the dogs with OA who received 2 mg/kg q 12 hours were found to be more comfortable and active with very few undesirable side-effects compared to placebo. (42).

POTENTIAL AREAS OF FUTURE VETERINARY RESEARCH

**Oncology**
- Anti-neoplastic activity
- Ameliorate cancer therapy side-effects
- Appetite
- Nausea
- Well-being

**Terminal disease – Hospice care giving**

**Neurodegenerative conditions**
- Spinal Stenosis
- GME
- DM

**Ophthalmology**
- Glaucoma
- Anterior Uveitis

**Pain Management**
- Synergy with other pain meds?
- Condition specific dosing for pain

**Behavioral Studies**
- Anxiety
- Noise phobias
- Social tension
- Aggression

**DOSING RATIONALE FOR VETERINARY CLINICAL USE OF CANNABIS**

Dosages in veterinary species have not yet been determined through Phase One tiered dosage studies where clinical response to incremental dosage escalation is measured to determine the optimal dosage for a given condition. In the two university-based studies from Cornell and Colorado State University, pharmacokinetics have been determined at dosages significantly higher than dosages that have been reported through hundreds to thousands of dog and cat administrations by veterinarians who have been recommending hemp based phytocannabinoids prior to Phase One studies, based on effective clinical responses. The higher dosages used in these pK studies confirmed that plasma levels of cannabidiol are detectable, and persist for sufficient time (5-6 hour half times) to allow for clinical response in
conditions of osteoarthritis and refractory epilepsy. The lower dosages reported to be clinically effective by veterinarians are on a order of 4-5 times lower than the university study dosages. It has been reported in the literature that there is a biphasic dosing response to phytocannabinoids, where lower dosages have one benefit, and higher doses have other benefits. This has been reported in the human literature, but not yet in the veterinary literature. (44) (45) The lower dosage tiers are referred to as: “Micro dosages” and the higher dosing tiers are referred to as “Macro dosages”. Microdoses can be considered to be less than 0.5 mg/kg BID of cannabinoids; macrodoses would be equal or greater than 2.0 mg/kg BID. Individual responses to dosage levels may vary. Based on anecdotal reports by veterinarians and pet owners who are using microdoses, the effective dosages used in dogs and cats have been reported to be as much as 40 times less than the macrodoses used in the CSU Safety study or 5 times less than used in the CSU efficacy study. EQUINE DOSING STUDY This author, in an unpublished study, gave 30 horses in three different stables, dosages of 25 – 50 mg of CBD in a zero-THC hemp extract once or twice daily to address complaints of anxiety, gait abnormalities, mild to severe laminitis, and metabolic syndrome. Study subjects averaged around 1000 pounds. It was found that for anxiety and mild cases of lameness or gait abnormalities, that administration of 25 mg once or twice daily was adequate to elicit a response with regards to anxiety from loading up into a trailer or at events, or for mild gait abnormalities. In one stable the horses were only able to be given their dose once daily, yet that single dose still produced good clinical results. For more severe conditions such as moderate laminitis, other sources of lameness, or metabolic syndrome, it was found that 50 mg once or twice daily was sufficient for clinical response. When horse owners were asked to discontinue giving the hemp extracts, so as to determine withdrawal times for CBD, for situations in which the horses may be drug tested for an event, many refused to stop administration of the hemp, as they were very pleased with their horses' response to the hemp extracts. Horses have evolved to be very efficient in absorbing fats from their diet, as their natural diet of forage is very low in fats and oils. pK studies in the equine are very much needed to better understand dosing intervals and levels. (46) POTENTIAL PROBLEMS WITH VETERINARY USE OF PHYTO-CANNABINOIDS UN-WANTED SIDE-EFFECTS In the macro-dosing safety studies performed by CSU and Cornell, elevations in serum alkaline phosphatase, diarrhea and sedation were observed. These adverse events have been reported, although less frequently with the use of micro-dosing. This same dose-dependent adverse event occurrence has been reported in the human as well. (45) ADVERSE EVENT REPORT FROM NASC FOR HEMP PRODUCTS The National Animal Supplement Council (www.NASC.cc) tracks adverse events in nutraceutical and nutritional ingredients submitted by its 175 members and has data on millions of administrations of these ingredients in veterinary species. The Adverse Event Report (AER) for all forms of hemp in veterinary species has recorded 4,746,313 administrations sold for all forms of hemp to dogs, cats and horses. Since this data has been collected starting in 2010, there have been three adverse events reported in dogs. 2 in 2013, and 1 in 2017. These were not deemed serious adverse events, which would have an incapacitating effect or a non-transient health effect. An adverse event as defined by the NASC is a type of complaint where a patient has suffered any physical effect or health problem that may or may not be connected to or associated with the use of a product. (47) DRUG HERB INTERACTIONS It's reported that in human medical marijuana patients, physicians have observed only rarely, clinically significant drug interactions. These two cannabis researchers state, based on their clinical experience and the pooled experience of medical cannabis physicians worldwide: “there is no drug that cannot be used with cannabis, if necessary.” (9) Depending on the strength of the affinity of the metabolite for the metabolizing cytochrome, serum levels of cannabinoids or pharmaceuticals may increase with inhibitors or decrease with enzyme inducers. It is known that THC and CBD, in the human, are oxidized by the p450 cytochromes (CYP2C9, 2C19, and 3A4). It is assumed that this is similar to the metabolic pathways in the dog and other veterinary
species, but studies still need to be conducted in each species to detail possible inter-species variation. McGrath (40) in her refractory epilepsy study measured both post-pill phenobarbital levels and potassium bromide (KBR) levels in her study subjects at the end of the 6 week study period. She found no statistical difference between the treatment and placebo groups for either anti-convulsant. This may indicate that, in spite of the theoretical potential for herb-drug interaction between CBD and P450 metabolized drugs, that at least in dogs on anti-convulsants, the interference may not be clinically relevant. Regardless, it is good medical practice to retest important serum drug levels 2 weeks following initiation of CBD therapy, especially if using macro-dose protocols. McGrath reported that the small study group size in this pilot work may not have given an accurate indication of whether there really is herb-drug interaction or not, and she these levels will be analyzed not just at the end of her AKC-Funded clinical trial, but at regular intervals though out that much larger clinical trial. (40)

WHAT DOES THE FUTURE HOLD FOR VETERINARY CANNABIS?

1. HEMP IN ANIMAL FEED INITIATIVE WITH FDA-CVM AND AAFCO
   - Current initiative to submit a Feed Additive Petition to the FDA-CVM to have hemp seed approved as an ingredient for dogs, cats and horses.

2. PLANT BREEDING FOR SPECIFIC CULTIVARS
   - Pharmaceutical companies such as the UK company, GW Pharmaceuticals that has recently had their Epidiolex drug containing CBD approved by the US FDA has been genetically breeding cannabis plants to increase individual cannabinoids to develop into additional pharmaceutical and OTC products.

3. FDA VETERINARY DRUG APPROVAL FOR PATENTED SYNTHETIC CANNABINOIDS
   - Several companies are following the FDA track for approval of synthetic cannabinoids as veterinary drugs.

4. CANADIAN FEDERAL LEGALIZATION IS HERE
   - What does that mean for US/Canadian Veterinarians
     - The Canadian legislation does not contain language specific enough to allow the use of medical cannabis by veterinarians, but that is currently being addressed by the Canadian veterinary community.

5. GMO CANNABIS IS COMING (GOOD OR BAD?)
   - This is an inevitable development based on current genetic technology. It remains to be seen if this will be a beneficial development.

CONCLUSION

Cannabis has come a long way since the days of Reefer Madness, but there still is much research and clinical work to be done before veterinarians can be 100% comfortable about using this unique and emerging popular therapeutic. The regulatory environment is loosening its restrictions on research and clinical use, and both veterinary clients and veterinarians are eager to get involved. University studies have shown safety and efficacy at macro-dosing levels, and NASC adverse event reports and field reports from veterinarians and their clients indicate similar safety and efficacy at lower doses. Herb-Drug interactions are a concern, given cannabidiol’s metabolic disposition through the P450 cytochrome system, but clinically, reports indicate that does not seem to be a problem.

FDA-approved cannabis drugs for humans are on the market, and similar approval for veterinary labeled drugs can not be far off. Similarly genetic breeding of cannabis cultivars for product development will improve the quality of veterinary and human drugs and cannabis nutraceuticals, and genetic engineering of the cannabis genome is awaiting Federal legalization.

REFERENCES

controlled clinical trial from the Cannabis-In-Cachexia-Study-Group. J. Clin Oncol. 2006;24:3394-400.


30. Scott KA, Dalgleish AG, Liu WM. The Combination of Cannabidiol and $\Delta^9$-Tetrahydrocannabinoid Enhances the Anticancer Effects of Radiation in an Orthotopic Murine Glioma Model. Mol. Cancer Ther 2014;13(12);2955-67


40. McGrath S Cannabinoid Clinical Trials in Dogs - CSU Paving the Way. In: Proceedings of the AVMA Annual Conference, Denver, CO, July 2018


44. Tishler J. Microdosing for the Medical Market: Why Who and How. Paper presented at the Institute for Cannabis Research, Colorado State University, Pueblo, April 27-28, 2018


47. NASC Ingredient Risk Report for all forms of hemp: July 18, 2018; www.NASC.cc
Top Twenty-one Tips from the ER  
Jon Geller, DVM, DABVP Emeritus, Canine and Feline Practice  
Fort Collins Veterinary Emergency and Rehabilitation Hospital, Ft. Collins, CO

1. Anticipate

A good hockey player plays where the puck is. A great hockey player plays where the puck is going to be.

• Kirby’s Rule of 20: The Art and Science of Anticipation

2. Bolus, re-assess, repeat

• Crystalloid Bolus: The “CSU Way”
  – Dogs: 10 mls/pound/10-15 minutes
  – Cats: 5 mls/pound/30 minutes

• The perfect colloid bolus

  Fill a 60 cc syringe with:
  – 30 mls 7% hypertonic saline
  – 30 mls Vetstarch

• Vetstarch: (130/0.4) -tetra starch is smaller and does not last as long, so can be given at a higher rate since it does not affect coagulation as significantly: 50 mls/kg over 24 hrs = 2mls/kg/hr (versus 1 ml/kg/hr for hetastarch.

  Give as an 8ml/kg bolus over 5 minutes, follow with isotonic IV fluids

3. FAST Scans

• Quick, sensitive method of detecting abdominal fluid

• Accurate predictor of need for surgical exploration in cases of hemoabdomen

• 4 positions, score 1 for presence of fluid at each position.
  – Score: 0-2, case can be medically managed
  – Score: 3-4, surgical exploration warranted

• FAST SCAN Targets

4. Sacrococcygeal blocks

• Especially useful in very sick cats

• Eliminates the need for sedation and reduces anesthesic requirements.

• How to do a Sacrococcygeal Block

5. Hyperkalemia in the Cat

• Average 10# cat:
  – 3 mls 10% Ca Gluconate over 3 minutes, IV
  – Monitor with EKG (or Alivecor)
  – Protective of cardiac muscle resting potential: life saving

• Recognizing hyperkalemia on the ECG
5. Use Checklists, especially for anesthesia and surgery
   • After introduction of checklists in 8 hospitals (4,000 patients):
     • 36% drop in complication rate
     • 47% drop in mortality rate
     • Check lists in veterinary medicine

6. Take the last-minute X-Ray
   • Just prior to inducing

7. Inducing emesis in the cat: it’s not that easy
8. GDV?- Large bore IVC, 1 L Fluids, Pain meds, trocharize
9. 9 ways to diagnose Ethylene Glycol toxicity
   (EG Test-can be non specific, insensitive)
10. Recognizing and treating shock in cats
    Hypothermia-Bradycardia-Hypotension
11. Air contrast: It’s cheap
    • Pneumocolongrams- inject 60 mls/air into the colon (+/- red rubber catheter) just prior to taking film
    • Pneumogastrograms- Inject 60-120 mls of air via orogastric tube just prior to taking film
12. Treating cluster seizures
    1) Valium 1mg/kg IV or intra-rectal-
       - Midazolam Intranasal (atomizer)
    2) Bloodwork to rule out hypoglycemia, ionized hypocalcemia
    3) Phenobarbitol loading dose (if not currently taking) 20 mg/kg total IV divided q 1-2 hrs
    4) Keppra 20 mg/kg IV/IM – continue tid
       * Extended release 500 or 750 mg bid
    5) Valium CRI .1-.2 mg/kg/hr
    6) Propofol CRI- ventilate as needed
       • The Atomizer- $4.15 each
13. Use of Acupuncture and Laser Therapy in the ER
    • Indications:
      1. Acute and chronic pain
      2. Neurologic dysfunction
      3. Musculoskeletal disorders
      4. GI motility disorders
      5. Circulatory compromise
      6. Urinary disorders
      7. Wound healing
      8. Intra-abdominal healing
14. Oxygen delivery
   • Oxygen concentrators
   • Nasal canulas

15. Trickle feeding
   • Accurate delivery of nutritional requirements
   • RER, 24 hrs = (BW kg x 30 + 70)
   • Adjust for metabolic and disease status
   • Clinicare: 1 ml/kcal
   • Place feeding tube down one nostril (medial and ventral), use lidocaine gel
   • Pass into distal esophagus
   • Confirm placement with radiograph
   • Use IV pump to deliver trickle

16. DKA Protocols
   • Inpatient, clinical signs, metabolically unstable
   • Outpatient, stable: Glargine insulin bid IM with intermittent SC

17. Mini-tips
   • Lidocaine gel for blood draws and IVC placement
   • Parvo enemas
   • Continuous Doppler blood flow monitoring
   • Spica Newspaper splint for mid-humeral fractures
   • Permissive hypothermia: 89-92 F. during extensive surgery. Use acepromazine premed as to protect against hypothermia induced arrhythmias.

18. Facilitated cut-downs: For hypovolemic patients
   • Use edge of bevel to cut skin adjacent to vessel
   • Pull cut skin over vessel to isolate and elevate
   • Place catheter directly into vessel, secure with suture tie circumferentially

19. Salvage Protocol for blocked cats
   • 65% of 15 cats responded (IVECCS study)
   • Protocol (these cats are not “sick”) includes Pain meds (buprenex), Cystocentesis (with 3 way stop cock for complete drainage), SC fluids, Dark, quiet cage, Low dose acepromazine or dexmedetomidine, Repeat cysto, pain meds and fluids as needed, +/- Class 3b or 4 Laser over bladder
20. Drug combos for sedation

- Neurologic sparing: Opiod (low dose)+dissassociative
- Cardiac sparing: Opiod + Benzo
- Respiratory sparing: Opiod + alpha-2 agonist
- Renal sparing: Opiod +benzo+ disassociative

21. Kitty Magic

- 0.05–0.1 MLS/4.5 kg cat. The drugs are combined in the same syringe and administered IM (decrease the dose by about 25% for IV administration). Use the low-end dosing for deep sedation and the high end for anesthesia.
  - Butorphanol
  - Dexmedetomidine (+O2)
  - Ketamine
A LOW TECH APPROACH TO THE DIAGNOSIS OF ANEMIA
L. Ari Jutkowitz, VMD, DACVECC
Michigan State University, East Lansing, MI

Anemia is commonly seen in veterinary emergency and critical care medicine. Patients may be brought in with the presenting complaint of anemia or may develop anemia during hospitalization as a result of their underlying disease or treatment. Anemia may contribute to patient morbidity, cost of treatment, and length of stay, frequently necessitating expensive interventions such as blood transfusion while the underlying disease is being treated.

Classification of Anemia
Anemia seen in veterinary patients may be classified into three broad categories that relate to cause; blood loss, hemolysis, and decreased production. Classification of anemia in this way is not merely academic but is crucial to the workup of anemic patients. Because regenerative and non-regenerative anemia have different sets of differentials and diagnostics, this classification will guide further testing and provide useful prognostic information.

Three simple, in-house diagnostic tests can be performed in all anemic patients to help classify their anemia. These tests are inexpensive, easy to perform, and will frequently provide a great deal of information about the cause of the anemia. They can all be performed in approximately 5 minutes, allowing the clinician to classify the anemia and provide an appropriate diagnostic plan while the owner is still present at the hospital during the initial exam.

The first test is the packed cell volume (PCV) and total solids (TS). The importance of interpreting the PCV in conjunction with the total solids cannot be overemphasized. If the PCV and TS are both low, acute blood loss should be suspected. In contrast, a low PCV with normal total solids would be consistent with hemolysis or decreased red blood cell production. To differentiate these two clinical entities, the plasma of the spun sample should be carefully evaluated for the presence of hemoglobin or bilirubin that may suggest intravascular or extravascular hemolysis respectively.

The second test that should be performed is the blood smear. Blood smears are useful for differentiating hemolysis from decreased production anemia as the presence of significant polychromasia and anisocytosis may indicate the presence of a regenerative response. Blood smears should also be evaluated for blood parasites and telltale alterations in red blood cell morphology. Heinz bodies are characterized by bulging of the red blood cell membranes and indicate oxidative red blood cell damage secondary to toxins such as onions, garlic, or propylene glycol. Spherocytes are small, round erythrocytes with loss of central pallor that result when antibodies bound to red blood cell membranes lead to a portion of the membrane being phagocytized or “pinched off” by macrophages. Large numbers of these cells are typically seen in dogs with immune-mediated hemolysis. Schistocytes are erythrocytes that have become fragmented as a result of passage through narrowed microvasculature. Schistocytes typically reflect microangiopathic causes of hemolysis such as caval syndrome, disseminated intravascular coagulation, hemangiosarcoma, or splenic torsion. Acanthocytes are red blood cells with long spiny projections that are frequently seen in patients with hepatic or splenic neoplasia, though they may also be seen in animals with disorders of lipid metabolism as well.

Finally, a slide agglutination test should be performed when hemolysis is suspected. In this test, a drop of anticoagulated blood from a purple top tube or capillary tube is mixed with five drops of saline. Autoagglutination may be evidenced by the observation of obvious flecks within the drop of blood. The saline is used to disperse rouleaux that may mimic agglutination. Autoagglutination is caused by cross-linking of antibodies bound to the erythrocyte membranes, and as such is diagnostic for an immune-mediated component to the hemolysis.

Formulating a List of Differential Diagnoses
Once the anemia has been classified as blood loss, hemolysis, or decreased production, a list of differentials may be formulated. (see table 1)
Table 1. Some Common Differentials For Anemia

<table>
<thead>
<tr>
<th>Acute Blood Loss</th>
<th>Hemolysis</th>
<th>Decreased Production</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma</td>
<td>Immunologic</td>
<td>Selective Red Cell Hypoplasia</td>
</tr>
<tr>
<td>Hemoabdomen</td>
<td>Autoimmune (AIHA)</td>
<td>Chronic disease</td>
</tr>
<tr>
<td>Hemotherax</td>
<td>Secondary IMHA</td>
<td>Renal disease</td>
</tr>
<tr>
<td>Hemoretroperitoneum</td>
<td>Drugs</td>
<td>Red Cell Aplasia/PIMA</td>
</tr>
<tr>
<td>Fracture sites</td>
<td>Neoplasia</td>
<td>FeLV</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Blood Parasites</td>
<td>Immune</td>
</tr>
<tr>
<td>Splenic/liver</td>
<td>Vaccination</td>
<td>Endocrine</td>
</tr>
<tr>
<td>Pericardial</td>
<td>Infectious disease</td>
<td>Hypothyroid</td>
</tr>
<tr>
<td>Retropertitoneal</td>
<td>FelV</td>
<td>Addison's</td>
</tr>
<tr>
<td>Other</td>
<td>Neonatal Isoerythrolysis</td>
<td>Generalized Hypoplasia</td>
</tr>
<tr>
<td>Primary hemostatic defect</td>
<td>Incompatible Transfusion</td>
<td>Toxic</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Chloramphenicol</td>
<td></td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Non-Immunologic</td>
<td>Estrogen</td>
</tr>
<tr>
<td>Von Willebrands</td>
<td>Parasitic</td>
<td>Antifungals</td>
</tr>
<tr>
<td>Secondary hemostatic defect</td>
<td>Oxidative toxins (onions)</td>
<td>Chemotherapy</td>
</tr>
<tr>
<td>Rodenticide intoxication</td>
<td>Zinc toxicity</td>
<td>Infectious</td>
</tr>
<tr>
<td>Hemophilia</td>
<td>PK/PFK deficiency</td>
<td>Ehrlichia</td>
</tr>
<tr>
<td>DIC</td>
<td>Microangiopathic</td>
<td>FeLV/viral</td>
</tr>
<tr>
<td>Gastrointestinal losses</td>
<td>DIC</td>
<td>Myelophthis</td>
</tr>
<tr>
<td>Ulcerative diseases</td>
<td>Caival syndrome</td>
<td>Myelofibrosis</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Hemangiosarcoma</td>
<td>Hypercellular bone marrow</td>
</tr>
<tr>
<td>Hemostatic defects</td>
<td>Organ torsion</td>
<td>Iron deficiency</td>
</tr>
<tr>
<td>Surgical/iatrogenic losses</td>
<td>Hypoposphatemia</td>
<td>B12/folate deficiency</td>
</tr>
<tr>
<td></td>
<td>Thermal damage</td>
<td>Lead toxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myelodysplasia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myeloproliferative neoplasia</td>
</tr>
</tbody>
</table>

**Acute Blood Loss**

Diagnosing acute blood loss is simple when an external source of bleeding is present. However, cavity bleeding, gastrointestinal losses, coagulopathies, and chronic blood loss may be more challenging clinical entities. History and physical exam are usually the key elements in identifying a source of acute blood loss. Additional diagnostic testing generally includes minimum database (complete blood count, serum biochemistry panel, and urinalysis) and imaging studies such as radiographs and/or ultrasound to identify the source of bleeding. Platelet counts and coagulation testing should be performed any time hemostatic defects are suspected. Gastrointestinal blood loss should be suspected when external or cavity bleeding is not identified. Significant gastrointestinal blood loss may occur before signs of melena, hematemesis, or hematochezia are noted.

Treatment of acute blood loss consists of providing hemostasis, administering fluids for volume replacement, and considering blood transfusion if volume support alone is insufficient to provide for tissue oxygen delivery and clinical signs of anemia are present.

**Hemolysis**

In patients presenting with hemolysis, CBC, chemistry, and urinalysis should be run as part of a minimum database. The presence of hemoglobinemia/hemoglobinuria or bilirubinemia/bilirubinuria may suggest intravascular or extravascular hemolysis, respectively. Leukocytosis is frequently noted on the CBC from patients with IMHA and may result from non-specific “gearing up” of the bone marrow, or more likely, from tissue damage secondary to hypoxia and thrombosis. White blood cell counts in excess of 40,000/µl have been associated with a poorer prognosis in dogs with IMHA. Platelet counts should also be evaluated. Moderate thrombocytopenias may suggest consumptive coagulopathy or tick-borne illness, while severe
thrombocytopenias (<50,000/μl) should prompt consideration of a concurrent immune-mediated thrombocytopenia. A Coombs test is indicated if hemolysis is suspected but autoagglutination is not present. The Coombs test, or direct antiglobulin test, is essentially a test for the presence of antibodies or complement bound to erythrocyte membranes. It is performed by adding anti-dog antibodies (immunoglobulins directed against canine IgG, IgM, or complement) to a sample of the patient’s red blood cells. If autoantibodies are present on the patient’s blood cells, the antiseraum binds to them and cross-linking occurs. Because the end (positive) result of this test is agglutination, the Coombs test need not be run if the patient is already autoagglutinating. Note also that the Coombs test is not highly sensitive. Review of cases seen at our hospital (unpublished data) identified a sensitivity of only 66%, comparable to other reports in the veterinary literature.

A search should also be conducted for possible trigger factors. History taking should include questioning about recent vaccinations or medications. Recent vaccination (ie. within 4 weeks) has been associated with the development of IMHA in retrospective studies. Sulfur drugs, penicillins, and cephalosporins have also been associated with IMHA by acting as haptons, substances that become adsorbed to erythrocyte membranes and subsequently are able to stimulate an immune response. Neoplastic processes such as hemangiosarcoma, lymphoma, leukemia, and histiocytic sarcoma are another common trigger factor, and chest radiographs and abdominal ultrasound are frequently performed to rule out these entities. Testing should also be performed for vector-borne illnesses such as Ehrlichiosis, Babesiosis, Bartonellosis, and Hemoplasmosis. FeLV and FIV testing should not be overlooked in the cat.

Non-immunologic causes for hemolysis should also be considered. In addition to the oxidative toxins such as onions, ingestion of zinc may result in fulminant intravascular hemolysis, hemoglobinuria, multi-organ dysfunction, and DIC. Hereditary diseases such as phosphofructokinase deficiency seen in English Springer Spaniels may result in episodic hemolytic anemia, easily confused with IMHA because of its “apparent” response to steroids.

Reticulocyte count should always be performed to assess regenerative response. Hemolytic anemias are typically strongly regenerative, though it may take three days for regenerative response to be noted. The presence of a non-regenerative anemia (absolute reticulocyte count < 60,000/μl) should prompt suspicion of non-regenerative immune-mediated anemia (NRIMA), bone marrow disease, or other forms of decreased production anemia described below. Immunosuppressive therapies like prednisone ideally should not be initiated until neoplastic and non-immunologic causes of hemolysis have been ruled out, as the use of these drugs may interfere with accurate diagnosis and subsequent therapies. However, in cases where IMHA is strongly suspected and clinical signs are severe, prednisone is typically started pending labwork to avoid excessive delays in therapy.

**Decreased Production**

An anemia should be considered non-regenerative when the reticulocyte count is less than 60,000/μL (corresponding to a corrected reticulocyte count of less than 1%). However, it should be noted that a regenerative response usually becomes apparent after a minimum of 2-3 days, so acute blood loss or hemolysis may initially appear to be non-regenerative. Once a non-regenerative anemia is identified, bone marrow aspiration is generally indicated. Differentials for decreased production anemia may be grouped according to bone marrow histopathology. Some diseases cause a selective hypoplasia of red cell lines, while others affect all cell lines within the bone marrow (see table 1 above).

**Selective Erythroid Hypoplasia**

Anemia of chronic disease, also termed anemia of inflammation, is immune driven. Cytokines and cells of the reticuloendothelial system (RES) induce changes in iron homeostasis, erythrocyte lifespan, production of erythropoietin, and proliferation of erythroid lines. Iron is diverted from circulation to storage sites within the RES, limiting availability for erythroid progenitors.
Inflammatory mediators (TNF, IL-1, IFN) suppress activity of erythroid precursors and decrease their responsiveness to erythropoietin. Release of erythropoietin is also inhibited. Finally, erythrophagocytosis and free radical mediated erythrocyte damage shorten RBC survival. In contrast to iron deficiency anemia, anemia of chronic disease tends to be normocytic, normochromic, rather than microcytic, hypochromic. Serum iron tends to be low, but bone marrow iron stores are adequate. Anemia of chronic disease is generally mild to moderate unless complicated by other factors such as blood loss or hemolysis. Treatment is therefore directed at correcting the underlying disease. Transfusion may be considered if anemia is associated with clinical signs. Iron supplementation for anemia of chronic disease is controversial, and indications for its use in veterinary patients with chronic disease is unclear.

**Chronic renal failure** is typically associated with mild to moderate anemia. Because of the gradual and chronic nature of this type of anemia, it tends to be well compensated until very advanced stages. Anemia in renal failure is multifactorial and results from decreased erythropoietin production by the kidney, impaired responsiveness of bone marrow precursors, shortened RBC lifespan due to uremia, and GI blood loss resulting from uremic ulcers. Anemia of renal failure is typically well compensated, though transfusions may be indicated in the event of concurrent losses or surgery. Human recombinant erythropoietin has been used to stimulate RBC production in veterinary patients with renal failure, but is increasingly being used only as a "last ditch effort" as antibody production against epogen may lead to antibodies being directed against the patient’s own erythropoietin as well. Canine recombinant erythropoietin has not been associated with antibody production in dogs with renal failure, but is unfortunately not commercially available. Anabolic steroids (Winstrol-V) have been used in patients with renal failure based on the observations that they increase RBC mass in healthy animals. A benefit in these cases has not been clearly identified.

**Pure red cell aplasia (PRCA)** and **precursor-targeted immune mediated anemia (PIMA)** are immune mediated diseases directed against erythrocyte precursors. In our bone marrow database, PIMA accounted for 75% of all isolated non-regenerative anemia (ie. no other cell lines affected) seen at Michigan State University. The anemia is non-regenerative, normocytic-normochromic, with normal leukocyte and platelet counts. Animals tend to present with marked anemia, as the progression of the disease is typically slow and there is adequate time to mount a compensatory response. Diagnosis is made on the basis of bone marrow aspiration or biopsy, with few to no erythroid precursors seen in PRCA. In cases of PIMA, left shifted erythroid hyperplasia is frequently seen, with maturation arrest at the level of the metarubricytes or rubricytes. Some animals with PIMA will also have immune mediated destruction of mature erythrocytes, resulting in concurrent hemolysis. Cats with PRCA should always have PCR or IFA performed to rule out feline leukemia C associated attack on erythroid progenitors, as this form of PRCA is typically fatal. Treatment for immune-mediated PRCA and PIMA relies on immunosuppressive therapies similarly to IMHA. Periodic transfusions may be needed until regeneration occurs. This may take weeks to several months. Clinical signs and progression tend to be less severe than IMHA, as the anemia results from decreased production, rather than hemolysis.

**Endocrine diseases** such as hypothyroidism and hypoadrenocorticism may also result in a decreased production anemia. Both cortisol and thyroid hormone have a permissive role in the response of red blood cell precursors to erythropoietin. These forms of anemia are generally mild unless complicated by concurrent blood loss and resolve with hormone replacement therapy.

**Generalized Bone Marrow Hypoplasia**

**Generalized bone marrow hypoplasia** may result from radiation, toxic, or infectious insults to the bone marrow. Common toxins include estrogen, chloramphenicol, phenylbutazone, antifungals, and chemotherapeutic drugs. Infectious diseases resulting in bone marrow hypoplasia include *feline leukemia* and chronic *Ehrlichiosis*. Generalized bone marrow hypoplasia may also result from the crowding out of normal bone marrow precursors by neoplastic cells, a process termed **myelophthisis**. The most common neoplastic causes are the
hematopoietic and lymphoid neoplasms including lymphosarcoma, granulocytic leukemia, and lymphoid leukemias. **Myelofibrosis**, the replacement of marrow spaces by connective/scar tissue, usually represents the endpoint of previous severe marrow injury (as in the case of estrogen toxicity and ionizing radiation) or it may occur spontaneously. Peripheral blood features of myelofibrosis usually include severe nonregenerative anemia, severe leukopenia, and a variable platelet response. Confirmation of the diagnosis depends on marrow core biopsy with a demonstration of connective tissue filling the marrow space.

**Normal to Hypercellular Bone Marrow**

**Iron deficiency anemia** results from chronic blood loss. In young animals, parasitic infection is the primary ruleout for iron deficiency anemia, while in older animals, gastrointestinal masses or ulcers are generally implicated. Chronic blood loss leads to depletion of bone marrow iron stores over time, resulting in inability to form hemoglobin. Nuclear maturation of RBC precursors is normal however. Precursors continue to divide, getting smaller in size because they never acquire a complete amount of hemoglobin. This results in a hypercellular bone marrow with a build up of metarubricytes. Diagnosis is based on the presence of microcytic, hypochromic anemia, thrombocytosis, source of blood loss, and a bone marrow smear containing no stainable iron. Low serum iron is not diagnostic as it may rapidly decrease with inflammatory disease as a result of tissue sequestration. Treatment is aimed at removal of the source of blood loss. Ferrous sulfate may be administered at a dose of 100-300 mg per day in dogs and 50-100 mg per day in cats if needed. Note that this dose refers to ferrous sulfate, not elemental iron. Reticulocytosis should develop within 3-4 days of supplementation.

**Myelodysplasia** refers to a poorly understood group of diseases characterized by non-regenerative anemia or pancytopenia and prominent dysplastic changes in the bone marrow. Abnormal erythrocytes are generally unable to completely differentiate and early cell death results. Myelodysplasia may result from idiopathic (primary), neoplastic, toxic, immune-mediated, or infectious (FeLV) causes. The myelodysplasias tend to carry a very guarded prognosis, with treatment aimed at immunosuppression, chemotherapy, and/or erythropoietin depending on the suspected cause.

**General Comments on the Treatment of Non-Regenerative Anemias**

Treatment of decreased production anemia is best aimed at identifying and eliminating any underlying disease processes or myelosuppressive drugs. Once this is done, clinical experience suggests that the most important thing we can do for patient is to buy time for the bone marrow to repopulate with normal precursor cells. Blood transfusions should be provided as needed until the patient is able to mount a regenerative response of their own. Most patients with non-regenerative anemia require transfusions every 4-6 weeks until their disease is well controlled. Broad-spectrum antibiotics are indicated in the event of severe neutropenia to prevent secondary infections. Immunosuppressive agents may be indicated if an immune-mediated disease (eg. red cell aplasia) is identified or strongly suspected. Myeloproliferative diseases and myelodysplasia tend to carry a poor prognosis, but other forms of decreased production anemia may respond well if the underlying disease or insult is eliminated and adequate time is provided for recovery.

**Conclusion**

A variety of diseases, both immunologic and non-immunologic in nature, may result in anemia and/or hemolysis in veterinary patients. Successful management relies upon accurate diagnosis and treatment of the underlying disease process. Simple test to help classify a patient’s anemia as blood loss, hemolysis, or decreased production may facilitate correct diagnosis. Initiation of immunosuppressive therapy prior to performing a methodical search for infectious, neoplastic, or other causes of anemia may result in therapeutic “missteps” and treatment failure.

**REFERENCES**

Cardiopulmonary Cerebral Resuscitation: 
Current Guidelines and their Application
L. Ari Jutkowitz, VMD, Diplomate ACVECC
Michigan State University

Introduction
Cardiopulmonary cerebral resuscitation (CPCR) refers to the re-establishment of circulation and preservation of neurologic function following an arrest. Since its inception in the late 1800’s, CPCR has saved the lives of countless human and veterinary patients. However, low overall survival rates following CPCR indicate that there is still much room for improvement in these practices. This session reviews current practices and updates on CPCR in the veterinary patient with an emphasis on evidence-based guidelines derived from the RECOVER initiative.

Basic Life Support
Basic life support refers to the process of establishing an airway, initiating positive pressure ventilation, and performing chest compressions. Because cardiopulmonary arrest (CPA) in veterinary patients is frequently initiated by respiratory arrest, an ABC approach is generally taken as described below. In recent years, there has been a paradigm shift prioritizing chest compressions above all other measures (CAB approach).

Circulation
Chest compressions are initiated at a rate of 100-120 per minute, compressing the circumference of the chest by approximately 30-50%. The patient should be in lateral recumbency during compressions. In smaller dogs, where the cardiac pump theory is believed to predominate, hands should be placed over the ventral third of the chest just behind the point of the elbow, corresponding to a position directly over the heart. In larger dogs, the thoracic pump theory is believed to be most important in generating blood flow, and hands should therefore be placed over the widest part of the thorax to create a maximal rise in intrathoracic pressure.

Airway
Orotracheal intubation is easily achieved in dogs, as the larynx can be directly visualized by retracting the tongue. The head and neck should be gently extended and a laryngoscope may be used to improve visualization of the larynx. In cases where hemorrhage, saliva, or gastric contents interfere with visualization, suction may be helpful. Alternately, the glottis may be palpated with one finger used to guide tube placement. Once tube placement is verified, the tube should be secured by tying to the nose or around the back of the head. The cuff should be inflated, and assisted ventilation provided. If chest wall excursion is not seen, lung sounds are absent, or abdominal distension is noted, tube placement should be reconfirmed by direct visualization and the cuff should be reinflated. Improper tube placement and tube dislodgement are common causes of CPCR failure.

Breathing
Once an endotracheal tube is in place, breathing is initiated at a rate of 10 breaths per minute with 100% oxygen to a tidal volume of approximately 10 ml/kg. An ambu bag with attached oxygen line is ideal for this purpose. If only one person is available to perform CPR, 2 breaths should be given for each 30 chest compressions. If several trained personnel are available, then breaths may be delivered independent of compressions. Chest wall excursion should be seen with each delivered breath. Airway pressures ideally should not exceed 20-30 cm H₂O. High airway pressures or inadequate chest wall excursion should prompt a search for pleural space disease, tube malposition, or tube occlusion.

A number of alternative techniques have been investigated that may help to augment blood flow during CPCR. Those that are directly applicable in veterinary patients include circumferential chest compression and interposed abdominal compressions. Circumferential chest compression is most commonly performed in cats and small dogs by encircling the chest with both hands to maximize the rise in intrathoracic pressure during chest compression. In larger animals, interposed abdominal compression may be implemented by having an additional person perform abdominal compressions during the relaxation phase between chest compressions. Interposed abdominal compressions increase venous
return to the heart, leading to greater stroke volumes and cardiac output, and have been associated with increased survival to discharge in human patients.

**Advanced Life Support**

Advanced life support consists of drug administration, determination of cardiac electrical activity, and application of electrical defibrillation if indicated. These techniques build upon basic life support to increase the likelihood of successful resuscitation.

**Drugs**

Establishing vascular access is one of the first priorities during advanced life support. While central lines are preferable for rapid distribution of drugs, peripheral catheters are acceptable, and drug delivery may be facilitated by following drug administration with a 10-20 ml IV fluid “chaser”. If vascular access is not immediately obtained, surgical cutdown or intraosseous techniques should be considered. The intratracheal route may also be used initially to deliver drugs. Epinephrine, atropine, vasopressin, lidocaine, and naloxone may all be given in this way by administering twice the normal dose of the drug (or using the “high” dose for epinephrine) and administering several large breaths to disperse the drug.

Drugs administered during CPCR include intravenous fluids, narcotic reversal agents, vasopressors, vagolytics, antiarrhythmics, and potentially sodium bicarbonate. Shock doses of intravenous fluids should be provided in cases where hypovolemia is believed to have played a role in the arrest. Moderate fluid rates should be used in euvolemic patients or patients with underlying heart disease, as rapid administration in these cases may excessively elevate right atrial pressure and consequently decrease myocardial and cerebral perfusion pressure.

Patients who have received narcotic pain relievers or other sedative/anesthetic drugs prior to arrest should immediately be given the reversal agent for that drug. Naloxone may be used to reverse most narcotics at a dose that is isovolumetric to the dose of the original narcotic, or at 0.02-0.04 mg/kg IV if the original dose is unknown. Flumazenil (0.02 mg/kg IV) may be used to reverse benzodiazepines, and yohimbine (0.1 mg/kg) or atipamazole (0.2 mg/kg or isovolumetric) may be used to reverse xylazine and medetomidine respectively. Any anesthetic gases, if still in use, should be discontinued and the anesthetic circuit flushed with fresh oxygen.

Vasopressors are commonly used during CPCR to increase blood pressure and redistribute blood flow to vital organs like the brain and heart. Epinephrine continues to be the vasopressor of choice during CPCR in veterinary patients, though its use is largely extrapolated from clinical studies in human patients. Both low dose and high dose epinephrine protocols are described in human medicine. While high dose epinephrine has been associated with increases in early return of spontaneous circulation, no long-term benefits have been identified. High dose epinephrine has additionally been associated with increased myocardial oxygen demand and worse neurologic outcomes. For these reasons, it is recommended that low dose epinephrine initially be administered every 3-5 minutes during CPCR, switching to the high dose only if there is a lack of response to the lower doses. Epinephrine dosing may be rapidly calculated according to the following rule of thumb: 0.1 ml per 20 lb of the 1:1,000 formulation for low dose, or 1 ml per 20 lb for high dose.

Vasopressin is another potent vasoconstrictor that is increasingly used in resuscitation of human patients. Unlike epinephrine, it does not increase myocardial workload, and its effect is not blunted by acidosis. Although clinical data in veterinary patients is currently lacking, animal models and human clinical trials suggest that vasopressin may be as effective as epinephrine. Vasopressin (0.8 units/kg IV) may therefore be considered as an alternative to epinephrine in dogs.

Atropine is another drug frequently administered during CPCR to reverse parasympathetic contribution to the arrest or to treat sinus bradycardia. Atropine is administered at a dose of approximately 1 ml per 20 lb (0.04 mg/kg) for asystole or pulseless electrical activity. When treating sinus bradycardia, only half this dose is needed.
Sodium bicarbonate use in CPCR is controversial, as it has been associated with numerous adverse effects including hypernatremia, paradoxical CNS acidosis, and decreased resuscitation rates in people. However, its use should still be considered during long duration (>10 minutes) arrests, as control of acidosis may improve response to catecholamines as well as post-arrest neurologic outcomes. Bicarbonate is typically given only after 10 minutes of CPCR at a dose of 1 mEq/kg and is repeated every 5 minutes thereafter.

Electrical Activity
ECG leads should be attached as soon as feasible to assess electrical activity. Connecting the leads to the skin of the lower forelimbs and hindlimbs will help to minimize motion artifact associated with resuscitation efforts. Four rhythms are commonly seen during cardiopulmonary arrest in dogs. Asystole and pulseless electrical activity are the initial arrest rhythms most commonly seen in dogs, followed by ventricular fibrillation and sinus bradycardia. Accurate ECG diagnosis is vital to a successful code. The presence of sinus bradycardia or suspicion of a vagal arrest should prompt administration of atropine. Asystole should be confirmed in more than one lead, to rule out the possibility of artifact related to poor contact. While some dogs in asystole will convert directly to sinus rhythm following resuscitation, many develop ventricular fibrillation and require electrical shock for conversion. Once ventricular fibrillation is identified, electrical defibrillation should immediately be administered, temporarily bypassing all other resuscitation measures. The greater the time that a dog spends in fibrillation, the lower the likelihood of successful conversion.

Defibrillation
Early application of electrical shock is the only effective method for converting VF to sinus rhythm. VF is a form of disorganized electrical activity with various portions of the heart muscle firing at different times. Electrical shock essentially "resets" the cardiac cells so that organized activity can resume. Practically speaking, applied current must pass through at least 30% of cardiac myocytes to effectively convert VF.

To accomplish defibrillation, the dog is flipped into dorsal recumbency immediately preceding defibrillation and handheld paddles are placed on either side of the chest directly over the heart. Ample conducting gel should be applied to the paddles to ensure good contact and prevent dispersion of current. The chest should be compressed between the paddles, minimizing impedance by narrowing the distance between paddles. If using a monophasic defibrillator, the energy for the first shock should be set at 3-5 J/kg. If defibrillation is not successful, CPCR is resumed for 60-90 seconds and a subsequent shock should then be given at the same energy setting. Electrical shock is discontinued once the rhythm converts from VF. Lower energy biphasic shock waveforms have been shown to be as effective as higher energy monophasic waveforms and exclusively used at this time in human patients. If using a biphasic defibrillator, the pediatric settings should be used (2-4 J/kg).

For shock-refractory VF, a search should be undertaken to identify problems such as improper paddle position, inadequate contact, insufficient conduction gel, or the presence of pleural space disease that may increase impedance. Drug-shock techniques may then be considered, administering epinephrine or amiodarone (5 mg/kg IV) prior to shock to lower defibrillation threshold. Lidocaine was previously used for this purpose as well, but has been reclassified as a therapy of indeterminate benefit in the most recent ACLS guidelines.

Open Chest CPCR
There are a number of absolute indications for open chest CPCR. These include cardiac arrest caused by or associated with pleural space disease (pneumothorax, pleural effusion, diaphragmatic hernia), pericardial effusion, or penetrating injury resulted in cardiac arrest. However, debate exists in veterinary medicine as to other indications for performing open chest CPCR. Some advocate open chest CPCR immediately in large breed dogs because of the limited success of restoring adequate circulation with external compressions while others prefer to perform external CPCR for 5 minutes and then open the chest if there is little or no evidence of effective circulation. Open chest CPCR has the advantage of allowing the clinician to directly compress the heart and improve stroke volume. In addition, opening the chest makes assessment of ventricular filling feasible aiding in the decision of volume delivery.
When opening the chest, it is critical to auscult the chest just prior to the incision to rule out ECG dysfunction as the cause of asystole. The left chest should be crudely clipped of hair at the left 5th-6th intercostals space and a chlorhexidine based antiseptic solution should be briskly applied. An incision should be made through the skin and subcutaneous tissues from just below the spinal musculature to the level of the costochondral junction. Between positive pressure breaths, mayo scissors should be used to poke through the intercostal musculature and the pleura and the chest is opened by sliding the mayo scissors dorsally and ventrally along the cranial border of the rib (to avoid the neurovascular bundle). The pericardium is opened at the pericardio-diaphragmatic ligament and the heart is compressed from the apex to the base. In large dogs, the heart can be compressed against the opposite chest wall.

In the event of return of spontaneous circulation, antibiotics should be instituted immediately, the chest should be lavaged with copious amounts of warm saline, and should be closed using sterile technique over a chest tube.

**ICU Care**

Following a successful code, a search for underlying causes or complications should be performed and any problems corrected. Blood gases, hematocrit and total solids, blood pressure, and oxygen saturation are carefully monitored and optimized during this time. This tends to be the most challenging phase of arrest management, as complications and recurrence of CPA are common. Neurologic recovery is promoted by maintaining arterial blood pressure and oxygen saturation. Because elevation in carbon dioxide levels leads to cerebral vasodilation and consequently increased intracranial pressure, hypercarbia should be prevented by employing mechanical ventilation if needed. Once cardiovascularly stable, mannitol (0.25-0.5 g/kg IV over 20 minutes) may also be indicated to treat cerebral edema and resultant elevations in intracranial pressure. Corticosteroids are associated with potentially deleterious hyperglycemia in post-arrest patients, and current protocols do not support their use.7

**Prognosis**

Recurrence of CPA in the post-arrest period is common, occurring in up to 70% of successfully resuscitated dogs. Intensive care and monitoring during this time is therefore essential. Survival to discharge following cardiopulmonary arrest has been reported in 4-11% of cases.5,6,8 Transient blindness, seizures, circling, ataxia, and decreased level of consciousness are common for some period of time following CPA, but the majority of survivors have a good prognosis for functional recovery.6

References


Table 1. Drugs Commonly Used in CPR
<table>
<thead>
<tr>
<th>Drug (conc)</th>
<th>Weight lb</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Weight kg</td>
<td></td>
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<tr>
<td></td>
<td>Dose</td>
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<td></td>
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<tr>
<td>Amiodarone (50 mg/ml)</td>
<td>5 mg/kg</td>
<td>0.5</td>
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<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
<td>3.5</td>
<td>4</td>
<td>4.5</td>
<td>5</td>
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<tr>
<td>Atropine (0.54 mg/ml)</td>
<td>0.04 mg/kg</td>
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<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
<td>3.5</td>
<td>4</td>
<td>4.5</td>
<td>5</td>
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<tr>
<td>Bicarb (1 mEq/ml)</td>
<td>1 mEq/kg</td>
<td>5</td>
<td>10</td>
<td>15</td>
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<tr>
<td>Epi low (1:1,000)</td>
<td>0.01 mg/kg</td>
<td>0.0</td>
<td>0.1</td>
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<td>0.2</td>
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<td>0.4</td>
<td>0.45</td>
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</tr>
<tr>
<td>Epi high (1:1,000)</td>
<td>0.1 mg/kg</td>
<td>0.5</td>
<td>1</td>
<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
<td>3.5</td>
<td>4</td>
<td>4.5</td>
<td>5</td>
</tr>
<tr>
<td>Naloxone ((0.4 mg/ml)</td>
<td>0.04 mg/kg</td>
<td>0.5</td>
<td>1</td>
<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
<td>3.5</td>
<td>4</td>
<td>4.5</td>
<td>5</td>
</tr>
<tr>
<td>Vasopressin (20 u/ml)</td>
<td>0.8 u/kg</td>
<td>0.2</td>
<td>0.4</td>
<td>0.6</td>
<td>0.8</td>
<td>1</td>
<td>1.2</td>
<td>1.4</td>
<td>1.6</td>
<td>1.8</td>
<td>2</td>
</tr>
<tr>
<td>External Defibrillation</td>
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<td>150</td>
<td>200</td>
<td>200</td>
<td>200</td>
<td>300</td>
<td>300</td>
<td>300</td>
<td>360</td>
</tr>
</tbody>
</table>

Note: Atropine, epinephrine, lidocaine, amiodarone, and naloxone may all be approximated using the rule of thumb 1 ml/20 lb.
Reproductive problems often arise after normal business hours, so it is not uncommon for them to fall into the domain of the emergency veterinarian. As most owners lack medical knowledge, they frequently look to the veterinarian to answer questions and to identify potential problems. The emergency clinician must therefore be familiar with normal reproductive behavior in addition to the common emergencies that may arise. With this goal, we will review the events surrounding normal parturition as well as the common complications that may develop during this period.

Normal Reproductive Physiology
Normal gestation length in the dog may range from 57-72 days from the time of first breeding, with an average length of 65 days.\(^1\)\(^2\) Because cats are induced ovulators, there is generally less variability in gestation length, which ranges from 63-65 days. Ovulation may not take place after the first breeding however, so in the event of multiple breedings, uncertainties with regards to gestation length may still be present in the cat. As the whelping date approaches, a number of clues may point toward impending parturition. Mammary development, vulvar enlargement, mucous vaginal discharge, and relaxation of the pelvic ligaments are early signs of approaching parturition. Onset of lactation may be noted in primiparous bitches within 24 hours of parturition, but in multiparous bitches may occur several days before parturition. A sudden drop in body temperature (>2°F) is generally noted within 24 hours of parturition\(^3\) in dogs and cats as a result of decreases in progesterone levels, but this finding is not always reliable. In one study, nadir temperature occurred >48 hours before parturition in 24% of dogs, and an appreciable drop in temperature (>1°F) was not seen in 35% of dogs.\(^4\)

Normal parturition proceeds in three stages. The first stage is characterized by subclinical uterine contractions and progressive dilation of the cervix. During this stage, which typically lasts for 6-12 hours, bitches may show signs of restlessness, apprehension, panting, nesting behaviors, hiding, and anorexia. Queens may be tachypneic, restless, and vocal, or may lay in their nesting boxes, purring. Active expulsion of the fetuses occurs during the second stage of labor. The first fetus is usually delivered within 1 hour of onset of stage 2 labor in cats, and within 4 hours in dogs, with subsequent deliveries every 15 minutes to 3 hours.\(^5\)\(^6\) Active straining generally results in expulsion of a fetus within 15 minutes. The entire process generally occurs over 2-12 hours, but may take as long as 24 hours with large litter sizes. The third stage of labor results in expulsion of the placenta. One placenta should be identified for each fetus delivered. Placentas are usually still attached to the fetus by the umbilical cord and emerge with the fetus, but may emerge within 15 minutes to several hours if they become detached. Lochia, a greenish vaginal discharge, indicates placental separation and may be seen during all stages of labor. Following parturition, the discharge gradually becomes red-brown, decreasing in volume over 4-6 weeks as uterine involution takes place.

Dystocia
Historical and physical exam findings that should prompt a clinician to suspect dystocia are as follows:\(^1\)

- A definite cause is apparent (ie. fetus lodged in birth canal, pelvic fractures)
- Gestation is prolonged (>70 days) with no evidence of labor
- Temperature has dropped to <100°F and returned to normal with no evidence of labor within 24 hours
- Lochia is noted and 2 hours have elapsed without expulsion of a fetus
- Strong and persistent contractions fail to result in the delivery of a puppy within 30 minutes
- Weak and infrequent contractions fail to produce a fetus within 4 hours.
- More than 4 hours have elapsed since the birth of a puppy with no evidence of ongoing labor
- Signs of systemic illness or severe pain are present
Dystocia may result from either maternal or fetal factors that prevent delivery from taking place. Uterine inertia is the most common maternal cause of dystocia, seen when the myometrium produces only weak and infrequent contractions that fail to expel a normal fetus through a normal birth canal. Primary uterine inertia is considered complete when gestation has exceeded its expected length with no evidence of progression into active labor. Primary uterine inertia is termed partial if the bitch initiates parturition and expels one or more healthy fetuses, but then subsequently fails to deliver the remaining fetuses as a result of myometrial fatigue. Uterine inertia may also be considered secondary if myometrial failure results from prolonged attempts to expel an obstructed fetus, and persists following relief of obstruction. Morphologic causes of dystocia are those in which an anatomic abnormality of the bitch or queen results in obstruction of the birth canal (eg. small birth canal, pelvic fractures).

Fetal factors that may result in dystocia include malpresentations, oversize, fetal malformations, and fetal death. Some of the commonly described malpresentations include transverse presentation, lateral or ventral flexion of the neck, anterior presentation with flexion of one or both forelimbs, posterior presentation with retention of both hindlimbs, and simultaneous presentation of two fetuses. It should be noted that posterior presentations are considered to be a normal variation in dogs and cats, occurring in approximately 40% of deliveries. Fetal oversize is another potential cause of dystocia, most commonly seen with single pup pregnancies. Fetal death is an infrequent cause of dystocia, increasing the likelihood of malpresentation because of failure to rotate and extend the head and legs, which commonly occurs immediately prior to parturition. Fetal malformations are another potential cause of dystocia, with anasarca (generalized subcutaneous edema), hydrocephalus, cerebral and cerebrospinal hernias, abdominal hernias, duplications, and rib cage malformations among the more commonly noted.

**Diagnosis of Dystocia**

Workup of a patient that is presented for dystocia begins with a complete history and physical exam, including digital vaginal exam. If a fetus is lodged within the birth canal, digital manipulation should be attempted. The fetus may be grasped around the head and neck, around the pelvis, or around the proximal portions of the hind limbs, depending on fetal presentation. Excessive traction should never be applied to a single extremity because of the ease with which these may be avulsed. With the dam restrained in a standing position, traction is applied in a posterior-ventral direction. The fetus may be gently rocked back and forth, and twisted diagonally to free shoulders and hips “locked” in the pelvic canal. If flexion of head or extremities is preventing delivery, a finger may be used to extend them. One cannot overemphasize the importance of using copious amounts of sterile lubricant during obstetrical maneuvers, applied digitally or infused around the fetus using a red rubber catheter.

Radiographs should be obtained in any animal experiencing dystocia. Radiographs are accurate for assessing the number, size, location, and position of fetuses, as well as maternal pelvic morphology and general status of the abdomen. Fetal viability is more difficult to assess from radiographs, unless evidence of fetal decomposition is present. Signs of decomposition include intrafetal or intrauterine gas patterns, awkward fetal postures, collapse of the spinal column due to loss of muscular support, and overlapping of the bones of the skull. Ultrasound may be a more useful tool for assessment of fetal viability, fetal malformations, and fetal distress. Normal fetal heart rates have been reported at 180-245 beats per minute in dogs and up to approximately 265 bpm in cats. Deceleration of fetal heart rates to less than 180 beats per minute and the presence of fetal bowel movements on ultrasound have been shown to correlate with severe fetal distress, and may indicate a need for rapid intervention.

Medical management should be considered if there is no evidence of obstruction, and fetal and pelvic size appear normal. Oxytocin is a peptide hormone that increases the frequency and strength of uterine contractions by promoting influx of calcium into myometrial cells. Oxytocin also promotes post partum uterine involution, aids in control of uterine hemorrhage, and assists in expulsion of retained placentas. The dose for oxytocin has traditionally been reported at 5-20 units IM in the dog and 2-4 units IM in the cat. However, with an increase in the use of uterine contraction monitoring (Whelpwise, Veterinary Perinatal Specialties Inc, Wheat Ridge, CO) in veterinary patients, there is a growing body of evidence to suggest that traditional doses may be too high, potentially causing uterine tetany, ineffective contractions, and decreased fetal blood flow. Recent data suggests that doses of 0.5-2 units are effective in increasing the frequency and quality of contraction. The oxytocin dose may be repeated in 30 minutes if expulsion continues.
of a fetus has not resulted. If labor proceeds and a fetus is delivered, oxytocin may be repeated every 30 minutes as needed to assist in expulsion of the remaining fetuses.

Calcium gluconate may be considered if weak, infrequent contractions are noted4,12 or when labwork reveals hypocalcemia. Retrospective studies have indicated that many patients who fail to respond to oxytocin alone may respond to a combination of calcium and oxytocin.3,8 The dose for calcium gluconate (10% solution) as a uterotonic agent is 11 mg/kg diluted in saline and given subcutaneously, or added to IV fluids and given slowly while monitoring an ECG for arrhythmias. If hypocalcemia is documented, a dose of 50-150 mg/kg intravenously should be used. Subcutaneous administration has been reported to result in irritation and potential granuloma formation, though this is an infrequent complication. Dextrose infusion should also be initiated if hypoglycemia is evident on labwork.

Surgical management should be considered for the following conditions:1

- Complete primary uterine inertia
- Partial primary uterine inertia or secondary uterine inertia where large numbers of fetuses remain and response to drugs is unsatisfactory,
- Fetal oversize
- Gross abnormalities of maternal pelvis (fractures, masses)
- Fetal malformations
- Malpresentation that is not amenable to manipulation
- Past history of dystocia or c-section
- Fetal putrefaction
- Maternal evidence of systemic illness
- Suspicion of uterine torsion, rupture, prolapse, or herniation
- Evidence of fetal distress with poor response to medical intervention

An anesthetic protocol for caesarian section should be selected with the goal of maximizing survival of neonates and dam. Attempts should be made to minimize exposure of the fetus to anesthetics by keeping the time from induction to delivery as short as possible. Ideally, the dam should be clipped and prepped prior to induction, equipment should be out, and the surgeon should be scrubbed and ready. Maropitant (1 mg/Kg SQ) may be given 30 min prior to induction to decrease risk of regurgitation and aspiration. Induction agents should be given to effect. Regional techniques such as line blocks and epidurals may help to minimize the need for other drugs. A line block can be performed using 2 mg/kg lidocaine infused along the ventral midline. Alternately, epidural lidocaine may be administered in dogs at a dose of 2-3 mg/kg, not to exceed a total volume of 6 ml. Propofol (4-6 mg/kg IV) or alfaxalone (1-2 mg/kg) followed by isoflurane or sevoflurane are most commonly used for caesarian section at this time, and have been associated with reduced neonatal mortality in dogs. Anesthetic agents that have been associated with increased neonatal mortality include thiopental, ketamine, xylazine, medetomidine (controversial), and methoxyflurane.13-15 A recent study suggested that the inclusion of lidocaine epidural in the anesthetic protocol (propofol, sevoflurane) resulted in significant improvement in neonatal viability as a result of decreased anesthetic requirements.16

**Neonatal Resuscitation**

A warm (90°F) incubator, hemostats, suture material, suction bulb syringes, emergency drugs, and an adequate supply of soft dry towels should be prepared beforehand. As each neonate is handed off, the umbilical cord should be clamped and ligated 1-2 cm from the umbilicus. Fetal fluids and amnion should be removed by rubbing briskly with a soft, clean towel. The oral cavity and nares may be suctioned with a bulb syringe. The old practice of “swinging” puppies to clear their airways is best avoided because of the potential for cerebral hemorrhage due to concussive injury. If vigorous rubbing is not successful at stimulating respiration, positive pressure ventilation may be initiated with a snug fitting mask, keeping the neonates head and neck extended to ensure adequate inflation of the lungs. Alternately, intubation may be accomplished using a catheter or small, uncuffed endotracheal tube. Because isoflurane is minimally metabolized, ventilation is the primary route of elimination. Thus, its depressant effects can not be reversed until the neonate breathes. Cardiac massage may be instituted if a heart beat is not detected once warming and ventilation measures have been instituted. Epinephrine (0.1 mg/kg) may be given
intratracheally, intrasosseously, or intravenously if cardiac massage is unsuccessful. Naloxone (0.1 mg/kg) should be considered if the dam received opioid analgesics as part of the anesthetic regimen. Although doxapram (dopram) is routinely administered in many practices as a respiratory stimulant, it is not used for this purpose in the resuscitation of human neonates and there is no evidence to support its use in veterinary patients.

The prognosis for medical management of dystocia is guarded, with success rates of 20-40% in the veterinary literature. Additionally, stillbirth rates have been shown to rise when dystocia is allowed to continue for greater than 4.5-6 hours from the time of onset of second stage labor in the dog. For these reasons, the decision to proceed to cesarian section should not be delayed if response to medical management is poor or unlikely to result in successful delivery. In recent studies, neonatal survival rates following surgical treatment of dystocia have been reported at 92% at birth, with 80% still alive at 7 days post c-section.

Fluid therapy: Do I really need anything other than what I’m already using?
L. Ari Jutkowitz, VMD, Diplomate ACVECC
East Lansing, MI

While there are numerous options when it comes to fluid therapy, the unglamorous truth is that a majority of situations encountered in clinical practice can be managed with just a replacement solution like Lactated Ringer’s solution. The remaining few clinical scenarios can usually be accounted for with the addition of 0.9% NaCl and a maintenance type fluid to the arsenal. Still, every now and again, a niche fluid type can come in handy. In this session, we’ll discuss the evidence surrounding the various fluid alternatives and specific situations where it may be worth deviating from your default fluid plan.

The choice of fluid is based on three factors: a) knowledge of the disease process (e.g., blood loss, Addison’s, “blocked” cat), b) laboratory data (e.g., hypokalemia, metabolic alkalosis, hypoproteinemia), and c) purpose of fluids (i.e., replacement or maintenance).

Replacement vs Maintenance fluids: replacement fluid therapy is designed to replace existing fluid deficits; this usually requires replacement of both water and electrolytes. Consequently, replacement fluids tend to have sodium concentrations similar to plasma. Maintenance fluid therapy is designed to meet the patient's daily fluid and electrolyte needs, assuming a normal extra cellular fluid volume (ECF) and no excessive ongoing losses. Because normal animals tend to lose primarily free water, with a lesser degree of electrolyte loss, these fluids tend to be hypotonic, lower in sodium and chloride, and higher in potassium than plasma. Maintenance fluids are not used when high fluid rates or rapid infusion (ie bolusing) are needed. Because many hospitalized patients are being treated for ongoing losses whether they be through vomiting, diarrhea, blood loss, or third spacing, maintenance fluids are used in the hospital setting far less commonly than replacement fluids.

Replacement Fluids
Lactated Ringer’s Solution (LRS) is a replacement (R) fluid that is balanced (electrolyte concentration similar to serum) and isotonic (osmolality similar to serum) solution. Na = 131, K = 4, Ca = 3, Cl = 110, Lactate= 28 mEq/L. Lactate is metabolized by the liver to CO₂ and H₂O, and in the process yields HCO₃. (Na C₃H₅O₃ + 3 O₂ → 2CO₂ + 2H₂O + Na HCO₃. LRS is an excellent ECF replacement fluid, and also of use in metabolic acidosis. It is the most commonly used fluid for a multitude of disease processes in all species (with the possible exception of the cow where metabolic alkalosis is so common). There are other solutions that are similar to LRS (Replacement (R) solutions):

Normosol R and Plasmalyte 148 (or A). These solutions are also balanced and isotonic. The Na, K' and, Cl concentrations are similar. Mg is present. The HCO₃ precursor may be Acetate or Gluconate for example, Normosol R has 27 mEq/L of acetate and 23 mEq/L of gluconate. Acetate and gluconate are metabolized to HCO₃ similarly to Lactate. These solutions are interchangeable with LRS. Since acetate is metabolized by more tissues than the liver, it is a better bicarbonate precursor and thus has theoretical, though little practical, advantage.

Replacement solutions (LRS, Norm-R, Plyte-A, 0.9% saline) may be combined with 25-50 grams of Dextrose (D5) per 1000 ml of solution to form a 2.5-5% solution. This is commonly done to prevent or treat hypoglycemia in susceptible patients. Hypoglycemia may occur in the young animal or the animal with liver disease or sepsis. The only real problem with these (dextrose supplemented) solutions is that they are hypertonic. The possibility of phlebitis is increased. If dextrose concentration exceeds 5%, the solution should be given through a central vein.

Normal Saline (0.9% NaCl) is a replacement fluid that is unbalanced and isotonic. Na = 154, Cl= 154. There is an excess of Cl. When Cl is high, HCO₃ tends to be low. (Remember the anion gap equation AG= (Na + K) – (Cl + HCO₃). This fluid therefore tends to produce a mild acidosis. There has been much discussion in the human literature recently about the effects that this may have, including altered acid-base status, decreased GI mucosal blood flow, increased risk of kidney injury, and longer hospital stays.
Normal saline is used for metabolic alkalosis, as a replacement fluid when hyperkalemia or hypercalcemia is present, and for patients with a deficit in total body sodium (eg, the Addisonian or diabetic ketoacidotic patient).

**Hypertonic saline (7.2% NaCl)** solution is unbalanced and hypertonic (8X normal saline). It works elevating the [Na⁺]. The elevated [Na⁺] draws water out of the cell to rapidly expand the extra cellular fluid compartment (ECF). In essence, hypertonic saline works by expanding the ECF at the expense of the ICF. Its main advantage is that the patient can be resuscitated with less volume, quicker. This is useful in shock states where vascular access is limited, or where excessive shifting of fluids into the interstitial and intracellular spaces could be deleterious (eg head trauma). This may also be of importance in giant breed dogs (the 200 lb Saint Bernard whose shock dose of fluids would be about 9 liters). You must follow with a fluid “chaser” to allow ICF to be replenished

**Maintenance fluids**
The purpose of a maintenance fluid (M) is to deliver the patient’s normal daily requirements of water and electrolytes. The approximate water requirement is 45-60 ml/kg/day; the Na and K requirement is 1-2 mEq/kg/day, with Nab eeing toward the higher and K the lower end of the range. As an example, a 12 kg. dog requires per day approximately: 750 calories, 625 ml H₂O, 20 mEq Na and 17 mEq K "Straight" LRS will contain, per 625cc, 82 mEq Na and 2.5 mEq K (too much Na and not enough K). Many times, “maintenance solutions are made by simply adding K⁺ to replacement solutions”, though these solutions will contain more Na than is needed.

Dextrose 5% (D5W) is made by placing 50 gm of dextrose in one liter of distilled water. It is unbalanced and isotonic. It contains no electrolytes and 0.17 kcal/ml. D5W is therefore used most commonly as a diluent for certain medications, or to address severe hypernatremia.

**D2.5 in 0.45% NaCl** is half-strength dextrose in half-strength saline. It is unbalanced and isotonic. Na⁺ = 77, Cl⁻ = 77 mEq/L, dextrose = 25 gm/L. It is a good maintenance solution, after the addition of K⁺ (see below). It may be used similarly to D5W.

**Plasmalyte-56 and 5% Dextrose** is a commercial maintenance fluid. It contains, in mEq per liter, Na 40, K 16, Ca 5, Mg 3, Cl 40, Acetate 16, and Glucose 50 gm, and has an osmolality of 362. **Normosol M** is another common maintenance solution.

**Colloids:**
**Hetastarch** is a branched polymer of glucose that is soluble in plasma. It has an average molecular weight of 450,000 (compared to 69,000 daltons for albumin) and a colloid onotic pressure of 30 mmHg. It is available as a 6% solution in 0.9% saline. It adds onotic pressure to the serum, and is therefore be used in hypoalbuminemic/hypooncotic states. Forty percent of the increase in vascular volume reportedly persists for 24-36 hours. Decreased platelet aggregation and inhibition of factor VIII have been reported at high doses. Colloids have historically been used in the management of hypooncotic states, but to date, studies have failed to show a positive effect of synthetic colloids on outcome. In 2013, the FDA issued a warning that “HES solutions should not be used in critically ill adult patients" due to "risk of mortality and severe renal injury." While some continue to use these solutions in the acute setting of hypotension, they have become considerably less popular in the critical care setting.

**Challenging Scenarios**

**Cardiac Disease**
When dealing with heart failure patients, fluid therapy is generally best avoided unless clearly needed. It is not uncommon for patients to develop mild azotemia following initiation of diuretic and vasodilator therapy. Often the best answer in these situations is to decrease the dose of diuretic and provide a bowl of water. Trying to juggle simultaneous parenteral fluid and diuretic administration is usually more complicated than the situation requires. For animals that are reluctant to drink due to their illness, a nasoesophageal tube is a useful way to meet free water needs.
When a patient with significant cardiac disease does require fluid therapy due to decreased intake and ongoing losses, caution must be taken to avoid excessive sodium load. Patients with heart disease (failure) are prone to sodium and water retention, so fluids with lower sodium concentration (0.45% NaCl, plasmalyte 56) are frequently selected unless hyponatremia or hypovolemia are present. Colloids should be avoided in heart failure cases. Fluid therapy should be titrated to effect. Once it is determined that a true need for fluid therapy exists, arbitrarily picking a “1/2 maintenance rate” is not likely to provide for the animal's deficit and ongoing losses. Careful monitoring of body weight, urine output, CVP, PCV/TS, Azo, and electrolytes during fluid therapy can help with decisions related to ongoing fluid and cardiac therapy.

Pulmonary Contusion
Following pulmonary contusion, excessive administration of fluids has been shown to result in increases in lung water in dogs, potentially worsening oxygen exchange. However, concurrent traumatic injury and hypovolemia frequently necessitate aggressive fluid resuscitation. Fluid therapy should not be withheld in these patients. Results of studies comparing colloids, crystalloids, and hypertonic saline do not show a clear benefit to any particular fluid type. Current recommendations for fluid therapy in patients with pulmonary contusion are to provide crystalloids as needed to maintain adequate tissue perfusion. Monitoring of vital signs, blood gases, lactate, and central venous pressure may be helpful in assessing adequacy of resuscitation. Once adequately resuscitated, excessive fluid therapy should be avoided.

Traumatic Brain Injury
Periods of hypoxemia or hypotension have been implicated as predictors of poor neurologic outcome in human patients that have suffered head trauma. Fluid therapy should therefore never be restricted in the head trauma patient. Options for fluid resuscitation of the head trauma patient include:

Isotonic Crystalloid Fluids: Isotonic crystalloid solutions (Normosol-R, 0.9% Saline) are reasonable resuscitation fluids for the patient that has sustained TBI. Only that volume necessary to restore euvoolemia, provide maintenance, and balance out ongoing losses should be administered. Under-resuscitation should be avoided. Similarly, over-resuscitation may predispose to worsening cellular edema and increased intracranial pressure and should also be avoided.

Hypertonic Saline: Hypertonic saline resuscitation has the advantages of smaller volume resuscitation, rapid restoration of intravascular volume, improved contractility, and its osmotic effect at the level of the brain decreasing intracranial pressure. There is some evidence that hypertonic saline may disrupt the blood brain barrier and thus nullify some of its beneficial effects. Hypertonic saline may be administered at a dose of 4ml/kg of a 7.5% solution by slow IV infusion. Diligent monitoring of serum sodium concentration is critical after administration especially with concurrent administration of other hypertonics such as mannitol. The effects of hypertonic saline may be short lived. Concurrent isotonic crystalloid administration for maintenance purposes and provision for ongoing losses will be required.

Blood Products: Blood products are a very desirable resuscitation fluid in the patient with concurrent injuries resulting in hemorrhage and hypovolemia. Packed red blood cells or fresh whole blood may be used.

Hypernatremia
Every now and then a patient is presented with severe hypernatremia, which almost always results from either central (lack of secretion of ADH) or nephrogenic (lack of renal response) diabetes incipidus in conjunction with a disease or situation where water intake becomes limited. When hypernatremia develops acutely (eg., salt overdose), it should be normalized just as quickly, to avoid development of cerebral dissipation from the osmotic effects of the sodium. However, when hypernatremia has developed gradually, the brain protects itself by taking up osmolytes like inositol to buffer the effects of the hypernatremia. In this situation, great care must be taken to avoid correcting the sodium too quickly, as this can lead to cerebral edema. Ideally, sodium concentration should be lowered no more than 10-12 mEq/day.
To accomplish this, it can be helpful to calculate free water deficit:
Deficit (L) = 0.6 x Kg x [(plasma Na/desired Na)-1]

So if a 40 kg dog had a plasma [Na] of 175 and you wanted to lower it to 165 in 24 hours,
0.6 x 40 x [(175/165)-1] = 1.45 L of free water in 24 hours or 60 ml/hour.

“Free water” is water that does not contain solute (like sodium). Free water deficits can be replaced by giving water enterally, or by giving hypotonic fluids intravenously in patients that are unable to tolerate the enteral route. Remember that a liter of 0.45% NaCl only has 500 ml of free water (because the remainder has a physiologic concentration of sodium in it). In patients that are initially hypotensive and hypovolemic, volume deficits should first be treated with rapid infusion of 0.9% NaCl, with further correction of hypernatremia taking place once shock has been addressed.

These formulas should serve only as a starting point. Often there are excessive ongoing free water losses that make correction of hypernatremia more challenging. Rechecking electrolytes every 6-8 hours is essential to guide further fluid planning. Additionally, conjunctival desmopressin (1-4 drops q12) can be helpful to limit ongoing free water losses when they result from diabetes incipidus.

TL;DR
In most cases, fluid type is not likely to impact outcome and a balanced isotonic crystalloid is generally the best choice for maintenance of acid-base and electrolyte balance. Sodium chloride (0.9%) should be selected in hyponatremia, hypochloremic metabolic alkalosis, hypercalcemia, and some cases of hyperkalemia (but not necessarily in urethral obstruction). Maintenance solutions should be used in the management of hypernatremia, excessive hypotonic fluid loss, and significant heart disease (but not with severe ECF deficits or ongoing losses). Hypertonic saline may be of benefit when small volume resuscitation is needed, as in a shocky patient with traumatic brain injury. Colloid usage in veterinary medicine is increasingly rare, but may be useful in the setting of acute hypotension.
Gastric Dilatation-Volvulus Syndrome
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Gastric Dilatation-Volvulus (GDV) refers to the progressive dilatation and rotation of the stomach culminating in progressive shock and eventual death without rapid intervention. GDV is a very common condition affecting approximately 60,000 dogs per year in the US with mortality rates ranging from 10-60% (overall mortality is much closer to the 10% range with aggressive care). Much research interest has focused on risk factors for GDV to identify measures that may help to decrease the occurrence of the problem. Presently, the following factors have been identified:

- **Age:** Older dogs are more likely to develop GDV than younger dogs.
- **Pure-Breed status:** Dogs of pure breeding are 4.4 times more likely to develop GDV than mongrel dogs.
- **Size / Conformation:** An increased thoracic depth to width ratio has been associated with an increased risk of developing GDV. This parallels clinical observations that GDV occurs with greater frequency in large, deep chested dogs.
- **First-degree relative with a history of GDV**
- **Stress**

Other risk factors with limited evidence include faster speed of eating, exercise after eating, eating one large meal per day rather than two smaller meals, and smaller kibble size. The use of raised feeding bowls has also been associated with development of GDV though this likely reflects case selection bias in a population at risk. Overall, risk of developing GDV is likely a result of a complex interplay between age, genetic, conformational, environmental, and behavioral factors.

Despite the importance of identifying risk factors for GDV, even more crucial is an understanding of the pathophysiologic alterations that occur secondary to GDV. It is a close understanding of these mechanisms that will allow the attending clinician to most appropriately direct therapy. On triage of the dog with GDV, it is not uncommon to identify evidence of compromise to all of the major body systems (cardiovascular, respiratory, and central nervous system). Most of the clinical signs of cardiovascular compromise encountered on assessment of a dog with GDV (pale mucous membranes, slow capillary refill time, elevated heart rate, and poor pulses) can be explained by hypovolemic shock (failure of oxygen delivery to the tissues due to inadequate circulating volume). Mechanisms of hypovolemia in dogs with GDV include decreased venous return from the abdominal caudal vena cava and portal circulation, blood loss from torn short gastric arteries, and sequestration (3rd spacing) of fluids into the gastrointestinal system. Sepsis can also contribute to some of the clinical signs seen in severe GDV cases.

Assessment of the respiratory system will often show increased respiratory rate and effort resulting from stress, pain, and increased pressure on the diaphragm from the distended stomach, shock, or the possibility of primary pulmonary condition like aspiration pneumonia. CNS assessment may show an altered level of consciousness (LOC) as a result of decreased oxygen delivery to the brain. Problems identified on major body systems assessment will require immediate intervention.

**Initial Stabilization:**

Initial stabilization of the dog presenting with suspected GDV should focus on the treatment of hypovolemic shock. Oxygen therapy should be administered initially by mask or flow-by techniques while venous access (14-18g) is acquired via the cephalic veins. Central venous catheters should be avoided due to their long length, relatively small radius, and complexity of placement. Hind-limb catheters should ideally be avoided due to the decreased venous return from the caudal vena cava seen in dogs with GDV, but may be considered if vascular access options are limited. From the catheter, a PCV / TS / Blood Glucose / Venous Blood Gas (Emergency Database) should be collected. If possible, an entire CBC and Serum Biochemical Profile should be drawn prior to fluid therapy. Baseline physiologic data in addition to those gained through major body systems assessment should be collected. These include blood pressure, ECG, and pulse oximetry reading (SpO2).
Full physical examination should be performed while support staff is gaining venous access. Because GDV happens most commonly in large and giant breed older dogs, we must strive to identify concurrent problems that also occur in large and giant breed older dogs like dilated cardiomyopathy (DCM) and neoplasia. A medical history should also be collected.

Assuming that there is no contraindication to aggressive fluid support, volume resuscitation should commence with isotonic crystalloid solutions (LRS, Normosol-R, Saline). A shock rate of fluids (90 ml/Kg/hr) should be calculated and then administered in increments of approximately ¼ of the calculated dose, reassessing vitals after each bolus. It is important to remember that the endpoint of fluid therapy should be the normalization of vital signs, not the administration of some arbitrary volume. Some dogs may not need the entire 90 ml/Kg, while others will need significantly more.

Gastric decompression should be considered once volume resuscitation is underway. The authors prefer a combination of trocharization and oro gastric intubation. Trocharization is performed using a 16-18g over-the-needle intravenous catheter placed transabdominally into the stomach. Anatomically in GDV, the fundus will most often be located on the right side. Palpation for gas distention will help identify the optimal location for trochar placement. It is important to avoid the often-distended spleen while placing the trochar catheter. Trocharization has the advantage of being quick and easy to perform with minimal risks. It releases stomach gas, is not stressful, and does not require sedation. Disadvantages of trocharization are the risk of puncturing another abdominal structure (eg. spleen), and inability to evacuate liquid and food material from the stomach.

Orogastric intubation is indicated once the patient is more stable. Orogastric intubation has the advantages of being able to completely decompress the stomach and to lavage out any food material within. The primary disadvantages of this technique are the high degree of stress associated with oro gastric intubation (often requiring sedation or anesthesia) and the risk of esophageal or gastric injury. During lavage, aspiration pneumonia is a potential risk. To decrease risk and to minimize stress in the patient, endotracheal intubation is preferred, though some clinicians will employ heavy sedation and a mouth gag (3 inch roll of tape or a length of PVC tubing work well) to facilitate tube passage. Orogastric intubation should be performed using a tube appropriate for the size of the patient, well-lubricated, and measured from the mouth to the last rib (approximate location of the stomach). A piece of tape as a marker will ensure that the tube is not advanced too far into the patient. Once the stomach is entered and the gas decompressed, lavage of the stomach with warm water is performed.

A dose of broad spectrum antibiotics is indicated early in the course of therapy and should be continued through surgical intervention and beyond if specific indications exist.

**Radiography**

Abdominal radiography is indicated for the definitive diagnosis and differentiation of gastric dilatation (GD) from GDV. Ideally, radiographs should precede decompression to maximize chances of obtaining an accurate diagnosis. However, if the patient is particularly unstable, emergency decompression may precede additional diagnostics.

Right lateral abdominal radiographs should be performed. The radiographic sign most consistent with GDV is compartmentalization of the stomach and displacement of the pyloric antrum dorsally. Other radiographic signs of note are gas within the stomach wall (indicating gastric necrosis), free peritoneal gas (most likely indicating gastric perforation), loss of abdominal detail (from peritonitis or bleeding from the short gastric arteries) and splenomegally (indicating splenic torsion or possibly splenic venous thrombosis). Most dogs with GD and GDV will not tolerate VD views well and such projections could compromise patient stability. In dogs that are older than 7 years of age, opposite lateral radiographic views of the thorax should be obtained to identify concurrent illnesses such as heart disease or neoplasia.

**Anesthesia for dogs with GDV:**

Anesthetic protocols for dogs with GDV should involve the utilization of drugs that are sparing of the cardiovascular system. Pre-oxygenation prior to induction is indicated in any critically ill patient undergoing an anesthetic procedure. Placement of an ECG, pulse oximeter, and oscillometric blood pressure monitor will facilitate monitoring during induction. Two appropriate anesthetic protocols are as follows:
(1) Hydromorphone (0.1 – 0.2mg/Kg), Midazolam (0.2mg/Kg), Lidocaine (1-2mg/Kg)
(2) Ketamine (100mg/ml), Valium (5mg/ml) Give 1cc/20lbs body weight of a 50:50 volume mixture. Example: A 100lb Great Dane would receive a total of 5cc (or 2.5ml of Ketamine and 2.5ml of valium).

Anesthetic drugs should always be administered “to effect”. Ongoing monitoring of oxygen saturation, ECG, and blood pressure are indicated throughout surgery. Even if the patient is breathing spontaneously, ventilation may not be effective and intermittent positive pressure breaths should be administered. Fluid therapy should be considered at approximately 10-20ml/Kg/hr or as needed to maintain intravascular volume and blood pressure.

**Surgical Management:**
Surgical goals in dogs with GDV should include replacement of the stomach into its normal anatomic location, control of hemorrhage, resection of areas of necrosis or suspected necrosis, splenectomy if indicated, and finally gastropexy. It is crucial to perform a complete abdominal exploratory in dogs with GDV and the abdominal incision should extend from xiphoid all the way to the pubis. Understanding the anatomy of GDV is crucial to restoring the stomach to its normal position. The most common direction of rotation is clockwise. When viewed from a caudal to cranial direction with the patient in ventro-dorsal recumbency, the pylorus has moved from the right side of the abdomen to the left side of the abdomen while tracking along the ventral abdominal wall. Clockwise rotation will result in the omentum being pulled over the stomach. Upon opening the abdomen, identification of the omentum over the stomach is an indication of a clockwise rotation. Complete gastric decompression will facilitate relocation of the stomach.

Hemorrhage commonly originates from torn short gastric arteries and gastric necrosis is most commonly identified along the greater curvature and up along the cardia of the stomach. Be sure to examine all sides of the stomach as necrosis is commonly found on the underside of the stomach as it is viewed from the surgical incision. Gastric resection should be performed using a two-layer technique. Splenic torsion or thrombosis is an indication for resection. If the spleen is torsed, it should NOT be de-rotated prior to removal.

Numerous methods for gastropexy (pyloric antrum to right abdominal wall) have been evaluated and despite differences in tensile strength evaluated in-vitro, incidence of recurrence has not been found to be significantly different between the various methods. Unacceptable methods for gastropexy include suturing the stomach into the abdominal closure line, and methods that do not involve an incision in the seromuscular layer of the stomach (simply scarifying the stomach and the right abdominal wall and suturing the two together). At our institution, the incisional gastropexy is preferred due to its ease and the speed with which it can be performed. A tube gastropexy has the advantage of allowing postoperative feeding and gastric decompression. Prior to closure, the abdomen should be lavaged and checked for sites of ongoing hemorrhage.

**Postoperative Management and Complications:**
Of greatest importance to the postoperative management of the dog with GDV, is the maintenance of appropriate delivery of oxygen to the tissues. Oxygen support in the immediate postoperative period will minimize the chance of bouts of arterial oxygen desaturation. If the patient is not saturating > 94% on oxygen support, or if there is increased respiratory rate and effort or abnormal lung sounds on auscultation of the thorax, thoracic radiographs are indicated to help identify the complicating process (pneumonia). Atelectasis plays a significant role in post-operative hypoxemia. Frequent alteration of patient position should help combat atelectasis. Early standing and short walks will also help combat this process.

Volume support should be directed to replace deficits, provide for maintenance, and to balance out ongoing losses (generally in the range of 3-5x maintenance requirements initially). It is not uncommon for patients to return from the surgical theater and require a bolus of fluids due to increased losses during surgery. Assessment of another Emergency Database will help direct fluid therapy and electrolyte supplementation. If the PCV drops below 20% and the patient is showing signs of pale mucous membranes, slow CRT, increased heart rate, or weak pulses, blood product replacement may be indicated.

Pain control is critical in the postoperative GDV patient. Pure agonists such as fentanyl (CRI: 3-5 ug/kg/hr), hydromorphone (0.1-0.2 mg/kg IV q4h, or CRI: 0.025 mg/kg/hr), or morphine (0.5-1 mg/kg SQ q4h) may be used for patients with moderate to severe pain. Ketamine can be useful for the relief of somatic pain, and may be used in conjunction with narcotics at a constant rate infusion of 0.15-0.6 mg/kg/hr.
Lidocaine may provide adjunctive analgesia in addition to free radical scavenging properties, and may also be added at a rate of 1.5-3 mg/kg/hr. If using constant rate infusions, a loading dose equal to the hourly rate should initially be administered.

Ventricular arrhythmias in the form of Ventricular Premature Contractions (VPCs), accelerated idioventricular rhythms, and ventricular tachycardia are common after GDV. Irritable ventricular foci likely develop due to decreased delivery of oxygen to the heart during shock, ongoing decreased oxygen delivery to the heart postoperatively, ischemia reperfusion injury, and electrolyte and acid-base disorders. Numerous recommendations exist as to when to institute treatment for these arrhythmias. Guidelines to consider prior to pharmacologic intervention are as follows:

1. Correct hypoxemia. SpO2 should read greater than 95%
2. Restore euvolemia and blood pressure to normal
3. Correct acid-base and electrolyte abnormalities
4. Provide appropriate analgesia

If arrhythmias persist at an overall heart rate of greater than 150bpm, in the face of attempts to correct physiologic derangements as described above, pharmacologic intervention in the form of lidocaine (2mg/Kg IV repeated once if necessary and followed by 50-80μg/Kg/min CRI ) will likely solve the problem.

Many dogs with GDV are predisposed to developing dilutional coagulopathy and consumptive coagulopathy. Assessment of a coagulation profile or, at minimum, an Activated Clotting Time (ACT) will direct the need for clotting factor support in the form of fresh frozen plasma. Vitamin K₁ will NOT be useful in the coagulopathy seen in dogs with GDV because GDV is not a process that antagonizes Vitamin K₁ recycling or absorption from the gastrointestinal system.

Following gastric resections, wound dehiscence may occur in a small percentage of cases. This will result in signs of peritonitis approximately 48-72 hours postoperatively. Twice daily, patients should have abdominal palpation performed to evaluate for abdominal pain. Abdominal pain, fever, or failure to thrive postoperatively is an indication for abdominocentesis and cytologic evaluation.

The final common postoperative complication of GDV is decreased gastric motility. This tends to be most common in the most critically ill of patients (generally those that had evidence of gastric necrosis). Placement of a nasogastric tube at the time of surgery, or using a tube gastropexy allows for gastric decompression and will minimize the likelihood of regurgitation, vomiting, and subsequent aspiration pneumonia. In addition, early “trickle” feeding can be instituted to begin nutrient delivery to the stomach and small intestine. Use of motility agents like Metoclopramide can be used to help combat this complication.

Prognosis:
Over the years, numerous studies have tried to identify prognostic factors for dogs with GDV. To date, the most substantiated of these are the presence or absence of gastric necrosis, and the blood lactate concentration prior to fluid therapy. In one large scale study, 98% of dogs without gastric necrosis survived and only 66% of those with gastric necrosis survived.⁶ Gastric necrosis itself does not necessarily cause mortality, but is more likely a marker for more severe compromise to the major body systems, and a more critically ill patient.

In the same study, 99% of dogs with a blood lactate <6.0mmol/L prior to fluid therapy survived and only 58% of those with a blood lactate >6.0mmol/L prior to fluid therapy survived.⁶ Lactate is a marker of anaerobic glycolysis (as occurs when decreased oxygen is delivered to the tissues) and has been found to be prognostic for numerous human medical and surgical problems.

Conclusion:
GDV is a complex disease process resulting in a variety of challenging physiologic derangements. Through early aggressive preoperative management, appropriate and skilled surgical intervention, and vigilant postoperative monitoring and supportive care, a positive outcome can be achieved in the vast majority of patients.
References:
Anemia is a common problem in the emergent patient and may result from blood loss, hemolysis, or decreased erythrocyte production. Anemia may be detrimental to the critically ill patient when it causes a decrease in oxygen delivery to the tissues. In the following session, we'll review indications for blood transfusion in veterinary patients as well as guidelines for ensuring safe collection, storage, and delivery of blood products. This document is meant to serve as a reference for practitioners who wish to provide blood product support, but lack blood banking capabilities.

The Transfusion Trigger

For many years there has been an ongoing search for a universal "transfusion trigger", a set of conditions under which transfusion is considered to be indicated and for which no further justification is required. One of the earliest examples of a transfusion trigger was the “10/30 rule”, first published by Adams and Lundy in 1942, which stated that presurgical patients should be transfused if their hemoglobin concentration was less than 10 g/dl or their hematocrit was less than 30%.

More recently, concerns in human medicine about transmission of infectious diseases and a growing awareness of other risks associated with blood transfusion lead to a reassessment of transfusion practices. It was recognized during this time that healthy animals and people could tolerate very low hematocrits as long as intravascular volume was maintained. Furthermore, human patients who declined blood transfusion for religious reasons were also able to survive surgical procedures despite suboptimal hematocrits. In recognition of these observations, the practice of transfusing to a target hematocrit has begun to give way in favor of risk to benefit analysis for the individual patient.

Risks of transfusion

Some of the more commonly cited risks of blood transfusion include fever (febrile non-hemolytic transfusion reactions), hypersensitivity reactions, and acute or delayed hemolytic reactions. These types of transfusion reactions are thought to occur in approximately 3% of veterinary patients receiving blood products. Disease transmission is another potential complication of blood transfusion, though the prevalence has not been reported in veterinary patients. Acute lung injury, microembolic disease, electrolyte and acid-base disturbances, and coagulopathy are other known risks. In addition to these well-recognized risks, there is also increasing evidence that blood transfusions may have significant immunosuppressive effects. In a landmark study of 1717 human patients admitted to an ICU trauma unit, patients receiving transfusions were 6 times more likely to develop nosocomial infections than those who did not receive blood products. Mortality in the transfused group was also twice that of the non-transfused group. Similar findings of increased infection rates and mortality following transfusion have been documented in human patients undergoing surgery for penetrating abdominal trauma, fracture repair, gastrointestinal cancer, cardiac bypass, spinal surgery, and hip replacement. Although the association between blood transfusion and harmful effects such as immunosuppression, acute lung injury, and proinflammatory responses are still poorly understood, there is enough evidence at this time to warrant caution in their use.

Benefits of transfusion

The main goal of red blood cell transfusion is to reduce the morbidity and mortality associated with inadequate delivery of oxygen to the tissues. In order for this goal to be valid, it is important to establish (a) that anemia in fact contributes to morbidity, (b) at what level adverse effects are likely to occur, and (c) that transfusion in these patients is associated with improved outcome. In experimental models, healthy animals subjected to acute hemodilution were able to tolerate hematocrits as low as 10-15% as long as intravascular volume was maintained. Below hematocrits of 15% electrocardiographic changes consistent with ischemia began to appear, and at hematocrits of less than 10%, increased lactate production, myocardial depression, and death occurred. Healthy human volunteers subjected to hemodilution were similarly able to tolerate hematocrits as low as 15% with no evidence of inadequate oxygen delivery. At the lowest hematocrits, many subjects complained of fatigue, but no other symptoms were reported.
However, there is some evidence that even mild to moderate anemia may contribute to mortality in clinical patients. In a study of nearly two thousand human patients undergoing surgery who refused transfusions for religious reasons, risk of death was shown to increase as hematocrits decreased below 30%. In this study, even mild anemia was associated with some increase in the risk of death, and patients with concurrent cardiovascular disease were much less tolerant of anemia than those without concurrent disease. Because no patient in this study received transfusions, the study was not able to show, however, that administration of transfusion would have resulted in an improved survival rate. In a follow-up study of transfused versus non-transfused patients undergoing hip surgery, at hematocrits of 24% or higher, the administration of blood transfusions was not shown to decrease mortality. In another recent study, human ICU patients were randomized to restrictive (hematocrits maintained at > 21%) or liberal (hematocrits maintained at >30%) transfusion strategies. Hospital mortality rates, multiorgan dysfunction scores, and cardiac complication rates all favored the restrictive transfusion strategy. Benefits to liberal transfusion were only seen in patients with underlying cardiovascular disease.

From studies such as these, it seems clear that anemia is associated with poor outcome. However, it is less clear that transfusion in certain populations of patients will provide benefits in terms of improved tissue oxygen delivery and survival. We may be able to extrapolate that critically ill patients with hematocrits below 15% may benefit from transfusion, while those with stable blood volume and hematocrits greater than 24% are unlikely to benefit from transfusion. In between these values is a gray zone where some patients may benefit from transfusion and others will not.

**When to Transfuse?**

Students frequently ask how low the hematocrit must fall before we decide to transfuse. Hopefully the point has been made by now that hematocrit levels alone should not serve as a transfusion trigger. However, they may still be used as a rough guideline for when to consider transfusion as a possible treatment. In *otherwise healthy patients*, current guidelines supports the safety of hematocrit levels as low as 18% as long as normovolemia is maintained. Other factors to consider when deciding on the need for blood transfusion should include clinical signs of anemia, the rate of ongoing losses, the chronicity of the anemia, and the presence of co-morbidity that may limit the ability of the patient to compensate for their anemia.

**Blood Donor Screening:**

Canine blood donors should be at least 25 kg (to donate ½ unit of blood or 225 ml), in good health and temperament, current on vaccinations, and not receiving any medications. At Michigan State University, donors are tested for all blood group antigens for which a commercial test is available, but at a minimum testing for 1.1 and 1.2 should be performed as these antigens are associated with acute hemolytic transfusion reactions in sensitized individuals. Infectious disease screening should include: Heartworm antigen, Babesia spp, Ehrlichia spp, Anaplasma, Mycoplasma, Brucella, and Leishmania. Trypanosoma and Bartonella should be considered in places where endemic. Feline blood donors should be at least 5 kg, strictly indoors, in good health, and current on vaccinations. Echocardiogram is strongly recommended for donor safety to rule out occult cardiomyopathy. Feline donors should be blood typed and screened for infectious disease as follows: FeLV, FIV, Mycoplasma, heartworm antibody, and Bartonella. Cytauxzoon and Ehrlichia should be considered in places where endemic.

**Blood Collection and Storage**

Blood is collected from the jugular vein into a closed collection system following aseptic preparation of the skin. If the blood is to be administered immediately, it may be anticoagulated with heparin (625 u per 50 ml blood) or 3.5% sodium citrate (1 ml anticoagulant/9 ml blood). Because these anticoagulants lack preservatives, the blood collected in this fashion may not be stored for any length of time. CPDA-1 (14 ml anticoagulant/100 ml blood) is an anticoagulant with a preservative that increases red cell survival by serving as a substrate for synthesis of ATP. Packed red blood cells in CPDA-1 may be stored for 4 weeks 4°C. The use of Adsol (an electrolyte solution containing adenine, saline, glucose, and mannitol) may further improve length of storage and post transfusion viability.
Typing and Crossmatching

Blood typing should ideally be performed in all dogs to optimize allocation of resources (administering 1.1 positive blood to 1.1 positive recipients and reserving universal blood for 1.1 negative recipients) and to avoid sensitizing recipients to blood alloantigens. In an emergency, a dog that has never been transfused previously may safely receive transfusion without blood typing, because dogs lack naturally occurring alloantibodies. Dogs that have previously been transfused (5 or more days prior), that have had litters of puppies, or that have an unknown transfusion history should have a major crossmatch performed prior to transfusion to rule out incompatibility.

All cats have naturally occurring alloantibodies to foreign blood types. Transfusion of type A blood to B cats results in a potentially fatal transfusion reaction. Transfusion of type B blood to type A cats may result in hemolysis and less severe transfusion reactions. Consequently, all cats must be blood typed prior to transfusion.

Recently, a new blood type (Mik), distinct from the AB blood group system, has been reported in cats. Most cats are positive for the Mik antigen. However, Mik-negative cats do exist, and possess naturally occurring Mik-alloantibodies. These cats may experience acute hemolytic transfusion reactions after receiving AB compatible blood. Given the clinical relevance of naturally occurring Mik alloantibodies, all cats should be crossmatched prior to transfusion, even if they have not previously received blood products.

Administration

The optimal route of blood administration is intravenously, through the largest catheter diameter possible for the size of the patient. When large quantities of blood and fluids must be given rapidly in the face of exsanguination, 14 or 16 gauge over the needle catheters (Angiocath®, Becton Dickinson Infusion Therapy Systems Inc) can be used in the jugular veins.

Refrigerated blood products should ideally be warmed to room temperature prior to administration to avoid inducing hypothermia in the recipient. Cold blood also has a much higher viscosity than that of warmed blood and as a result cannot be given as quickly. Packed cells may be warmed by immersing the unit in a warm water bath at 37°C or by passing the coils of the transfusion tubing through a fluid heater or basin of warm water. Plasma may also be warmed in a warm water bath at 37°C. Care should be taken when handling plasma prior to warming as the plastic blood bag is fragile when frozen and susceptible to cracking.

Blood products should always be administered using a commercial blood administration set with an in-line filter (Baxter Healthcare Corp, Deerfield, IL) to remove blood clots and particulate debris. For small blood volumes administered to cats and small dogs by syringe, a pediatric 18 μm blood filter (Hemo-Nate Filter, Gesco International, San Antonio, Tx) may be placed between syringe and extension set.

Blood should not be administered with any fluid other than 0.9% sodium chloride. Calcium containing solutions such as lactated Ringer’s (B. Braun Medical Inc, Irvine, CA), Normosol (Abbott Laboratories, Abbott Park, IL), or Plasmalyte (Baxter Laboratories, Deerfield, IL) may bind citrate and thus initiate coagulation. Hypotonic fluids like D5W or half-strength saline may result in red cell lysis as a result of osmotic fluid shifts. Concurrent administration of other drugs through the same catheter should also be avoided.

Volume and Rate

The volume of blood to be transfused can be calculated as follows:

Transfusing 10 ml/kg of packed cells or 20 ml/kg of whole blood typically raises the PCV by approximately 10%. In the setting of acute blood loss, PCV may not provide a reliable indicator of blood loss. In these cases, blood should be given to effect or in proportion to estimated losses.

Rates for blood transfusion are variable, depending on the degree of blood loss and the rate of ongoing losses. Ideally, the transfusion time for a unit of blood should not exceed 4 hours due to concerns about bacterial proliferation once the product has been warmed. An initial rate of 0.25 ml/kg for the first 30 minutes has been recommended while the patient is monitored for transfusion reactions. If no adverse effects are
noted, the rate may then be increased to as much as 10-20 ml/kg/hr. However, in the setting of exsanguinating injury and imminent death, blood may be given as rapidly as possible.

**Autotransfusion**
Direct aspiration and reinfusion is easily accomplished using a 60 cc syringe and three-way stopcock. Using this method, blood may be aspirated from the thoracic or abdominal cavity into a blood collection bag or container and then reinfused through a micropore filter (Hemo-Nate Filter, Gesco International). Anticoagulants are generally not needed because blood has been sitting in contact with pleural or peritoneal surfaces for over one hour undergoes defibrination. However, with rapid hemorrhage there is insufficient time for defibrination, and CPD-A should be added at a dose of 7 ml per 50 ml blood. Direct aspiration and reinfusion can be very useful in cases like hemothorax secondary to rodenticide or trauma where evacuation of the chest cavity is needed and preservation of viable cells is desired.

Autotransfusion has a number of advantages and disadvantages that need to be taken into consideration prior to administration. The biggest advantage is a ready source of compatible blood that can be given quickly and inexpensively, without the need for warming, typing, crossmatching, or infectious disease screening. Disadvantages include the potential for hemolysis, coagulopathy, blood contamination, and acute lung injury. Autotransfused blood is subject to hemolysis as a result of prolonged contact with injured serosal surfaces and from mechanical trauma when suction systems are used. Coagulopathies may also be a concern because pleural or peritoneal contact activates the coagulation system, resulting in reduced platelet and clotting factor levels in autotransfused blood that may lead to dilutional coagulopathy when large volumes are reinfused. Additionally, because autotransfused blood may contain large amounts of FDPs, red blood cell fragments, activated leukocytes, platelets, and inflammatory mediators that may initiate coagulation, autotransfusion may exacerbate consumptive coagulopathies. Acute lung injury is another potential complication following autotransfusion, and is believed to result from microembolization of cellular aggregates, fat, and protein to the pulmonary vasculature. For this reason, the use of micropore filters (18 μm) has been recommended when autotransfused blood is administered. Because there is the potential for spread of neoplasia if the shed blood contains tumor cells, and for contamination with bacteria if GI perforation may have occurred, autotransfusion should not be used in these situations.

**Monitoring of blood administration**
Prior to administration of blood products, baseline values for PCV, total solids, and vital signs should be obtained and blood components should be checked carefully for signs of discoloration. During administration, vital signs should be rechecked initially every 15 minutes for the first half hour, then every 30 minutes thereafter to monitor for signs of transfusion reaction. A rise in body temperature of 1°C, the development of tachycardia, bradycardia, tachypnea, vomiting, urticaria, erythema, angioedema, or pigmenturia should prompt investigation into the possibility of transfusion reaction.

**Conclusion**
Blood transfusion is a valuable and potentially life-saving technique that should not be withheld in patients at risk. However, there is a growing body of evidence that transfusions may contribute to increased morbidity and mortality, and this perception has lead to a change in the way that transfusion requirements are assessed. By weighing the risks of transfusion against the potential benefits, by carefully screening blood donors, and by using appropriate precautions in the collection, storage, and administration of blood products, clinicians may be better able to optimize blood transfusion management in the emergent patient.

**References**

WHERE THERE’S SMOKE, THERE’S FIRE:
MANAGING HOUSE FIRE VICTIMS
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The emergency clinician is occasionally called upon to treat smoke and burn injuries resulting from house fires or other sources of thermal, chemical, or electrical injury. Most burn wounds seen in veterinary medicine are relatively minor, possibly because animals with severe burns and smoke inhalation are less likely to be rescued from the scene of a house fire. However, life threatening burns and inhalation injury are being seen with increasing frequency and the emergency clinician should therefore be familiar with their pathophysiology and management.

Smoke Inhalation
Smoke inhalation can be associated with a variety of problems, including respiratory irritation or distress, neurologic effects, and complicating factors such as the development of bacterial pneumonia. Additionally, the presence of toxic gases such as carbon monoxide, hydrogen cyanide, hydrogen sulfide and others may further contribute to respiratory irritation and impaired oxygen delivery. The majority of fire-related deaths in animals result from carbon monoxide poisoning rather than the fire itself.

Inhalation of superheated smoke particles has numerous adverse effects. Smoke is a potent respiratory irritant, resulting in bronchoconstriction. The larynx and glottis can become edematous as a result of thermal burns, leading to upper airway obstruction. Chemical and thermal damage to the cells lining the airways leads to sloughing of the tracheobronchial mucosa, impairment of the mucociliary escalator, and formation of cellular casts that may obstruct the lower airways and promote bacterial growth. Disruption of respiratory epithelium and vascular endothelium leads to exudation of proteinaceous fluid into the terminal airways and further contributes to respiratory compromise, impaired surfactant production, and bacterial growth.

Carbon monoxide poisoning frequently occurs in conjunction with inhalation injury. Carbon monoxide has approximately 200 times the affinity for hemoglobin that oxygen does, allowing it to displace oxygen from the hemoglobin and form carboxyhemoglobin instead. Oxygen delivery to the tissues is therefore decreased, and tissue hypoxia may occur, particularly to organs with high oxygen demand, such as the brain and heart. Clinical findings include cherry-red mucous membranes, dyspnea, vomiting, dizziness, headache, altered mentation, and loss of consciousness. Serious intoxications may lead to pulmonary edema, seizures, coma, and death. Delayed neurologic sequellae have also been reported in dogs.

Burn Injury
Skin burns significantly affect patient outcome and increase morbidity and mortality. Animals that have been close enough to the fire to sustain skin burns usually have the most severe pulmonary complications associated with smoke inhalation. Burns are commonly classified according to the extent of body surface involved and the depth of injury to the skin. Extent of injury is initially estimated in human burn patients using “the rule of nines”. This rule divides the adult human body into areas corresponding to 9% of the total body surface area, or multiples of 9%. For example, each forelimb comprises approximately 9% of total body surface area; each hind limb, 18%; head and neck, 9%; chest and abdomen, 18%; back, 18%; and perineum, 1%. Body surface area percentages vary in children, and as such, the rule of nines is not typically used in children less than 10 years of age. Although the rule of nines has been cited in veterinary texts, it seems similarly unlikely that these percentages accurately describe the majority of veterinary patients. Other methods of estimating extent of injury include serial halving (Do burns cover more than half the patient’s surface area? If not, do burns cover ¼-½ the surface area? and so forth), or measuring the burn area in centimeters and using a chart to calculate meters squared from the patient’s body weight in kilograms. Recently, resuscitation burn cards have also been used in veterinary patients. These 8.5 x 5.3 cards are identical in size to plastic credit cards and have a surface area of 45 cm². The number of cards needed to cover the burn is consequently multiplied by 0.45 to get total surface area. This number is then divided by the patient’s m² obtained from a weight to m² chart as described above.

Depth of injury may be described as first-, second-, or third-degree, or using the more recent terms, partial- and full-thickness. First-degree burns involve only the epidermis (like a sunburn), and are bright red, non-blistered, and painful. First-degree burns typically heal within 5 days without scarring, and are therefore...
not included in the calculation of extent of burn injury unless they exceed 25% of body surface area. Second-degree, or partial-thickness, burns involve all epidermal layers and extend to various depths within the dermis. Superficial partial-thickness burns involve the epidermis and less than ½ of the dermis, and are characterized by blisters, pain, blanching in response to pressure, and intact hairs. The surface may appear moist, red, or mottled. Injuries of this depth typically heal without serious scarring within 2-3 weeks. Deep partial-thickness burns involve destruction of the deep dermal layers and may appear dry, or blistered and moist. As skin thickness is not uniform, partial-thickness burns may interdigitate with full thickness burns, appearing mottled-red intermixed with whitish areas. Deep partial-thickness burns do not blanch, lose hair easily, and heal more slowly, producing scarring and loss of function. They may easily progress to full-thickness injuries as a result of edema, infection, thrombosis, or mechanical injury. Third-degree, or full-thickness, burns involve destruction of the entire dermis, usually extending into the subcutaneous tissues. They are dry, leathery, lack sensation, and appear white or charred. Healing of these injuries can occur only by contracture and epithelial migration from the periphery, or through excision and grafting. Depth of injury can be difficult to assess initially, and usually requires repeated evaluation over the first 24 hours for accurate determination. Once this information is collected, burned patients may be divided into minor, moderate, or severe categories for the purposes of treatment planning.

Pathophysiology of Burn Shock

Following severe burns (>20% TBSA), a severe systemic inflammatory response may develop within minutes, leading to cardiovascular collapse and multiorgan system failure if not quickly addressed. These systemic manifestations are driven by loss of the protective skin barrier, as well as release of inflammatory mediators from within the damaged tissues. A diffuse “capillary leak” syndrome develops, resulting in marked decreases in effective circulating volume as well as the development of edema in injured and non-injured tissues. Extensive tissue edema leads to tissue hypoxia at the junction between burned and non-burned tissues (the “zone of ischemia”), and may have adverse effects on depth of burn injury. Cardiac output decreases within the first eight hours of burn injury secondary to hypovolemia and myocardial depression associated with release of inflammatory mediators. Arterial blood pressure may be misleading however, as burn patients may have normal or increased blood pressure despite significant hypovolemia due to vasoconstrictive substances released from the burn wound.

Following successful resuscitation, microvascular leak typically “seals” after 18-24 hours. Hypermetabolic response develops during this time resulting in weight loss, protein catabolism, and insulin resistance. The hypermetabolic response typically persists until all wounds are closed, and continues for some time afterwards.

Sepsis is one of the major causes of death among burn patients. In addition to wound infections, respiratory infections, and catheter-related infections, decreased gastrointestinal perfusion in the first 24 hours following burn injury leads to compromised integrity of the mucosal barrier and allows passage of bacteria and endotoxin.

Prehospital Treatment of the Burned Patient

The first consideration in treatment of the burned patient is to stop the burning process. Flames should be extinguished and any collars or harnesses that may become constrictive should be removed. Because the skin is slow to cool, the burning process may continue for some time after the patient is removed from the heat source. For this reason burned areas should be cooled with running water for up to 10 minutes. Alternatively, cool wet towels can be placed over the burn areas. Ointments should not be applied at this time as these may hinder the subsequent assessment of extent of injury. Cold water or ice should also not be used as this can rapidly decrease the patient’s body temperature and may contribute to increased wound depth by inducing vasoconstriction. To avoid hypothermia during transport, the patient should be wrapped in several clean, dry sheets or blankets.

Primary and Secondary Surveys

A primary survey should be performed to determine the extent of injury and to institute treatment as needed. Ensuring a patent airway and supporting breathing should be the first priority, followed by shock resuscitation. 100% oxygen should be administered to any patient suspected to have smoke inhalation injury to hasten the elimination of carbon monoxide. Intubation or emergency tracheostomy may be required if airway edema is severe.
Vascular access may be difficult in hypovolemic, burned patients. Ideally, short peripheral catheters should be placed in non-burned areas, though burned areas may be used in the first 24 hours. If burned sites are used for catheterization, the catheters should be removed within 24-48 hours due to bacterial colonization of these areas. Intraosseous catheters are another good alternative for patients in whom vascular access is limited. Central lines may be required in patients with large burns, those needing parenteral nutrition, or those requiring central venous pressure monitoring, but their use should be avoided whenever possible due to the risks associated with hypercoagulability in burned patients.

Following initial stabilization, a secondary survey should be performed to identify concurrent injuries. The face, oral cavity, and pharynx should be examined for the presence of burns or particulate debris that may indicate inhalation injury. The eyes should be evaluated for the presence of conjunctivitis, particulate material, or corneal ulceration. Corneal ulcers are common secondary to thermal injury or abrasion by particulate material, so fluorescein staining should always be performed. A topical anesthetic such as proparacaine may be used to facilitate examination behind the third eyelids for foreign material, and the eyes should be copiously flushed with sterile saline. Corneal ulcers may be treated with triple antibiotic ophthalmic ointment and atropine ophthalmic drops.

Baseline radiographs should be obtained to evaluate for changes related to smoke inhalation or traumatic injury. Chest radiographs may be normal initially, or bronchial markings may be present. The development of pulmonary infiltrates or lobar consolidation may suggest pneumonia.

Complete blood count, serum biochemistry panel, and urinalysis should be obtained upon admission. The presence of myoglobinuria may indicate a need for higher fluid rates to avoid renal tubular damage. Coagulation testing should be performed, as burned patients may suffer from hyper- or hypocoagulable states. Blood typing may be indicated if surgery is anticipated for large burns, as these procedures frequently result in significant blood loss.

**Fluid Therapy**

The goal of fluid therapy in the burn patient is to restore and maintain perfusion to the tissues while keeping edema fluid to a minimum. The greatest amount of fluid loss in burn patients occurs during the first 24 hours as a result of “capillary leak”. Fluids given during this time rapidly leave the vasculature, with colloids having no benefit over crystalloids due to the leakiness of the endothelium. Crystalloids, such as lactated Ringer's solution, are therefore usually the fluids of choice for the first 24 hours. Fluid requirements can be estimated based on percentage of body surface area burned using the Parkland formula, now termed the “Consensus” formula. LRS is given at 4 ml/kg x % TBSA (total burn surface area), with one half of the calculated volume given within the first eight hours, and the second half given over the next 16 hours. Urine output should reach 0.5-1 ml/kg/hr within the first three hours. If it falls below 0.5 ml/kg/hr, more fluid is needed. Lasix should not be used to increase urine output, as this will further deplete effective circulating volume as well as invalidate the use of urine output as an indicator of shock resuscitation.

Many resuscitation formulas recommend adding colloids at 0.5 ml/kg/day x % TBSA after 24 hours, as colloids are more likely to be retained within the vasculature at that time. (Note: some formulas advocate colloid supplementation as early as 8 hours post-burn). Hetastarch, fresh frozen plasma, or albumin may be used, though it is interesting to note that albumin supplementation in burn patients has not been associated with decreased mortality nor mobilization of tissue edema within the first week. Crystalloids are continued only at doses needed to maintain urine output, approximately 1.5 ml/kg/day x %TBSA.

It is important to emphasize that these fluid formulas should be used only as guidelines, and should be frequently reevaluated and adjusted based on physiologic parameters. Additionally, because these formulas have been derived from experiences with human patients and experimental models in animals, they should be applied cautiously in clinical veterinary patients, and dose reduction may be appropriate in cats.

**Wound Care**

Patients with small burns rarely develop overwhelming wound sepsis, and medical management for several days usually allows better determination of wound depth and extent. Wounds should be gently clipped of hair and then rinsed or soaked in dilute povidone-iodine solution. Animals with thick coats may hide more extensive wounds than initially suspected, so liberal clipping should be performed in these cases. After the wounds are cleaned, topical agents may be applied to decrease pain, prevent desiccation, and delay bacterial growth. Silver sulfadiazine is used most commonly as it has broad antibacterial activity, is soothing, and has no systemic effects. Topical agents can be applied directly to wounds with a clean tongue.
depressor, or the burn can be covered with impregnated dressings. Gloves should be worn at all times during wound care to avoid spread of resistant organisms.

The choice of dressing is a much-debated topic. Of critical importance is the maintenance of a moist environment to promote rapid wound healing. This may be accomplished through the use of semi-occlusive dressings, manuka honey, or various types of hydrogel shown to speed healing and to decrease scarring of partial thickness wounds. Bandages should be loose enough to avoid putting additional pressure on the wounds.

Patients with more extensive burns generally do better if full thickness wounds are excised within the first week, starting 24-48 hours following burn injury. Early wound excision has been shown to circumvent the development of wound sepsis and reduce morbidity and mortality, length of hospital stay, and pain in patients with large burn wounds. Burns >20% total body surface area may require staged procedures, and burns > 50% TBSA make closure with autograft impossible. Once autograft closure is no longer feasible, temporary closure may be performed using cadaver allografts, porcine xenograft, or synthetic skin substitute. Following the recent Camp fires, tilapia skin received attention as a promising biologic dressing in veterinary patients, and cod skin is now commercially available for similar use (Kerecis Phytoceuticals, Iceland). Research is currently underway to evaluate the use of synthetic membranes such as Integra (Integra Life Sciences, Plainboro, NJ) that mimic vapor transmission characteristics of normal skin and allow fibrovascular ingrowth from the host, ultimately undergoing biodegradation.

Prophylactic antibiotic usage is controversial as penetration of the eschar is unlikely and the potential for development of antibiotic resistance exists. As such, antibiotic therapy is generally reserved only for documented infections and should be based upon culture and sensitivity of full thickness eschar biopsies. Excision of eschar has been associated with bacteremia however, so intraoperative antibiotic administration has been recommended.

Inhalation Injury
Management of smoke inhalation is typically supportive. The head should be elevated and excessive fluid therapy avoided to minimize development of edema. Bronchospasm may be treated with systemic β agonists such as terbutaline, or inhaled albuterol administered via spacer (Aerokat, Trudell Medical, London, Ontario). Prophylactic antibiotics have not been shown to reduce morbidity or mortality associated with smoke inhalation, and may contribute to resistant infections. Antibiotics should therefore be reserved for documented infections, and should be based on tracheal wash culture and sensitivity when possible.

Supplemental oxygen should be provided as needed. Carbon monoxide poisoning, if present, may be treated with hyperbaric oxygen therapy, but in most cases administration of 100% oxygen for 6 hours constitutes appropriate therapy without the increased risks and cost involved in transporting a critically ill patient to a facility with a hyperbaric oxygen chamber. Administration of 100% oxygen has been shown to shorten the half-life of carboxyhemoglobin from several hours to approximately 74 minutes (range 26 to 148 minutes).

Nutritional Support and the Hypermetabolic Response
Nutritional support is an important component of burn care, and should ideally be provided as soon after resuscitation as possible. Enteral nutrition using a nasogastric or esophagostomy tube is ideal, as this is believed to decrease gut atrophy, possibly decreasing bacterial translocation and subsequent sepsis. Resting energy requirements may be calculated using the formula [RER= Weight (kg) x 30 + 70]. Although the use of an illness energy requirement calculation (IER) has largely fallen by the wayside in veterinary medicine, multiplying resting energy requirements by an IER of 1.3-1.7 may be appropriate in the burned patient to compensate for the anticipated hypermetabolic response. Critically ill patients or those with very large burns may not tolerate their full nutritional requirements because of ileus or vomiting, and these patients may benefit from the supplementation of parenteral nutrition through a designated central line.

Pain Management
Pain can be reduced initially using cool compresses and soothing ointments such as silver sulfadiazine. Once burn shock has been adequately controlled, narcotics may be administered. Pure agonists such as fentanyl (CRI: 3-5 ug/kg/hr), hydromorphone (0.1-0.2 mg/kg IV q4h, or CRI: 0.025 mg/kg/hr), or morphine (0.5-1 mg/kg SQ q4h) are recommended for patients with moderate to severe pain. Ketamine can be useful for the relief of somatic pain, and may be used in conjunction with narcotics at a
constant rate infusion of 0.15-0.6 mg/kg/hr. Lidocaine may provide adjunctive analgesia in addition to free radical scavenging properties, and may also be added at a rate of 1.5-3 mg/kg/hr. If using constant rate infusions, a loading dose equal to the hourly rate should initially be administered.

References
Alternatives to Tooth Extraction
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Extractions are not the only option for treating dental injuries and disease

Fractured teeth:
- Complicated crown fractures (pulp exposure)
  - Treatment options
    - Root canal (referral)
    - Vital Pulp Therapy (referral)
      - Young dog <18 months of age
      - Fracture known to have occurred within the last 48 hours
  - Complicated crown fracture with open apex
    - Apexification
    - Surgical endodontics
  - Uncomplicated crown fracture
    - Fracture into enamel/dentin that does not expose the pulp
      - Radiograph for vitality (repeat in 6-12 months)
    - Contouring with diamond bur
      - Bond/seal with unfilled resin
    - Restore
      - Composite restoration to restore anatomy
      - Can be difficult to maintain

Caries
- Dogs can get true cavities, less common
- Typically occurs in bunodont teeth, (molar teeth with pits and fissures)
- Radiograph to evaluate for endodontic disease, treat if indicated
- Debride to healthy tissue
- Restore with multi-layer composite restoration

Abrasion
- Teeth wearing abnormally due to behavior, job (police dog)
  - Endodontic treatment if indicated
  - Crown preparation and placement
  - Non precious full metal jacket crowns most common
    - Tooth colored options not as strong

Crown lengthening procedures
- Type I and Type II
  - For increasing the height of a tooth to enable crown placement
    - Involved periodontal surgery
  - Post and core prosthetic crown

Discolored/non-vital teeth
- Pulpitis
  - Reversible vs. non-reversible
  - One study showed that 98% of teeth that were discolored were non-vital
  - Nidus for infection
  - Anachoretic infection (via the bloodstream)
  - If the patient is very young the pulpitis may be reversible and the tooth can be remain vital
    - Large blood supply, open apex
Non-vital teeth can be treated endodontically

Avulsed/luxated teeth
- Time sensitive- complete avulsion should be treated within 24 hours
  - Replace into socket via splinting
  - Semi-rigid fixation
  - Wire and acrylic appliance
- Root canal must be performed due to disruption of blood supply
  - Root canal performed at splint removal in 4-6 weeks

Treatment of malocclusions
- Interceptive orthodontics would involve strategic extraction of teeth to allow for functional bite.
- Passive or active appliances can be used to move teeth into more normal position to improve bite
  - Good for linguoverted mandibular canines

Crown reduction and vital pulp therapy/endodontic therapy
- Alternative to extraction
- Better for mandibular canine teeth
  - Large teeth, support of mandible compromised by extraction
  - Young dogs with linguoverted mandibular canine teeth

Periodontal disease
- Not all teeth affected by periodontal disease require extraction
- Normal gingival sulcus depth = 2-3 mm in a dog, 1 mm in a cat
- Greater depth may indicate periodontal attachment loss

- Pockets < 5 mm
  - Closed root planing
  - Gracey curette for subgingival debridement
  - Debridement of epithelialized surface/pocket
  - Local antibiotic therapy
  - High concentration products that deliver sustained local release
  - Encourage reattachment of periodontal tissues (periodontal ligament, cementum)

- Pockets > 5mm
  - Open root planing
    - Curettes and diamond burs for debridement and bone contouring
    - Requires flap development
    - Local antibiotic therapy
    - Bone augmentation
  - Guided Tissue Regeneration
    - Membrane placement
    - Orderly regeneration of periodontal tissues
    - Periodontal ligament
    - Bone
- Without proper technique the pocket will re-epithelialize and not resolve
- Without membrane bone may be first to reattach and will result in ankylosis

- Flap procedures
  - Used for severe periodontal tissue loss, with or without a membrane or bone augmentation
  - Debridement of tissues
  - Smooth root surface and tapered/feathered alveolar bone
  - Apically repositioned flap
    - Significant bone loss unable to be regenerated
- Mandibular incisor teeth
  - Coronally repositioned flap
  - Laterally repositioned flap

- Implants
  - Being performed in dogs
  - Canines and carnassial teeth, incisors
  - Success rate highly variable
  - Hygiene concerns
Oral/Maxillofacial Emergencies
• Fortunately, not very many!
  Time Dependent:
  • Acute tooth fracture in an animal < 18 months of age
  • Tooth avulsion or luxation
  Not time dependent or stabilize first:
  • Chronic tooth fracture
  • Acute tooth fracture in an animal > 18 months of age
  • Jaw fractures
  • Temporomandibular joint luxations

Acute Tooth Fracture in an Animal < 18 months of age
• Root may not be completely formed
• Apex open
• Tooth is inherently weak
• Little secondary dentin deposition
• Tooth will be more functional and stronger if pulp/blood supply can be kept vital

Vital Pulp Therapy
• Must be performed within 48 hours of pulp exposure
• Performed using aseptic technique
• Works best in young animals with large pulp/blood supply
• Only procedure that can keep the tooth “alive” to continue developing normally

Vital Pulp Therapy Procedure
• Remove inflamed/contaminated pulp
• Place medicament to encourage pulp healing
• Seal pulp chamber from oral cavity
• Recheck radiographs at defined intervals

Tooth Avulsion or Luxation
• Removal of the tooth from the socket
• Generally occurs as a result of trauma
• Dog fight
• Vehicular trauma
• Nose stuck through fence
• Avulsion = complete removal of the tooth from the socket
• Luxation = displacement of the tooth within the socket

Tooth avulsion is a true emergency
• Replacing the tooth within the socket as soon as possible is very important
  • “Golden Hour”
• Tooth should be placed in milk as a transport medium
• Not in water
• Do not clean or debride tooth
• Once tooth is replaced it is held in place with a splint
• The tooth will reattach to the socket and be functional
• Because the blood supply to the tooth has been disrupted, the pulp will become nonvital
• Complete dentition in a show dog is important for most breeds
• Replacement of a tooth avulsed by trauma would not be considered unethical alteration of a show animal
Maxillofacial Trauma

Causes:
• Vehicular trauma
• Dog fights/cat fights
• High rise falls
• Pathologic fractures
• Numerous others

Maxillofacial Trauma
• First and foremost - assess and stabilize the patient
• Severe trauma is likely to have occurred to cause injuries to the bones of the skull
• Assess patient for respiratory status, shock, or neurologic damage
• ABC’s
• Chest radiographs
• Serial neurologic examinations
• Repair of fractures/luxations can be delayed until patient is completely stable

Maxillofacial Trauma
• Diagnosis/Presentation
• History of trauma
• Obvious malocclusion
• Anorexia/dysphagia
• Ptyalism

Diagnostic tests
• Radiographs
• Sedated oral exam
• CT/MRI

Maxillofacial Fracture Repair Principles
• Most important goal is to achieve a functional occlusion
• Occlusion must be checked throughout the procedure and during healing process
• Avoid damage to important structures
• Don’t forget about the teeth!!!
• Major arteries, veins, and nerves
• Repair techniques must be tailored to the individual patient

Maxillofacial Fracture Repair Principles
Muzzle stabilization
• Can be useful in addition to other fixation techniques
• Muzzle as only method of stabilization will generally result in severe malocclusion
IM pinning
• Complete destruction of mandibular artery, vein, and nerve
• Severe damage to multiple tooth roots
External fixation
• Can be a useful method of repair for bilateral mandibular fractures
• Requires careful placement of pins to avoid damage to tooth roots or other vital structures
• Other techniques are preferred

Special Considerations
How will you determine if occlusion is normal during fracture repair?
• Intubate via pharyngotomy or tracheotomy
• Will the animal have a functional mouth once the fractures have healed?
• Recheck weekly or bi-weekly
How will the animal eat during the healing process?
• Attempt repair techniques that will create a functional mouth immediately post-op
• Feeding tubes
• Nasogastric tube
• Esophageal tube
• Gastrostomy tube

Maxillofacial Fracture Repair
• Using the teeth as anchors, stainless steel wires can be used to stabilize fractures by wiring teeth together on either side of the fracture
• Application of cold curing acrylic can significantly strengthen wire repairs
• No two cases are ever exactly the same
• Creative use of multiple techniques based on the unique characteristics of the fracture and the patient are often successful

Interdental Wiring
• Multiple techniques
  • Ivy loop
  • Stout loop
  • Risdon
  • Essig
Dental Radiography Interpretation
Kendall Taney, DVM, DAVDC, FAVD
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This lecture will briefly review basics of dental radiography and then focus on images for interpretation and discussion.

The importance of radiographs when treating dental disease cannot be stressed enough! Dental radiographs are vital for complete evaluation of the teeth. Up to 70% of the tooth is not visible without radiographs. Certain pathologies cannot be identified or successfully treated without dental radiographs.

Many things can be identified through dental radiographs:
- Bone loss from periodontal disease
- Periapical abscesses
- Resorptive lesions
- Root fractures
- Identification of impacted teeth
- Evaluation of endodontic or restorative procedures
- Detection of oral cancer

Tools for Dental Radiography
- Digital systems
  - Direct digital radiography (DR)
    - Digital sensor with software - hardwired
    - Sensor is most commonly size 2, some available with size 1
    - Dedicated computer
    - Dental x-ray generator
  - Indirect digital radiography (Computed radiography - CR)
    - Phosphor plates
    - Variable sizes from 1 to 4
    - Plate processor
    - Converts image to digital
    - Dedicated computer
    - Dental x-ray generator

Personal Safety
- Dental x-ray machines produce less radiation than standard machines
- Use of a digital system reduces the radiation even further
- 6 feet away from machine is standard
- Do not stand in front or behind the primary beam
- Do not hold films in the patient's mouth
- Use radiation exposure badges

Direct vs. Indirect Digital Dental Radiography
Direct digital system advantages (DR)
- Extremely fast image production
- Quick retakes without having to reposition patient
- Image enhancement/better detail

Direct digital system disadvantages
- One sensor size
- Inflexible sensor
- Expensive

Computed radiography (CR) system advantages
- Variable phosphor plate size
Flexible films, easier to place in mouth
– Inexpensive (phosphor plates)

**CR system disadvantages**
– Processing system slightly longer
– Higher probability of user error

**Taking Dental Radiographs**
Without proper technique, results will be poor and inconsistent. This often results in the lack of desire or confidence to routinely perform dental radiographs and therefore lesions are missed. Developing a sound technique and routine when taking dental radiographs is key to consistency and successful film taking.

**Basic Positioning Techniques**

**Parallel Technique**
– Film and long axis of the tooth are parallel to each other and the primary beam is directed perpendicular to these

**Bisecting Angle Technique**
– Angle between the film and long axis of the tooth is bisected, and the primary beam is directed perpendicular to this imaginary line

**Oral Anatomy Considerations**
Dogs and cats have a relatively flat hard palate
This makes it impossible to use a parallel technique to image teeth in the maxilla
Other areas of the mouth cannot be imaged with the parallel technique
– Rostral mandible

**Feline Radiographs**
Cats have 30 teeth
Full mouth feline radiographs can be taken in six views
Full mouth radiographs are important in any case with suspected tooth resorption

**Canine Radiographs**
Dogs have 42 teeth
Number of films for full mouth radiographs will depend on the size of the dog
Additional views are sometimes needed of multi-rooted teeth
– Endodontic treatment
– Lesion evaluation

Dental radiograph interpretation and discussion
Dentistry Tips for General Practice
Kendall Taney, DVM, DAVDC, FAVD
Center for Veterinary Dentistry & Oral Surgery
Gaithersburg, MD

Getting a Dental Program Started
- Check AAHA dental guidelines
- First item on the list of necessary equipment:
  - Dental radiography
  - Initial investment may seem steep, but with an established dental program the return will be swift
- Helping Clients Understand
  - Keep them informed throughout the process
  - Help to allay fears about anesthesia
  - Make correlations they can understand
- Provide a valuable service
  - Follow up after the procedure
  - Start with an awake examination during the initial visit
    - Have the owner look with you in the mouth
    - Explain your initial findings
    - Use periodontal disease charts and tooth models
  - Provide a detailed estimate with a range of costs
  - Explain that the plan may change with evaluation under anesthesia
- Awake Oral Examination
  - From AAHA Dental Guidelines:
    "Explain the two-part process involved in a diagnostic dental cleaning and patient evaluation to the client. It is critical that he/she understand the hospital protocol to minimize miscommunication and frustration. The procedure involves both an awake component and an anesthetized component for a complete evaluation. It is not until the oral radiographs have been evaluated that a full treatment plan including costs of the anticipated procedure(s) can be successfully made with any degree of accuracy."
- Client Fear of Anesthesia
  - Informed Consent
  - Clients feel better when they know what to expect for their pet’s procedure
  - Explain the anesthetic protocol
  - Discuss the expected procedures to be performed
  - Flaps and tooth removal

Get a contact number where the client can always be reached
- One of the top reasons for malpractice complaints is unauthorized tooth extraction
- Have the client sign a consent form stating the risks involved
- A line can be included that states necessary procedures will be performed in the event that the client cannot be reached while the pet is under anesthesia
- Don’t depend on this!
- Stress the importance of being able to reach the client at all times

Evaluation of the patient under anesthesia
- Under Anesthesia
  - Complete examination with probing and charting
  - Dental radiographs
    - Full mouth or focal
  - Pictures of the teeth and oral cavity before procedures are performed
  - Update the estimate and call the client to discuss
Document the conversation in the record
Include topics discussed such as teeth to be extracted and expected costs, record time

What Should You Charge?
- Extractions
  - Different charges for different teeth
  - Different levels of difficulty
- Radiographs
  - One charge for full mouth
  - Individual per film
- Flaps
- Anesthesia Time
- Materials
- Bone augmentation
- Local antibiotic gel treatments

Performing Successful Dental Procedures

Proper equipment is essential
- Tools for suturing
- Periosteal elevators
- Winged Dental Elevators
- High speed handpieces and burs
  - Larger round burs for bone removal (#2 or #4)
  - Small round burs for severing the periodontal ligament and creating a space for the elevator to begin luxation of the tooth/root (#1/4 or #1/2)

Extraction tips
- Get in the habit of creating mucoperiosteal flaps
  - Provides adequate exposure for bone removal
  - Allows for closure of extraction site
- Section multi-rooted teeth
  - 701L cutting bur
- Remove 1/3 to ½ of the buccal alveolar bone with a round bur
- Sever periodontal ligament with ¼ round bur
  - Removes minimal bone but allows space for elevator
- Slow deliberate elevation of tooth roots
  - Turn and hold for 10 seconds
  - Continue until periodontal ligament is fatigued and root can be extracted
- Post extraction radiographs to confirm complete root removal and intact jaws
  - Pathologic fractures
- Release the tension on the flap to allow for adequate closure
  - Periosteal release
- Closure with absorbable suture of choice
  - SH or SH-1 needle
  - 4-0 chromic gut, monocryl

Avoiding complications related to extractions
- Radiographs Convey the Extent of Disease
  - Dilacerated Roots
  - Risk of pathologic fracture
- Bone removal should be the right balance of just enough for exposure and elevation
- Excessive bone removal can lead to jaw fracture or pushing a root into the nasal cavity or mandibular canal
  - What do I do if I pushed a root into no man’s land?
    - Don’t panic
    - Inform the owner of the complication
- Refer if not comfortable retrieving the root tip
  - Be aware of neurovascular structures
    - Infraorbital vessels exit the foramen below the maxillary PM3 and PM4 teeth
    - Mandibular canal - roots of mandibular M1 are very close and sometimes within the canal
    - Mental foramen
  - Avoid levering against healthy teeth
    - Can break adjacent healthy teeth with too much force
  - Common areas for pathologic fractures
    - Parasymphyseal fractures from attempted extraction of the mandibular canine tooth
    - Mandibular body fractures during extraction of the 1st molar tooth

**Tooth Resorption in cats**

True or False: If I see a resorptive lesion I can just perform a crown amputation and the remainder of the tooth and roots will dissolve and never be a problem

**Tooth resorption cannot be appropriately treated without dental radiographs**

- Evaluation of periodontal ligament
- Ability to distinguish root/cementum from alveolar bone
- Crown Amputation vs. Extraction
  - Periodontal ligament is not visible
  - Difficult to distinguish between root and alveolar bone
  - Note that crown amputation is generally only appropriate in late stages of resorption

**Tooth Resorption in Dogs**

- In dogs, there can be an exception to the rule of extraction for tooth resorption
- Radiographs may reveal many teeth with evidence of root resorption
- If a lesion is not grossly visible or palpable with an explorer, it can be appropriate to leave the tooth and monitor for changes

**Other Feline Dental Issues**

- Chipped Canine Teeth
  - Pulp chamber is located very close to surface of the crown
  - Any chip fracture in a cat can potentially involve pulp exposure
  - Root canals can also be done in cats
- Maxillary Canine Extraction
  - Extraction of maxillary canine tooth in the cat can have certain consequences especially if ipsilateral mandibular canine is still present
  - Lip ulceration
  - Inability to completely close the mouth or elevation of the upper lip
- Buccal alveolar bone expansion causing difficult flap creation and closure
- Maxillary Canine Extraction
  - Difficult closure due to alveolar bone expansion
- Extraction Complications
  - Iatrogenic jaw fracture
  - Tooth resorption/ankylosis
  - Bone loss from periodontal disease
- Stomatitis
  - Inflammation of the oral cavity
  - Full mouth extractions offers possible cure
  - Surgical extractions in an inflamed environment can be difficult
  - Extremely friable tissue
  - Excessive hemorrhage

**Discharging Dental Patients**

- Detailed discharge Instructions
  - Medication
  - Feeding
  - Home dental care
Before and after photographs and radiographs
Give information on what to expect
  Behavior after anesthesia
  Bleeding/pain
  What to do in an emergency, who to call with questions
Make future plans
  Recheck
  Routine dental care plan

Keep Your Clients and Patients Happy
  Call that evening and the next day to check on the patient
  Be available for questions
  Intervene early if complications arise
  Being thorough with the owner will understand the value even with a higher bill

Resources
American Veterinary Dental College
www.avdc.org
www.avdc.org/nomenclature.html
American Veterinary Dental Society
Journal of Veterinary Dentistry
www.avds-online.org
The etiology of stomatitis remains elusive…..
Multiple theories exist:
  o Sensitivity to plaque/overzealous immune response
  o Relation to feline calicivirus
  o Bartonella
  o FELV/FIV/Herpes virus

While many cats with stomatitis test positive for shedding of feline calicivirus, no single agent bacterial or viral has been shown to have a direct correlation to development of stomatitis

Early onset (juvenile) gingivitis
  o Initially localized to gingiva
  o Can progress to more severe inflammation/chronic stomatitis
  o Chronic inflammation puts pressure on the alveolar bone and erodes the periodontal support structures
  o Teeth become diseased overtime necessitating extraction due to periodontal disease

Tooth resorption may or may not be present, no correlation

Plaque control remains a cornerstone of treatment
No teeth = no plaque

Initial intervention:
Can try professional cleanings every 6 months
  • Plaque will be back on teeth within hours
Home dental care
  • Daily brushing
  • Oral rinses
  • Patients may not be amenable to home dental care due to discomfort

Surgical treatment:
  • Full mouth extractions
    o Every tooth and every root must be confirmed to be removed radiographically
  • Partial mouth extractions
    o Shifting of inflammation
    o Poor resolution of caudal stomatitis

Medical management
  • Results will be unpredictable with teeth still present
  • Consider reserving medical management for patients with refractory stomatitis after full mouth extractions
    o Steroids
    ▪ Immunosuppressive to lowest effective dose
    o Cyclosporine protocol
    ▪ Should not be given to immunocompromised patients
      • Perform pretreatment CBC and renal function tests
      • Cyclosporin A recommended, bioavailability differs among products
      • Begin at 2–2.5 mg/kg q12h
• Retest renal and CBC values at 3 weeks
• Test blood cyclosporine levels at 6–8 weeks to ensure minimum value of 300 ng/mL
• If blood value is too low, adjust dose to as much as 5 mg/kg q12h
  o Low-dose doxycycline protocol
    • 2 mg/kg PO BID for 21 days, then 1 mg/kg BID indefinitely
    • Anti-inflammatory effects at a lower dose
  o Omega interferon
    • Difficult to obtain in US, variable results
  o Stem cells
    ▪ Variable results
    ▪ Technique sensitive
• Reduce allergic response
  o Recommend hypoallergenic diet, replace plastic food bowls with stainless or ceramic, ensure parasite control is adequate

Other therapies
• CO2 laser ablation
  o Reserved for refractory cases
  o Success rate variable
• Therapeutic “cold” laser

Anecdotal observations
• Increased likelihood of refractory stomatitis
  o Chronic cases
  o Severe proliferative cases
    ▪ Caudal stomatitis
    ▪ Lingual proliferation
  o Clinical symptoms at presentation
    ▪ Pain when eating
    ▪ Poor grooming
    ▪ Weight loss
  o Partial mouth extractions
  o Incomplete root removal
  o Concurrent viral infection
• Increased likelihood of success
  o Inflammation limited to paradental tissues
  o Young patients
  o No clinical signs
• It can be difficult for owners to accept that their pet will have a good quality of life without teeth
  o Counsel owners that pets do not need their teeth to survive/be happy
    ▪ Not catching their dinner
    ▪ Not defending their territory
  o Patients will eat hard food even with no teeth
  o Full mouth extractions can be curative for stomatitis, ultimate goal is resolution of inflammation/clinical signs that does not require chronic medical management
  o If signs do not completely resolve most cases will have improvement and clinical signs can be kept under control with medication
    ▪ Don’t treat the redness, treat the patient

CUPS in dogs
 o Chronic ulcerative paradental stomatitis
 o Similar presentation to cats
 o Ulcerative “kissing” lesions where mucosa contacts the teeth
Treatment via full mouth extractions in severe cases
  - LWD, schnauzers prone to developing

Recent studies on the effectiveness of extractions on resolution of feline gingivostomatitis:
Tooth Resorption
Kendall Taney, DVM, DAVDC, FAVD
Center for Veterinary Dentistry & Oral Surgery
Gaithersburg, MD

Feline teeth have unique anatomy that must be considered. Cats are more prone to disease processes that make extractions difficult such as tooth resorptive lesions and stomatitis. The importance of radiographs when treating feline dental disease cannot be stressed enough! Dental radiographs are vital for complete evaluation of the teeth. Up to 70% of the tooth is not visible without radiographs. Certain pathologies cannot be identified or successfully treated without dental radiographs.

Many things can be identified through dental radiographs:
- Bone loss from periodontal disease
- Periapical abscesses
- Resorptive lesions
- Root fractures
- Identification of impacted teeth
- Evaluation of endodontic or restorative procedures
- Detection of oral cancer

Tools for Dental Radiography
- Digital systems
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Taking Dental Radiographs
Without proper technique, results will be poor and inconsistent. This often results in the lack of desire or confidence to routinely perform dental radiographs and therefore lesions are missed. Developing a sound technique and routine when taking dental radiographs is key to consistency and successful film taking.

Basic Positioning Techniques
Parallel Technique
- Film and long axis of the tooth are parallel to each other and the primary beam is directed perpendicular to these

Bisecting Angle Technique
- Angle between the film and long axis of the tooth is bisected, and the primary beam is directed perpendicular to this imaginary line

Oral Anatomy Considerations
Dogs and cats have a relatively flat hard palate
This makes it impossible to use a parallel technique to image teeth in the maxilla
Other areas of the mouth cannot be imaged with the parallel technique
- Rostral mandible
Feline Radiographs
Cats have 30 teeth, dogs have 42
Full mouth feline radiographs can be taken in six views
Full mouth radiographs are important in any case with suspected tooth resorption

Tooth Resorption- go to www.avdc.org/nomenclature for images and definitions
Known aliases:
– Feline dental resorptive lesions (FDRL)
– Feline odontoclastic resorptive lesion (FORL)
– Neck lesions
– Cervical line lesions
Can occur in cats and dogs

Stage 1 (TR 1): Mild dental hard tissue loss (cementum or cementum and enamel).
- Very difficult to visualize grossly or radiographically
- May be able to identify by tactile probing with shepherd’s hook
Stage 2 (TR 2): Moderate dental hard tissue loss (cementum or cementum and enamel with loss of dentin that does not extend to the pulp cavity).
Stage 3 (TR 3): Deep dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth retains its integrity.
Stage 4 (TR 4): Extensive dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth has lost its integrity.
  TR4a Crown and root are equally affected;
  TR4b Crown is more severely affected than the root;
  TR4c Root is more severely affected than the crown.
Stage 5 (TR 5): Remnants of dental hard tissue are visible only as irregular radiopacities, and gingival covering is complete.

Crown Amputation vs. Extraction
When is it appropriate to use crown amputation or intentional root retention as a treatment for tooth resorption?
The only way to know is with dental radiography
  – Evaluation of periodontal ligament
  – Ability to distinguish root/cementum from alveolar bone
Same approach is made for either
  – Flap for surgical extraction
  – Attempt to perform standard surgical extraction
  – Closure of flap after extraction or alveoloplasty

Crown amputation requirements:
  o Periodontal ligament is not visible
  o Difficult to distinguish between root and alveolar bone
  o Note that crown amputation is generally only appropriate in late stages of resorption

Extraction is indicated or should be attempted:
  o Periodontal ligament is visible
  o Distinct demarcation between root and alveolar bone
  o The decision is not always straightforward!
  o Two different techniques may be utilized in the same tooth
  o Always attempt standard extraction techniques

Tooth Resorption in Dogs
  o In dogs, there can be an exception to the rule of extraction for tooth resorption
  o Radiographs may reveal many teeth with evidence of root resorption
  o If a lesion is not grossly visible or palpable with an explorer, it can be appropriate to leave the tooth and monitor for changes
Other Feline dentistry topics
Chipped Teeth
- Pulp chamber is located very close to surface of the crown
- Any chip fracture in a cat can potentially involve pulp exposure
- Watching and waiting not a good option
- Root canals can also be done in cats
Maxillary Canine Extraction
- Extraction of maxillary canine tooth in the cat can have certain consequences especially if ipsilateral mandibular canine is still present
  - Lip ulceration
  - Inability to completely close the mouth or elevation of the upper lip
  - Buccal alveolar bone expansion causing difficult flap creation and closure
Oronasal fistula
- Maxillary Canine Extraction without proper flap development, bone removal, and closure
Extraction Complications
- Iatrogenic jaw fracture
  - Tooth resorption/ankylosis
  - Bone loss from periodontal disease
Stomatitis
- Inflammation of the oral cavity
- Full mouth extractions often only possible cure
- Surgical extractions in an inflamed environment can be difficult
  - Extremely friable tissue
  - Excessive hemorrhage

Dental radiographs are the key to identifying pathology and developing treatment plans. Certain disease processes common in cats can make extractions challenging. Using radiographs and being aware of potentially negative scenarios can greatly reduce complications
Canine Allergy: What to do if the newest drug doesn’t work!

Darin Dell, DVM, DACVD

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We will start with a basic overview of atopic dermatitis. Atopic dermatitis is a life-long condition which usually requires life-long therapy. Achieving a true clinical cure for allergic dermatitis is possible but rare. Atopic dermatitis typically manifests as pruritus and erythema. However, some animals may develop recurrent pyoderma or otitis externa instead. While it is difficult to estimate, the current assessment is that 10-15% of the canine population suffers from atopic dermatitis. (Many suspect the number is considerably higher.) In technical terms, atopic dermatitis is a genetic predisposition to hyper-react to allergens in the environment. Unlike what many of us were taught in Veterinary School, allergen exposure occurs mainly through the skin. True inhalant (only) allergic dermatitis is rare. Respiratory symptoms that occur along with allergic dermatitis may represent irritation caused by debris rather than a true allergic process. Allergen exposure is enhanced by defects in the epidermal barrier. One of the many functions of the epidermis is to “keep the outside out and the inside in.” Animals that are genetically predisposed to allergic dermatitis have genetically programmed defects in their epidermal barrier. These defects result in increased allergen exposure. It is important to realize, and communicate, that allergic inflammation causes more than just the itching we can observe in the exam room. Allergies increase transepidermal water loss, dermal and epidermal inflammation, and the risk of secondary infection.

The first step of treating an allergy patient is achieving an appropriate diagnosis. This begins with a good physical exam. Many allergy patients demonstrate erythema on the ventral aspects of the front paws and in the ear canals. Typically, the pinnae themselves are not primarily affected. Pinnal lesions should raise concern for Sarcoptes scabiei and extension of infection from otitis externa. Dogs with environmental allergies are also known for not having lesions on their dorsal lumbar area. This is a location more commonly associated with flea allergy dermatitis. Superficial pyoderma, malassezia dermatitis, alopecia, and seborrhea are common with all types of allergy so observing these symptoms should trigger you to look at common allergy affected areas. Of course, the other crucial part of examining a dog is questioning the owner. Important allergy related questions to ask are: “At what age did your dog start having skin/ear problems?” “When did this episode begin?” “Have you observed any change with the weather or seasons?” “Do you know anything about your dog's parents or siblings?” “Are there any other pets in the house?” “If so, are the other pets affected?” The last part of a good allergy exam is client education. Education is critical because allergies are chronic and frustrating. It is best to have educational information available in multiple formats. Paper hand-outs, informational emails, in-office videos, and internet resources are all readily available.

Now that you have collected a good history and performed a thorough physical exam the next step is working down the diagnostic pathway of allergic dermatitis. Unfortunately, there is not one single test that can diagnose allergies. Atopic dermatitis is a diagnosis of exclusion. This often needs to be explained to our clients. Proper diagnosis of atopy requires appropriate history, consistent clinical signs, and proof that the pruritus and skin disease are not caused by infections, parasites, metabolic disease, and endocrine disease. Cytology from the ears and skin is the first step. Cytology is almost always indicated. Cytology allows you to quickly identify yeast and bacteria and provides a semi-quantitative method of monitoring progress. A dry microscope slide can be pressed on moist lesions, scraped under crusts, or used to break pustules. Another useful method of collecting cytology is with clear packing tape. Packing tape is most helpful for dry lesions, folds, and nail beds. When using tape you do not need to “heat fix” the sample or use the fixative step of your three step staining protocol. Skin scrape sample collection is often needed but not as frequently as cytology. We all know the basics of skin scraping. But here are a few tips to improve your success. First, shave the area you intend to scrape. Second, apply mineral oil to the sample site and pinch the area firmly. Third, scrape until you obtain capillary bleeding.
You should observe red blood cells on the slide when you look at it under the microscope. Lastly, it helps to have mineral oil waiting on your slide so that all the debris you remove from the skin surface stays where you can view it. The last of our skin related tests is DTM culture. DTM cultures can be particularly frustrating for veterinarians and technicians. Idexx now offers a dermatophyte PCR which can provide results in 1-3 days. You need the same type of sample for PCR as for culture. When collecting your samples for DTM culture it is best to collect material from the edge of lesions. Broken hairs are especially helpful. It is also useful to use a fresh tooth brush to pick up dander, debris, and hair from the entire surface of the animal. If you choose to perform DTM culture in house please remember: 1) Use plate type media not jars or test tubes. 2) Do not close the culture tightly. 3) Keep the culture in a dark area with approximately 30% humidity and at 86 degrees fahrenheit. 4) Color change does not confirm diagnosis of a dermatophyte. Many contaminants can cause the medium to change from orange to red so microscopic examination of the fungal growth is essential to confirm dermatophytosis.

The one very obvious diagnostic that we have not discussed yet is allergy testing. First and foremost, allergy testing is not a tool to diagnose allergies. In other words, it is not a screening tool. Rather, allergy testing is used to define the allergy more precisely, predict flares, direct environmental modification, and formulate immunotherapy. Allergy testing is not a screening tool because positive results don’t immediately prove that allergy is the cause of the skin symptoms. Rather, you must have a supportive history and clinical signs along with evidence that you have eliminated other causes of skin disease. There are two accepted and peer-reviewed methods of allergy testing: serum testing and intradermal testing. Serum testing only requires the collection of a blood sample. This type of test is quick and easy for the general practitioner and does not require any special equipment. Serum allergy testing is generally touted as not being affected by drug therapy such as steroids or antihistamines. However, these drugs can contribute to poor results in some dogs. In addition, serum allergy test results will be affected by season of the year. Within the past ten years numerous companies have begun offering serum allergy testing. I caution you not to choose an allergy testing company solely on cost. While all serum allergy companies use a similar testing model there are unique differences that can be quite important. Lastly, a paper published last year highlighted the difficulty with this testing method by sending samples from the same patient to multiple labs. Agreement between the labs was very poor. Intradermal allergy testing is typically only performed by veterinary dermatologists because of the need to keep expensive antigens in stock for testing and because of the learning curve necessary to read an intradermal allergy test accurately. Intradermal allergy testing also requires sedating the pet and shaving a patch of hair on the side of the thorax. Perhaps the most confusing factor in recommending intradermal allergy testing is knowing the drug withdrawal times required prior to the test. In general, the withdrawal time for oral steroids and antihistamines is two weeks. For injectable steroids like triamcinolone or dexamethasone the withdrawal time is 2-3 weeks. For Depo-Medrol, the withdrawal time is three months. Topical steroids should be stopped 48 hours prior to the test. Fortunately, there is NO withdrawal time for Atopica, Apoquel, or Cytopoint. There are many unique benefits to intradermal allergy testing. First, this test is not affected by season. Second, it allows a veterinarian to test the organ affected and observe the true intensity of an allergic reaction. Lastly, known positive and negative reactions are designed into every skin test. This helps us adjust for inevitable patient to patient variation.

Before you can develop a good allergy treatment plan you must realize that no single therapy is 100% effective. It is also important to understand that no two patients are exactly the same and that it is easier to prevent rather than suppress flares. Multimodal therapy is recommended because it allows intervention of allergic inflammation at multiple points in the disease process. It is easier to think of allergy therapy as core and supportive treatments. Most patients need a core therapy and one or two supportive therapies. However, severe patients need multiple core therapies and supportive therapies.

For ease of discussion we will consider six core allergy therapies: 1) antihistamines, 2) steroids, 3) Atopica, 4) Apoquel, 5) Cytopoint, and 6) Immunotherapy. We will focus on Immunotherapy, Atopica, Cytopoint, and Apoquel today. Immunotherapy is still considered the “gold standard” of allergy therapy. Immunotherapy allows us to modulate the allergic response without drugs. This occurs via multiple
mechanisms including the development of IgG blocking antibodies, a decrease in allergen specific IgE and an increase in the number of regulatory T cells. Consequently, immunotherapy provides many unique benefits that drug therapy cannot. Immunotherapy may also prevent new allergies from developing and is the only therapy that could potentially result in a clinical cure. Because immunotherapy is not a drug there are no major side effects or drug interactions. Anaphylaxis can occur during immunotherapy but this is rare. Immunotherapy is tailored to each individual so animals at higher risk for anaphylaxis can be induced more gradually. Risk for anaphylaxis is based on breed and the intensity of the allergy test reactions. Immunotherapy has classically been administered as subcutaneous injections. However, within the past three years, sublingual immunotherapy drops have become available for pets. Both routes of administration can be effective. Early publications suggested that oral immunotherapy would be more efficacious but my experience has been that the two forms are equally successful. Multiple schedules for administering these products are available based on the laboratory used and the dermatologist involved. When discussing immunotherapy with clients it is important to clearly communicate that immunotherapy is not a fast acting treatment with many dogs not showing significant benefit for 6-12 months. As a general rule, animals should receive immunotherapy for at least a year before deciding whether it is effective and worth continuing. Because of the slow onset, many patients need additional therapy in the beginning. This might include antihistamines, steroids, Atopica, Apoquel, or Cytopoint. While immunotherapy can provide a clinical cure, it is rare and most dogs require immunotherapy for life. As a general rule, immunotherapy is considered approximately 70% successful with 45-50% of those dogs requiring some type of additional supportive therapy long term.

Fortunately, we have three safe and effective medication options for treating allergy symptoms. Atopica (modified cyclosporine) became available commercially for dogs more than ten years ago. Atopica works via suppression of IL-2, T-helper, and T-suppressor cells. By far the most common side effects of Atopica are vomiting and diarrhea. Usually these are mild and do not require specific therapy or cessation of therapy. Another side effect that sometimes occurs is gingival hyperplasia. It is typically seen only in patients receiving high doses of cyclosporine or after many years of therapy. In most cases gingival hyperplasia resolves when Atopica is discontinued. Atopica is a very useful drug but there are a few items to keep in mind. First, because Atopica may take 4-6 weeks to see full effect it is not helpful for immediate control of flares. I typically recommend a 30 day recheck so that I can evaluate the patient’s progress. To help prevent vomiting you can freeze the capsules, give the medication with a small meal, divide the dose throughout the day or start with a low dose and ramp up to your target dose over two weeks. Lastly, you will commonly want to combine Atopica with a steroid during the first two to three weeks of treatment. The steroid provides immediate relief while the Atopica ramps up.

Apoquel was released in January 2014 and has become very popular because of its ability to provide quick relief and is usually very effective. Apoquel is a completely different medication than Atopica and it works via an extremely different mechanism. Apoquel (Oclacitinib) works via blocking IL-31 at the JAK-STAT pathway. IL-31 is the cytokine linked to the feeling of itch. By blocking IL-31 there is also suppression of Epithelial Langerhans cells and T-cells. Because of overlap in the Jak-Stat pathways Apoquel also suppresses IL-2, IL-4, IL-6, and IL-13 which are also involved in allergy. Apoquel’s serious side effects are linked to this overlap as well. The most concerning side effect to watch for is decreased hematopoiesis. One of the benefits of Apoquel is the speed on action. Most dogs will improve in 24-48 hours but I have had a few patients not respond until 5-7 days. Vomiting is far less common with Apoquel (as compared to Atopica) but it can occur and it can be severe.

The newest product is Cytopoint. During conditional release this product was referred to as Canine Atopic Dermatitis Immunotherapeutic. Cytopoint is a once a month injection of a monoclonal antibody designed to target IL-31. Side effects are extremely uncommon. This product can be given to puppies and dogs with other health problems. Some dogs will achieve more than one month of relief from a single Cytopoint injection. However, be watchful because the length of affect can vary based on the patient’s allergy season.
With all of the products available for treating atopy in dogs you might think it would be an easy task. However, every patient has different allergies, different primary signs, and different secondary problems. Consequently, you need to have a consistent treatment strategy.

Step one of this strategy is to eliminate current infections. Eliminating infections reduces pruritus and inflammation while also improving the patient's odor and appearance. The relief that a patient derives from resolving infections may be dramatic. This is also the time to impress upon the client the importance of secondary infections. In many cases our clients may ignore or be oblivious to the signs of infection. When present, infections can negate the improvement obtained by the actual allergy treatment. Another major problem of recurrent secondary infections is antibiotic resistance. For this reason it is imperative to prescribe an appropriate antibiotic for an appropriate length of time. It is also important to consider topical antimicrobial therapy. Topical therapy can provide immediate relief for the pet. More importantly topical therapy also works synergistically with the oral antibiotic / antifungal medication to reduce the risk of resistance. Of course, dealing with infections includes dealing with ear infections. Otoscopy is always a useful part of the allergy exam. Remember that otic cytology is important any time you suspect an ear infection. Cytology helps you determine which ears are inflamed due to allergy and which ears have infection. Cytology also helps you track the progress of your therapy. When managing otitis externa remember to choose both your ear wash and ear medication carefully.

Improving the epidermal barrier is step two. The epidermal barrier is composed of lipids and corneocytes in the stratum corneum. The dominant lipids in the stratum corneum are called ceramides. Free fatty acids and cholesterol are also found in the lipid portion of the stratum corneum. However, ceramides play a crucial role by helping align the other lipids. When intact, the lipid portion prevents water loss as well as allergen and antimicrobial penetration. Consequently, there is less allergen exposure, less risk of infection and less pruritus. Ceramides are now available in multiple forms. You will find ceramides in shampoo, sprays, conditioners, and spot-on products.

Conscientiously choosing a core treatment is step three. In order to make a good recommendation to your client you must consider the patient’s underlying medical conditions, the severity of the allergy, the primary symptoms, and the limitations of the dog and owner. You also want to steer your clients to the safest therapy for long term use. This entails considering Immunotherapy, Atopica, Apoquel, and Cytopoint.

Step four is adding supportive therapy as needed. What you add is based on what the patient requires and what the owner can reasonably accomplish. Supportive therapies include antibacterial and antipruritic shampoos, wipes, and sprays. Oral antihistamines, oral essential fatty acids, oral zinc supplementation, and PEA-um should be considered as well. Lastly, topical ceramides are available in shampoo, spray, and spot-on formulations.
Canine Otitis: Follow the Ear Wax (or the smell)

Darin Dell, DVM, DACVD.

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Patients with otitis externa generally present for head shaking, ear scratching, and odor from their ears. Pet owners may also notice purulent material coming from the ear canal(s), changes in their dog's behavior, or whining and discomfort. Otoscopic exam typically reveals varying degrees of erythema, edema and debris. These changes can make visualizing the tympanic membrane very difficult or even impossible. Unless the patient is aggressive/dangerous you should try your best to see every tympanic membrane that enters your exam room.

KEY POINTS

1) Otitis externa is most often a clinical sign of underlying skin disease, not a diagnosis in and of itself.
2) Identifying and resolving / controlling the underlying cause is essential to long term success in otitis externa.
3) Allergy is the most common cause of otitis externa in the canine
4) Topical therapy is the most effective therapy for treating otitis externa
5) EAR CPR! Cytology (clean) – Plan (persuade, purge) – Recheck (re-engage)

Identifying the underlying cause of otitis externa can be difficult. The PSPP system can help you work through the potential causes and also make it easier to discuss otitis with your clients. PSPP stands for Primary, Secondary, Predisposing, Perpetuating. The Primary category includes things that can cause disease in a normal ear. The list includes: allergy, auto-immune disease, foreign objects, mass/polyps, endocrine dysfunction, immune mediated disease, and parasites. The most common primary problem I see is allergy, but in general practice you probably see a fair amount of ear mites and foreign bodies too. Whatever the primary factor, it has to be resolved or controlled before you are going to achieve lasting success. The Secondary category includes things that create disease in an abnormal ear. The secondary list includes: bacteria, yeast (malassezia), fungi, medication reactions, and over-cleaning. Because of the way we commonly communicate about otitis externa it is easy for our clients to misunderstand and think that bacteria or yeast are primary causes of otitis. Sometimes it helps to point out that ear canals are not sterile. There is a normal flora in the ear canal just like on the skin. Infections must be resolved but they are not the root cause. Predisposing factors are fairly simple to understand and are typically what most clients blame for ear disease. Predisposing factors are present prior to otitis but cannot by themselves cause otitis. This list includes: conformation, excess moisture, obstruction, systemic disease, and treatment effects. Sometimes it helps to reassure our clients that there are Cocker Spaniels without ear disease despite their floppy ears and Poodles without ear disease despite their excess ear hair. The last category, Perpetuating, is the one most often neglected in veterinary practice. Perpetuating factors occur as a result of the otitis and increase the likelihood of another infection. These factors are: excess cerumen production, altered epithelial migration, edema of the ear canal, rupture of the tympanic membrane, and otitis media. I believe that the first three issues in the perpetuating category are most overlooked and least understood in private practice. Excess cerumen production occurs any time there is inflammation in the ear canal. The body’s response is to make more cerumen in an attempt to push out whatever is happening. Unfortunately, this excess cerumen can be a great growth medium for yeast and bacteria. Cerumen production can continue to be excessive for several weeks after the infectious component of the otitis initially resolves. For this reason, it is beneficial to continue ear
cleaning even after ear infection has resolved. Altered epithelial migration also develops during otitis externa. Normal otic epithelial migration starts at the tympanic membrane and marches distally out the aural orifice. This too is designed to help move debris out of the ear canals. However, inflammation within the canal disrupts this process resulting in build-up of debris in the canal. Altered epithelial migration is another reason why stenotic ears and cobblestone ears demonstrate wax build-up. Again, it is necessary to continue ear cleaning until this process is re-established. Edema in the ear canal is at least a problem you can see through the otoscope. But, the importance of edema is often underestimated. Edema will also trap cerumen which can potentially lead to a better environment for bacteria or yeast growth. Edema can also cause discomfort and pain which could result in ear pruritus and trauma.

Treatment for otitis externa starts with the PSPP system. In most cases, you can run through the PSPP list in your mind just like you would a check list for any other disease. Usually you can quickly rule out ear mites, foreign objects, and polyps. You might need to perform blood tests to look for endocrine disease if other suggestive signs are present. Similarly, you might need to perform skin biopsy to look for auto-immune or immune mediated disease if other supportive lesions are present. In the majority of cases you are not going to find any of the problems listed above in this paragraph. The majority of otitis externa in the canine is secondary to allergic dermatitis. In that case, the first question to ask yourself is whether the allergy is controlled or not. If the allergy is generally well controlled and the otitis externa is due to a flare or a dietary indiscretion then resolving the problem will be easier. Well controlled allergy patients may still have one or two episodes of otitis externa each year. If the allergy is unknown or untreated then you will have more work to do. Not the least of which will be convincing the owner that their dog has allergies. Still, you may choose to focus initially on the otitis and address the allergy in two to four weeks.

The otoscopic exam is extremely important. You need to assess pain, pruritus, edema, erythema, constriction, and exudate as well as the tympanic membrane. Next you will need to perform ear swab cytology. It is best to collect exudate from both the horizontal and vertical portions of the ear canal. Obviously, you are checking for Malassezia, coccoid bacteria and rod-shaped bacteria. But you are also looking for nuclear streaming, white blood cells, red blood cells, and evidence of biofilm. Bacterial culture from the ear canal may also be necessary depending on the situation. Ear cultures are not universally helpful for two reasons. First, you might culture normal flora. Second, MIC’s are usually based on serum levels of antibiotics. In the ear we are concerned about topical / direct exposure to the antibiotic. The essence is of the problem is that some antibiotics to which the bacteria are listed as “Resistant” will actually be “Sensitive.”

Now that you have performed an exam and evaluated cytology you have to choose a therapeutic plan. I want to stress that there isn’t one universal plan for otitis externa. We can’t group treatment into levels such as easy, moderate, and severe either. However, asking yourself the following six questions can help you make better treatment decisions.

1) Is there an allergy and are you treating it now?
2) How much debris is in the ear canal?
3) How is the conformation of the ear canal?
4) What type of infection is present?
5) How much edema and erythema are present?
6) How much pain and anxiety are present?

Now, in more detail:

1) Is there an allergy and are you treating it now? You may not treat allergy at the first visit for otitis externa. But, you should at least start the conversation about allergy.
2) **How much debris is in the ear canal?** This will help you decide what type of cleaner to use and how often. For thick sticky wax you will probably want a micellar solution or one with squalene. For mucoid exudate you will probably want a Triz EDTA product with Chlorhexidine.

3) **How is the conformation of the ear canal?** Is it constricted? Cobblestoned? This too will help you decide what type of ear wash to use and whether to use a topical medication that is a gel, ointment, or liquid. The more the canal is constricted the more you need a wash that is better at dissolving cerumen. Ointments are less likely to travel deep into a constricted or cobblestoned ear canal so you probably want a liquid medication.

4) **What type of infection is present?** This will help you pick a topical treatment. The side note is that YOU have to know what drugs are in the products on your shelf. Infection with rod shaped bacteria will also encourage you to use an ear wash with Triz EDTA. Most rod shaped bacteria are gram negative. Triz EDTA damages the gram negative membrane and forms channels which allow antimicrobials into the bacteria.

5) **How much edema and erythema are present?** This will tell you what strength of steroid to use. Topical steroid therapy may be sufficient or you might need oral steroid therapy as well. If the ear canals are completely constricted then you will definitely need help from an oral steroid. Again, you have to know what ingredients are in the products on your shelf! Common steroid ingredients in otic medications, in order of potency are as follows:
   a. Prednisolone
   b. Betamethasone
   c. Mometazone

6) **How much pain and anxiety are present?** This will tell you if you need to prescribe additional pain relief or anti-anxiety medications. These medications are short term but can really help both the dog and the owner. This might require a prescription of Tramadol, Rimadyl or Xanax. Don’t underestimate the pain or anxiety related to ear infection! How many clients have told you that their dog runs away when they see the ear wash bottle or tube or ear ointment?
FOOD ALLERGY TRUTH AND LIES
Darin Dell, DVM, DACVD
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Cutaneous adverse food reaction

- Clinical signs
  o Classically “ears and rears”
    ▪ Recurrent otitis externa
    ▪ Scooting or peri-anal licking
  o Some also paw at their muzzle
  o Distal limbs also commonly affected.
  o May be pruritic anywhere

- Age
  o Food allergy can develop at any age.
  o We most commonly recognize food allergy in very young patients (those with symptoms starting less than 12 months of age) and older patients (those with symptoms starting after 8 years of age).

- Common historical features
  o There is no known genetic inheritance for food allergy so siblings and parents are typically normal.
  o No seasonal variation is observed
  o Many have GI symptoms as well
    ▪ soft stool / diarrhea
    ▪ frequent vomiting
    ▪ increased number of bowel movements per day
    ▪ increased flatulence.

Food allergy truths

- There is no quick and accurate test for food allergies in pets.
  o Blood tests are inaccurate and unrepeatable.
  o Saliva and hair tests are not based on scientific principals
- Proteins are the most common allergens
- Grains are rarely a source of food allergy
- Raw diets are not healthier
- Changing the diet frequently is not beneficial
- Prescription diet trials are the only method of diagnosis
  o Contamination is prevented
    ▪ During ingredient handling
    ▪ During processing and packaging
  o Integrity of ingredient list
- Flavored medications can cause inadvertent exposure to food allergens
- If a grain allergy has not been diagnosed then there is no benefit to feeding a grain free diet. In fact, doing so might be dangerous.
Recent new concerns

- Boutique, grain free, and exotic protein dog foods have been linked to DCM
- Taurine levels are low in some dogs that develop DCM
- Most of the diets linked to DCM contain a large proportion of lentils or chickpeas
- Dietary modification reverses the DCM in some patients.
- Taurine supplementation helps some patients with suspected dietary related DCM.
- To learn about reporting a case of diet associated DCM to the FDA: https://www.fda.gov/AnimalVeterinary/NewsEvents/ucm630993.htm

Useful articles to empower your conversations with clients and critics

1) Diet-associated dilated cardiomyopathy in dogs: what do we know?
   a. Published December 1, 2018 in JAVMA. (Vol 253, No11, pg 1390-1394)
   b. There appear to be two groups of dogs that develop diet-associated DCM. Those with taurine deficiency and those with normal taurine levels.
   c. Measuring taurine levels is recommended when a dog eating a boutique, exotic protein, or grain free diet is diagnosed with DCM.
   d. Taurine supplementation is always recommended despite the taurine levels.
   e. Repeat echocardiogram is recommended 3-6 months after changing the diet.

2) Evaluation of serum allergen-specific IgE for the diagnosis of food adverse reactions in the dog.
   a. Published January 5, 2002 in Vet Dermatology. (Vol 9, Issue 3)
   b. “The failure of the monoclonal ELISA to detect any of the clinically diagnosed dogs was disappointing.”

3) Food-specific serum IgE and IgG reactivity in dogs with and without skin disease: lack of correlation between laboratories.
   a. Published June 1, 2014 in Vet Dermatology. (Vol 25, Issue 5)
   b. “These laboratories’ tests appear to have dubious predictive clinical utility because they neither correlate nor distinguish between dogs of different disease status.”
   c. “serological tests are not likely to yield useful results in cases of food intolerance because such responses are thought not to be mediated by serum antibodies. ”
   d. “The pathogenic significance of IgG antibodies to food antigens in dogs has not yet been elucidated; the traditional view was that their detection merely reflects previous exposure and tolerance and not a specific food-related pathogenesis”

4) Hair and saliva analysis fails to accurately identify atopic dogs or differentiate real and fake samples.
   a. Published January 24, 2019 in Vet Dermatology.
   b. “The direct-to-consumer hair and saliva test for pet allergies examined in this study performed no better than chance and the results were not reproducible.”
   c. “Positive test results were provided by the direct-to-consumer pet allergy for all submitted samples, including synthetic fur and saline. The test results for healthy and atopic animal samples were no different from each other or from synthetic fur and saline samples.”
5) Determination of mammalian DNA in commercial canine diets with uncommon and limited ingredients.
   a. Published February 5, 2019 in Vet Med Science
   b. “Twenty-one OTC adult canine diets marketed as limited or single protein source diets were purchased. Multiplex PCR was used to screen for DNA of 10 mammalian species with species-specific primers……The presence of DNA from one or more species not declared on the label was identified in all 21 diets.”
   c. “….one diet was negative for the declared species [listed on the label as the protein source]”
One of the biggest challenges in Dermatology is the fact that most of the conditions we treat never truly go away. As you are probably painfully aware, dermatologic diseases are typically managed for life. Dermatologic care requires multiple veterinary visits and usually some form of recurring in-hospital diagnostic tests as well as at home therapies. This can be challenging for all involved and very easily cause client frustration and pet anxiety. It is no surprise that client compliance in the realm of dermatology is very difficult.

Fortunately, we have a unique tool that can’t be found on Google. We have perspective gained from our experiences. We must combine our perspective along with medical knowledge and physical exam findings to communicate and educate our clients. Keep in mind that our clients typically need to hear information multiple (3-7) times before they remember and take action. Also remember that people have different learning styles. Some learn best from a face to face conversation while others like to read the material or watch a video. Consider your job as supporting your client’s education, not just about the visit today but about what to expect throughout their pet’s life.

Surely some of you are thinking, “I became a Veterinarian to help animals, not to babysit people.” But, are you sure you are thinking about helping the whole pet when you see a dermatologic condition? Are you controlling pain and helping avoid or manage anxiety surrounding skin and ear problems? Do you have patients that hide or show their teeth when they see the otoscope? Do your clients ever tell you that their dog runs and hides when it sees the ear wash bottle?

Here are some simple tips to make a life time of dermatology issues easier:

1) It is ‘OK’ to stop. (AKA: Know when to post-pone)
   a. Sometimes the best thing you can do is stop.
      i. Veterinary staff are usually very adept at recognizing warning signs from their patients.
      ii. Instead of gearing up for a fight simply choose not to fight.
      iii. Give yourself permission to stop (Obviously if the situation is life-threatening you must proceed. But those events are rare in dermatology)
   b. Listen to your patient – your client will thank you.
      i. Clients don’t like to see their pet struggle
      ii. Your client may not mention previous difficulties at the Vet’s office because they are embarrassed or blame their previous Vet and hope it will be better in a new hospital.
      iii. Explain the benefits of postponing and most clients will happily agree.
   c. Don’t force your way through an exam or procedure. The idea of "don't let them win" really creates a lose – lose situation.
      i. You might “win” this time with a muzzle and enough force. But, what about next time?
      ii. Pets learn from every experience at your hospital. What do you want their “take away” to be?
      iii. Eventually anxiety will escalate. Then one of your staff may get injured, or the client may go somewhere else.
d. Postponing in order to get medication on board helps everyone
   i. Use pain relievers to make the patient more comfortable and help prevent
      escalation of anxiety.
   ii. Use pain relievers and sedatives/tranquilizers to improve the success of your
      exam.

e. Dermatology example:
   i. Mr. Jones brings his dog Princess (a 45kg Rottweiler) in for head shaking and a
      foul odor coming from her ears. Princess has never had ear problems before
      and she is generally a calm, friendly dog. You are able to perform your physical
      exam and obtain ear cytology samples with only a mild increase in Princess’s
      stress level. But, when you try to examine her ears with the otoscope she cries
      and escapes to sit in the corner as far from you as possible. This is your chance
      to stop and say something like: “Obviously Princess’s ears don’t feel very good.
      She’s such a great dog and she is politely trying to tell us she has had enough.
      I’d like to examine her ear canals and tell you about her ear drums but we should
      post-pone that for now. Don’t worry, we’ll still be able to start treatment today.
      I’m sure that after we help manage Princess’s pain she’ll allow me to perform a
      full ear exam.” NOTE: It is ideal to visualize the ear drum before you begin otic
      therapy. However, in truth it is nearly impossible to see the entire structure with
      a hand-held otoscope even in the calmest, most well-trained dog.

2) Be mindful in your exam
   a. We’ve all had the client who, instead of talking with us first, shoved their pet across the
      exam table at us and jabbed at wherever they thought their pet was hurting. How scary
      for the pet!
   b. Examine the problem area last.
      i. Explain why you are saving the problem area for last.
         1. Looking for additional clues
         2. Don’t want to get distracted and miss something
      ii. Think of your exam as an interview with the pet.
         1. You wouldn’t start with the toughest question.
   c. Be consistent and gentle in your touch.
      i. Maintain contact and glide your hands to new areas to avoid surprises.
      ii. I think of how I was taught to examine a horse.
      iii. Keep a steady pace and tone in your “interview.”

3) Treats, treats, and more treats
   a. Treats can be used to reward desired behavior.
      i. You may have to educate your client on the proper way to treat
   b. Treats can be used as distraction
   c. Have a wide variety of treats to improve your chance of success

4) Prescribe pain relievers for otitis cases
   a. Every ear infection hurts!
   b. Otic steroids alone are not sufficient to control the pain associated with otitis externa
   c. NSAIDS can be helpful as long as the dog’s condition doesn’t need systemic steroid
      therapy.
   d. Gabapentin and Trazodone are extremely beneficial and can be used with systemic
      steroids. Both can cause sedation so advise your clients appropriately.
   e. Gabapentin has a very large dose range. Doses of 10mg/kg to 20mg/kg are common for
      pain relief. I usually start around 7mg/kg every 8-12 hours.
f. Trazodone is dosed between 2.5mg/kg and 12mg/kg. I typically start at 5-7mg/kg for anxiety related to ear treatments. You might recommend your client give this medication every 8-12 hours on a schedule. Or you might suggest they give it once daily, 1-2 hours before they plan to medicate their pet’s ears.

g. These medications are usually only needed for 7-14 days and both are inexpensive.

h. **This is the most important thing you can take from this lecture**

5) Think ahead about In-hospital sedation protocols
   a. Having a plan in place saves times and keeps your staff at ease.
      i. When your staff is at ease it shows in their confidence and proficiency which helps the client remain calm.
   b. Many sedative combinations contain Dexmedetomadine because it can be reversed.
USEFUL BACKGROUND INFORMATION

In order to talk meaningfully about Staph infections, we have to start with some basic definitions. We will start with minimum inhibitory concentration (MIC). This is the lowest concentration of an antimicrobial which inhibits bacterial growth. Most laboratories also correlate MIC with a suggested antibiotic dose when applicable. Think of dosing antibiotic therapy at the MIC like doing the least amount of work possible to keep your job. In contrast, mutant prevention concentration (MPC) is the threshold above which the selection of mutants is expected to occur only rarely. This is because multiple mutations are necessary to survive the MPC.

Terminology is the other key point we need to discuss. Many of us went to Veterinary College back when *Staph Intermedius* was the primary bacteria on dogs. Consequently, I think a brief history of *Staph pseudintermedius* is helpful. Information on Staph Intermedius was first published in 1976. Over the following thirty years researchers noticed variability amongst staph intermedius isolates. In 2005 *Staph pseudintermedius* was identified as a separate species. Soon after researchers changed the taxonomy of Staph Intermedius. They placed three specific isolates: *Staph intermedia*, *Staph pseudintermedius*, and *Staph delphini* into the *Staph intermedia* group (SIG). It was discovered that *Staph pseudintermedius* is actually the organism most commonly found on the dog. Unfortunately, some diagnostic laboratories still lag behind and either name organisms improperly or identify them incorrectly.

Over the past ten years there has been an explosion of information about methicillin resistance. It is worth mentioning that bacterial isolates are no longer tested against methicillin. Many years ago, diagnostic labs switched to oxacillin but we have kept the term methicillin resistance. Second, methicillin resistance is based on the expression of the mecA gene. The mecA gene codes for an alternative penicillin binding protein called PBP2A. Third, the mecA gene is located on a mobile gene element called the staphylococcal cassette chromosome mec (SCC mec) element. It is important to note that this is a mobile gene element because being mobile allows the resistance gene to be shared among bacteria. Lastly, it is important to understand that there is already documented evidence of bacterial resistance (gene or mutation) for every antibiotic available to Veterinarians today. Methicillin resistant *Staphylococci* has gained media attention but there are many other highly resistant bacteria we need to be concerned about.

With all the concern about methicillin resistant infections it is important for us to understand factors that contribute to development of these infections. Several papers have been published evaluating risk factors for developing methicillin resistant staph infections. A 2016 study by Hensel et al looked at 53 patients with MRSP. Ninety-eight percent of the patients who grew MRSP on culture had received systemic antibiotic therapy at least once in the past three years. Furthermore, the number of antibiotic prescriptions and the variety of antibiotics used directly correlated to the risk of developing MRSP. Other statistically significant factors that increased the chance of MRSP were exposure to beta-lactam antibiotics and concurrent immune modulatory therapy. A 2013 study by Eckholm et al also found that previous antibiotic therapy was important in the development of MRSP. In addition, this research found that hospitalization within the past year increased the risk of MRSP. Interestingly, in the Eckholm study there was no statistically significant difference in the occurrence of MRSP between patients examined at a primary care facility (general practice) and a tertiary care center (specialty or referral hospital).
BACTERIAL CULTURES

With the development of more and more antibiotic resistance it is important to know when and how to culture. It is never wrong to perform a bacterial culture when you are presented with a patient with a bacterial infection. However, there are times when performing a culture is more important. First, an infection which has not responded to your first antibiotic choice should be cultured. Second, a patient who has experienced a resistant infection in the past should be cultured when a new infection develops. Third, deep or penetrating wounds should be cultured. Fourth, patients with immune suppression who develop an infection should be cultured. Immune suppression includes metabolic disease such as diabetes, hypothyroidism, and Cushing’s as well as patients who are receiving long term immune modulatory therapy for autoimmune disease like IMHA, ITP or allergy. I also suggest that you consider culturing sooner in patients who live with an immune compromised human and those owned by medical professionals.

How you collect your culture sample is also very important. Sampling an intact pustule is relatively easy. You can use a sterile needle or the edge of a clean “fresh” glass slide to gently tear the pustule open then collect the material with your culturette. A similar technique can be used to gently abrade the top of papules. (The glass slide is easier to use on papules compared to a needle.) However, many cases may only present with crusts. Choose the crust you sample carefully. Ideally you want to sample a crust which is associated with erythema. If that isn’t available, then look for tightly adherent crust. Again, use a sterile needle or the edge of a clean “fresh” glass slide to pry up the edge of the crust. Then rub your culturette swab underneath. The leading edges of the crust or collarette is going to be most useful diagnostically. You want to avoid crusts that are loosely attached or trapped in the hair. Culture results are very helpful for guiding therapy, but it is also important to note that MR staph often change their resistance pattern during the course of treatment.

TOPICAL THERAPY REVIEW

Topical antimicrobial therapy is extremely important in the fight against bacterial skin infection regardless whether the bacteria is methicillin resistant or not. As a general rule we need to change our mentality to always use topical antimicrobial therapy and add systemic antibiotic therapy only when necessary. Such protocols will reduce antibiotic use and thus antibiotic resistance. When systemic antibiotics are necessary we will reduce resistance by attacking infection with two different mechanisms of action. Borio et all published a study in 2015 comparing the efficacy of topical antimicrobial therapy to systemic antibiotic therapy and found the two methods to be equally effective in treating superficial bacterial pyoderma. Topical therapy can have many additional benefits besides just being antimicrobial. First, adverse reactions to topical treatments are relatively rare. Furthermore, if a negative reaction does occur the product can be washed away with cool water. Second, topical therapy removes infectious agents reducing the load which needs to be addressed with antimicrobials. Third, topical therapy also removes inflammatory mediators which perpetuate inflammation and itch. Fourth, topical therapy can be soothing and reduce itching. Lastly, topical therapy with added ceramides can help restore the epidermal barrier.

Choosing a topical therapy regimen can be confusing. The first step is to consider the delivery method. Will your client and patient be amenable to frequent bathing? Do you want to follow bathing with a medicated conditioner? Or would a spray, mousse, or wipe be more appropriate? Shampooing is generally the best choice because the physical act of shampooing has benefits that other methods do not. However, it just isn’t feasible for many of our clients. It is critical to explain the importance of topical therapy to your clients and then seek their input to determine the most appropriate therapy in their unique situation.

There are a limited number of topical antimicrobial ingredients and they are roughly the same across the application methods. The most common antimicrobial ingredients include chlorhexidine (various concentrations and sometimes combined with miconazole), benzoyl peroxide, ethyl lactate, sodium hypochlorite, hypochlorous acid, acetic/boric acid combination, and, nisin. Chlorhexidine is generally considered the most effective, but it is sometimes necessary to use multiple types of ingredients in severe pyoderma. The most common reasons why topical therapy fails are poor compliance and improper technique. Consequently, it is important that you explain the treatment plan to your client and demonstrate what they need to do.
Efficacy data is limited but worth reviewing.

2016: An in vitro study found TrisEDTA alone did not inhibit bacterial growth. This study did show that combination therapy with Chlorhexidine and Miconazole resulted in MIC’s lower than for either drug alone.6

2016: An in vitro study compared the efficacy of topical therapy with Chlorhexidine, Isopropyl alcohol, sodium hydroxide, hypochlorous acid, lime sulfur, Manuka honey, and hydrocortisone aceponate. Manuka honey had no effect on bacteria. Hydrocortisone aceponate and lime sulfur had minimal effect. Chlorhexidine, sodium hypochlorite, and hypochlorous acid had the best efficacy.7

2016: An in vitro study compared the residual antibacterial activity of hairs treated with various topical antimicrobial sprays. The study compared 1% chlorhexidine; 2% chlorhexidine combined with 2% miconazole and Triz EDTA; 3% Chlorhexidine; and 4% chlorhexidine combined with Triz EDTA. Hairs treated with the product containing chlorhexidine, miconazole, and TrizEDTA showed the greatest bacterial inhibition. The 4% chlorhexidine spray was second best.8

2013: A study evaluated the residual antibacterial activity of hairs after being shampooed with six different products. Products tests were: 0.8% Chlorhexidine, 2% chlorhexidine, 3% chlorhexidine, 4% chlorhexidine, ethyl lactate, and benzoyl peroxide. The 2% and 3% chlorhexidine products produced the best results.9

2011: A study evaluated antibacterial properties of the following shampoos: 2% Chlorhexidine with 2% Miconazole; 3% Chlorhexidine; 4% Chlorhexidine; Benzoyl peroxide; Chloroxylenol; Ethyl lactate, and Acetic acid/Boric acid. The Chlorhexidine products were most effective followed by benzoyl peroxide. Ethyl lactate was only effective at very high concentrations. Chloroxylenol and Acetic/Boric acid were not effective.10

BASIC TREATMENT GUIDELINES:

1) Bathing is always preferred when feasible because it washes away debris, inflammatory mediators, and infection. Bathe weekly for mild pyoderma, twice a week for moderate pyoderma and every other day or daily for resistant/severe infection. Contact time really does matter for effective shampoo therapy. If a client can’t or won’t bathe properly then try a different formulation.

2) Sprays and Mousse products are great options when owners can’t bathe or can’t bathe more than once a week. I recommend these products weekly for prevention, twice weekly for mild pyoderma and daily for resistant infections.

3) Wipes are great for small wounds, skin folds, and paws. For prevention use weekly. For infection use daily or every other day.

4) For resistant infections I like to combine products with different active ingredients. For example, you might recommend bathing weekly with a Chlorhex/Miconazole/Silver shampoo and alternate daily therapy with a chlorhexidine mousse or a hypochlorous acid spray.

REFERENCES


The whole idea of this lecture is to provide product recommendations for those of you who don’t love the nuances of dermatology so I’ll keep the notes short and sweet. Keep in mind that there are a multitude of great products that are not listed here. This is a bare-bones list to cover 95% of what you deal with daily. I am not suggesting you keep all of these products in your clinic. Pick one product from each category. Depending on your clientele and preferences you might even skip an entire category. Beware generic or distributor products that claim to be the same. Usually those products have lower percentages of active ingredients.

- **Topical therapy:**
  - **Antiseptics**
    - **Shampoo:**
      - Dechra MiconaHex + Triz.
      - Douxo Chlorhexidine PS.
      - VetBiotek Biohex
    - **Spray:**
      - Vetericyn VF.
      - Microcyn AH.
      - Dechra MiconaHex + Triz
    - **Mousse:**
      - Dechra MiconaHex + Triz.
      - Douxo Chlorhexidine PS
    - **Wipe:**
      - Douxo Chlorhex PS
      - Dechra MiconaHex + Triz
    - **Conditioner / leave on lotion**
      - VetBiotek KC conditioner
  - **Moisturizers**
    - **Spray:**
      - Dechra Dermalay Spray.
      - Douxo Seborrhea spray
    - **Shampoo:**
      - Dechra Dermalay shampoo.
      - Douxo Seborrhea shampoo
    - **Spot-on:**
      - Dermoscent 6 spot-on
  - **Ear wash**
    - General cleansing and most ear infections: Epi-Otic advanced by Virbac
    - Cleansing when treating otitis with “rods” identified on cytology: A wash containing Triz EDTA. The best being products made by Dechra. I typically use Malaket plus
Topical ear medications:
- Long acting options: Claro or Osurnia
- First line options: Mometomax or EasOtic
- Second line options: Posatex or Surolan

Topical therapy for allergic otitis (prevention when no infection present)
- VetBiotek Ultra Otic rinse and concentrate
- Zymox HC drops and wash

Parasiticides
- All of the isoxazoline class of flea and tick preventatives will kill both sarcoptes and demodex mites.
- Consider Bravecto because it comes in both topical and oral formulations and can be used in dogs and cats.
- Revolution plus is a new option that combines Selamectin and Saraloner for cats

Antifungals
- Elanco’s Itrafungol is an itraconazole liquid that is very effective for ringworm and very easy to administer.
- For ringworm in dogs I recommend oral terbinafine at a dose of 30-40mg/kg/day. Terbinafine tablets only come in one size (250mg) and a reasonably priced generic is usually available.
- For Malassezia I suggest oral ketoconazole (5mg/kg/day with a meal) or oral fluconazole (5mg/kg/day with a meal)

Prescription food for dietary allergy trials
- Royal Canin Select Protein KO or Ultamino (both are only available in dry kibble)
- Rayne Clinical Nutrition Rabbit, Kangaroo, or Crocodile formulas
- BalanceIT for home cooked recipes options that are easy to generate and facilitate

Diets for allergic dogs without food allergy
- Avoid grain free, exotic proteins and boutique diets!
- Hill’s Prescription JD
- Royal Canin Veterinary diet Mobility Support
- Royal Canin Skin support
- Hill’s Sensitive skin and stomach
Hepatocutaneous Syndrome

The pathogenesis of hepatocutaneous syndrome involves death of keratinocytes in the upper layer of the epidermis due to presumed amino acid starvation. Most affected dogs have a distinctive chronic hepatopathy; but, serum chemistry evaluation may not reveal any abnormalities. Potential causes of the hepatopathy include phenobarbital, primidone, mycotoxin, and gastro-enteritis. In humans this syndrome is almost always associated with glucagonoma. However, Glucagonoma is rare in dogs and accounted for only 8% of cases in one study.

Hepatocutaneous syndrome is generally a disease of older dogs. Only four cases have been reported in cats. Skin lesions are typically the first sign as opposed to more common systemic signs of liver disease. Crusts and erosions occur in areas of trauma / wear. Thus, the paw pads are usually severely affected. The elbows, hocks, and muzzle are frequently affected as well. Many affected patients are often reluctant to walk due to painful erosions and fissures on the paw pads.

Diagnosis requires biopsy of skin lesions with intact crusts. Abdominal ultrasound can also be very helpful. A classic “honey comb” pattern to liver is present in most cases of Hepatocutaneous syndrome. However, the degree of change found on ultrasound does not necessarily correlate to severity of skin disease. CBC, serum chemistry, and urinalysis are also recommended. Nonregenerative anemia is common due to chronic disease. Liver values may or may not be elevated. Hyperglycemia is common and may require insulin therapy. Glucagon levels are elevated in patients with glucagonoma. However, glucagonoma is rare in dogs and cats and glucagon measurement is not readily available.

Management of Hepatocutaneous syndrome is difficult because this disease is a marker of severe internal disease. Consider referring these patients. Affected animals may need both a dermatologist and an internist.

For glucagonoma related disease it is recommended to remove the glucagonoma surgically. Unfortunately, glucagonomas have usually metastasized to the liver and abdominal lymph nodes by the time dermatologic lesions manifest. Cats with glucagonoma may also develop metastasis to the lungs and intestines. Even if metastasis has not occurred, affected patients are typically geriatric and my not be good surgical candidates. Octreotide, a synthetic somatostatin analogue, may be helpful for glucagonoma related disease. Octreotide binds to somatostatin receptors 2 and 5 to inhibit the release of glucagon. Octreotide will not affect the actual tumor but can yield quick and dramatic improvement in skin lesions. Octreotide is given two to four times daily indefinitely until the neoplasm progresses to the point of euthanasia or natural death. Theoretically, Octreotide would also be helpful for hepatic neuroendocrine tumors causing elevated glucagon levels and thus hepatocutaneous syndrome. However, hepatic neuroendocrine tumors are extremely rare with only one case reported in the dog and one in the cat.

A more common therapy is intravenous Aminosyn. Aminosyn can be useful regardless of the underlying cause but is most effective for hepatopathy related disease (which is the most common form). However, Aminosyn does not fix the underlying liver problem. Aminosyn provides nutrition to the starving keratinocytes. Aminosyn injections are typically given once to twice weekly initially and then spread out with injections given every 4-8 weeks long term. Aminosyn can yield a clinical response for up to twenty-two months. Unfortunately, Aminosyn is expensive and the injections must be given over several hours which requires hospitalization. A typical Aminosyn dose is 500ml/dog or 25mg/kg over 6-8 hours.

Supportive nutritional therapy is always recommended for Hepatocutaneous syndrome. Nutritional therapy involves increased protein intake via supplementation with egg yolks and cottage cheese, increased fatty acid intake, and Zinc supplementation (zinc methionine 2mg/kg/day). The typical life expectancy with supportive therapy alone is 2 to 5 months.
Steroid therapy can provide temporary improvement of clinical signs. However, patients eventually become resistant to steroid effects and steroid administration predisposes to Diabetes Mellitus (remember that many patients are hyperglycemic at presentation). Topical steroid sprays or ointments may be very useful for focal lesions and carry less risk of inducing Diabetes.

Monitoring for and addressing secondary infection becomes a constant battle in Hepatocutaneous syndrome. Bacterial infection and Malassezia dermatitis are common due to damage of the epidermal barrier. Bacterial culture and oral antibiotics may be necessary. Many of these patients do not eat well and it may be difficult for the owner to administer an oral antibiotic. Consequently, antiseptic sprays and wipes are particularly helpful.

**Cutaneous Lymphoma (not auto-immune but can mimic auto-immune skin disease)**

These neoplasms are important even though they are rare because they imitate many other diseases. Non-epitheliotrophic lymphoma typically manifests as single or multiple nodules. Exfoliative erythroderma may occur separately or in addition to nodular disease. Patients with exfoliative erythroderma can easily be misdiagnosed as allergy, scabies, or seborrhea. If the mucus membranes and/or muzzle are affected by non-epitheliotrophic lymphoma it can appear visually indistinct from lupus erythematosus, pemphigus vulgaris, and bullous pemphigoid.

The most common epitheliotrophic lymphoma is mycosis fungoides. This condition displays multiple clinical manifestations. Erythroderma is typically present (same as non-epitheliotrophic). Once again, this erythroderma may appear visually indistinct from allergy, scabies, and seborrhea. Focal lesions progress from patches to plaques to tumors. The final stage involves wide-spread dissemination of tumors with lymph node involvement. Multiple types of lesions can be present at the same time and the speed of progression is not predictable or consistent. Additionally, initial lesions can be very subtle. For example, a client may notice the development of dry flaky seborrhea. During examination you might find a couple small patches of alopecia without inflammation and a nodule which the owner cannot remember. Diagnosis is relatively straight forward via biopsy. The point of this lecture is merely to encourage you to biopsy older animals or animals with sudden onset of disease more quickly.

Therapy depends on the location and the extent of the disease. Consultation with an oncologist should always be recommended. Survival time varies greatly based on aggressiveness of the neoplasia and when the disease is diagnosed. In my clinical experience, most patients survive 2-3 months after diagnosis but this can range from a few weeks up to 18 months. For clients un-interested in oncology referral or classical “chemotherapy” I recommend steroids as monotherapy. Steroid monotherapy can provide 1-3 months of quality time by reducing the intensity of lesions and subsequent discomfort.

**Pemphigus foliaceus**

Pemphigus foliaceus is one of the most common auto-immune skin diseases seen in dogs and cats. This disease is characterized by pustules and honey colored crusts. This condition is typically idiopathic but it can develop secondary to drug exposure. Pemphigus foliaceus is often seen in patients previously diagnosed with allergic dermatitis; however, no link between the two has been proven.

In pemphigus foliaceus the immune system is attacking a particular protein in the complex structure (called a desmosome) that links keratinocytes together. Destroying the bonds between keratinocytes is termed acantholysis and results in acantholytic cells. Acantholytic cells are typically plump and round because they are no longer connected to their neighbors. They stain darkly and have a clearly visible nucleus. Different forms of pemphigus exist and one of the primary differences between them is what layer of the skin this acantholysis occurs. For pemphigus foliaceus the damage occurs in the two uppermost layers (the stratum corneum and the stratum granulosum). More serious forms of pemphigus affect deeper layers of the skin and cause significantly more damage. As acantholysis occurs, vesicles and sterile pustules are formed. These are fragile and easily damaged because they are located in the uppermost layers of the epidermis. Depending on the intensity of the immune response, pustules can develop and rupture in under an hour or over the course of days. For comparison, pyoderma pustules develop more slowly and are more resilient (more difficult to break). In addition, pyoderma pustules are
typically centered around a hair follicle. Both pemphigus pustules and pyoderma pustules will contain neutrophils but intact pemphigus pustules will not contain bacteria.

Lesions can occur anywhere on the body but are commonly found on the face and trunk. Pustules can develop inside the aural opening resulting in serum leakage and crust debris falling into the ear canals. The result is typically a wicked otitis externa. In many cases the nasal planum is also abnormal. The planum typically becomes dry, thick, and crusted. Ulcerations of the nasal planum can occur secondary to crust being traumatically removed. However, pemphigus foliaceus does not cause ulceration of the oral cavity or mucus membranes. The paw pads may be affected as well. Discreet pustules may be seen on the pads but more often the pads are thickened, dry, and crusted. Some dogs will be reluctant to walk but that is uncommon with pemphigus (much more common with hepatocutaneous syndrome).

Diagnosis is via biopsy. Intact pustules are preferred because they offer the clearest picture of the disease process. However, crusts are also very useful biopsy specimens. When collecting biopsies for potential pemphigus foliaceus it is critical not to scrub the skin. In most cases it is advised to avoid shaving the animal’s fur as well. Even the slightest disturbance to the skin can damage the fragile pustules seen with this condition. In the event that no pustules are present, the proof of pemphigus might be in the crust on top of the skin rather than in the skin sample itself. Consequently, always include crust debris in the formalin jar and request the crust be processed when you biopsy for pemphigus.

Treatment, which is really to say management, is almost always successful but required life-long. Some cases of drug induced pemphigus foliaceus will remain in “remission” even when immune suppressive therapy is discontinued. Initial therapy requires steroid administration. Oral daily prednisone dosages of 2mg/kg to 6mg/kg are often required. Steroid therapy often yields dramatic improvement in two to four weeks when dosed adequately. Some patients will respond better to other steroids such as dexamethasone or triamcinolone. Recheck examinations every two to four weeks are critical to assess response to therapy and tailor drug therapy. Secondary bacterial infection is common in pemphigus and your clients will not be able to discern the difference between a pyoderma pustule (which needs antibiotics) and pemphigus pustule (which would cause you to evaluate your immune suppressive plan). In general, the goal is to slowly taper steroid therapy once clinical “remission” has been achieved. Over the course of three to four months some dogs will achieve good clinical response and can be maintained with every other day steroid therapy. However, the majority of patients will experience significant steroid side effects (such as weight gain, polyuria, polydipsia, polyphagia, behavioral abnormalities). Because of steroid side effects and the fact that most patients require life-long immune suppressive therapy it is typically necessary to add another medication as a steroid sparing agent. First line drugs for this purpose are cyclosporine and azathioprine. Second line drugs include mycophenolate and leflunomide. In most cases, I will start a steroid and a steroid sparing drug at the beginning of treatment. All of the above listed steroid sparing drugs have a delay of four to eight weeks until they become clinically effective.

**Erythema Multiforme**

Erythema Multiforme (EM) is an uncommon disease of the skin and mucus membranes that often presents with distinctive clinical lesions. EM is typically triggered by an infection, a drug, connective tissue damage, or neoplasia. Searching for a trigger once you have diagnosed EM is important because the trigger may be life-altering. If a trigger is left unresolved then it is difficult, if not impossible, to manage EM with pharmacologic therapy. Idiopathic cases have been reported but some of these may represent a situation where the infectious trigger has passed and the pet never manifested typical clinical signs. Another possible explanation for idiopathic cases is neoplasia present in small enough quantities as to avoid detection (initially).

Histologically, EM manifests as keratinocyte apoptosis in all layers of the epidermis. The location of the damage and how quickly it occurs will affect the type of lesions you observe clinically. If keratinocytes are dying rapidly or predominantly in the lower layers you are more likely to observe erosions and ulcerations. If the keratinocytes are dying more slowly or in the upper layers then you are more likely to observe seborrhea or epidermal thickening.

Clinically the primary lesion is target like and most often develops on the caudal ventral abdomen. (These lesions start with a central erythemic papule that spreads outward and clears centrally.) The
target shaped lesions quickly blend together to yield a serpiginous pattern. Urticarial plaques, vesicles, and bullae may also be noted. Lesions on the mucocutaneous junctions occur in nearly half of all cases. The paw pads and pinnae may develop focal ulceration.

Treatment involves removing the underlying trigger if there is one and immune suppression. Oral steroid therapy is the mainstay of treatment initially. Most cases require long term treatment so it is best to attempt to transition to Cyclosporine or Azathioprine.

**Sebaceous Adenitis**

Sebaceous glands are found throughout haired skin associated with hair follicles. These glands open through a duct into the infundibulum of hair follicles. (In other words, they don’t open directly onto the skin surface.) The sebum or oil that these glands produce is responsible for keeping the skin soft and pliable as well as giving the hair a glossy sheen. Sebaceous adenitis occurs when granulomatous inflammation attacks and damages these glands.

Poodles, Akitas and Samoyeds are on the top of the list of breeds commonly affected. Other breeds often seen with this condition include Vizslas, Havanese, Lhasa apsos, and Springer spaniels. Affected dogs are usually young to middle aged. Males are more commonly affected than females. Lesions are usually first noticed on the dorsal neck and/or head. Over time the entire dorsum usually becomes involved. The early lesions (erythema and scale) are mild and often mistaken for other diseases. Short coated and long coated breeds present differently.

**Short coated symptoms:** Patches of alopecia and scale that enlarge peripherally. The coat may have a moth-eaten appearance with papular crusted folliculitis. Scale is typically very fine, white and non-adherent. Follicular casts may be absent.

**Long coated symptoms:** The first observed symptom might be a change in hair color (lighter or darker) or a change from curly to wavy or straight hair. Scaling and follicular casts are common. Hairs become dull and brittle. Pyoderma is common.

Biopsy is needed to diagnosis sebaceous adenitis. As usual with dermatology cases, it is important not to scrub or clip the area prior to biopsy sample collection. Include any crust that falls off with your biopsy sample. From a histopathologic stand-point the diagnosis is easy. The pathologist searches for and examines the sebaceous glands. There is benefit to biopsy beyond simply obtaining a definitive diagnosis. A good pathologist can tell you the status of the glands, the severity of the inflammation, and give an approximation of the number of glands remaining. This information can help you communicate with the client about prognosis. In chronic cases all of the sebaceous glands are typically completely destroyed or absent. These cases are less likely to benefit from pharmacologic therapy.

Medical therapy usually takes a two-pronged approach.

1) Systemic therapy to suppress the inflammation damaging the sebaceous glands. This is most successfully accomplished with cyclosporine. In addition to stopping the inflammatory damage to the sebaceous glands there is evidence that cyclosporine can encourage the remaining glands to function better. (Similar to why we treat "dry eye" with cyclosporine drops.) Steroids are not generally useful except for reducing pruritus and erythema. Synthetic retinoids have been used in the past; however, due to cost and numerous safety concerns this therapy is rarely recommended.

2) Topical therapy aims to remove the build-up of debris and replenish the oils which are absent. Care should be taken to avoid harsh seborrhea shampoos (such as coal tar and sulfur) as these can severely irritate the skin. In general, it is better to moisturize and loosen the debris rather than strip it off. The ideal topical products contain ceramides or phytosphingasine to replenish the natural oils missing from the skin. Typically, a multi-step approach is needed with medicated bathing two or three times weekly along with daily application of a moisturizer (spray, spot-on, or conditioner). Long ago, dogs with severe sebaceous adenitis were soaked in propylene glycol or baby oil for hours each week. (Drenched might be a better term.) While this can be effective the process is a logistical nightmare.
Polyuria and Polydipsia
David Bruyette, DVM, DACVIM

Introduction

Polyuria and polydipsia (PU / PD) refer to excessive water consumption and urine production respectively. These are common clinical signs in both dogs and cats. Water consumption exceeding 100 ml/kg or urine production exceeding 50 ml/kg body weight per day is considered abnormal and should be pursued. These numbers have been established in laboratory-reared dogs and may not reflect "normal" water consumption in pets. They are to be used only as guidelines. Water consumption can vary greatly from day to day so it is important to have owners subjectively assess water consumption in the home environment for several consecutive days in order to obtain an accurate picture before beginning unnecessary and expensive diagnostic tests. Actual quantification of water consumption can be very difficult and may not be practical for the majority of pet owners.

Normal Water Homeostasis

Extracellular fluid volume is maintained by regulation of fluid intake and urine production. The thirst center is stimulated by an increase in plasma osmolality (sodium concentration) and/or a decrease in blood volume (hypovolemia) resulting in an increase in water consumption. Increasing plasma osmolality and hypovolemia also stimulate osmoreceptors in the anterior hypothalamus and baroreceptors in the aortic arch resulting in the release of antidiuretic hormone (ADH) from the anterior pituitary. ADH circulates and binds to receptors on the renal tubular cells of the distal tubules and collecting ducts resulting in the production of cAMP. This causes the opening of pores in the luminal membrane of the tubular cells and allows for reabsorption of water from the glomerular filtrate resulting in a concentrated urine. In order for water to be pulled out of the tubule it must move along a concentration gradient maintained by the hypertonic renal medullary interstitium. Loss of this gradient (medullary washout), will result in an inability to concentrate urine even in the face of normal ADH activity. Urea and sodium are largely responsible for maintaining the hypertonicity of the interstitium. E. The sensation of thirst and secretion of ADH are suppressed when plasma osmolality and blood volume are returned to normal.

Differential Diagnosis: Mechanisms of PU/PD

A. Renal disease:
   1. Chronic renal failure: A decrease in the number of functional nephrons causes an increase in tubular flow in the remaining nephrons and leads to a solute diuresis. A decrease in urine concentrating ability may be the only laboratory abnormality indicating renal disease (especially in feline patients) presented for PU/PD.
   2. Pyelonephritis: Bacterial induced tubular destruction and an increase in renal blood flow cause a decrease in medullary hypertonicity.
   3. Primary renal glycosuria (Fanconi's Syndrome): A proximal tubular defect results in renal glycosuria leading to an osmotic diuresis. The blood glucose is normal.
   4. Post-Obstructive diuresis: May be seen in previously blocked cats. Due to osmotic diuresis from loss of large amounts of sodium and urea into the urine following relief of urethral obstruction.

B. Diabetes mellitus:
   Hyperglycemia results in glycosuria and an osmotic diuresis. Threshold for renal glycosuria is a blood glucose of 180 – 220 mg/dl (dog) and 240 – 300 mg/dl (cat).

C. Liver disease:
   PU/PD may occur as the result of: (1) decreased production of urea which is a major component of the hypertonic medullary interstitium, (2) increased renin and cortisol levels due to a lack of hepatic degradation, (3) increased aldosterone concentration leading to increased sodium concentration, and (4) hypokalemia (see hypokalemic nephropathy).
D. Hyperthyroidism:
Increased total renal blood flow reducing the tonicity of the medullary interstitium. Psychogenic polydipsia or primary polydipsia is reported in humans with hyperthyroidism.

E. Hypercalcemia:
(1) Interference with cAMP activation by ADH, (2) damage to ADH receptors, and (3) mineralization of renal tubular cells.

F. Hyperadrenocorticism:
Glucocorticoids interfere with the action of ADH at the renal tubule and decrease ADH secretion by reducing osmoreceptor sensitivity to rising plasma osmolality.

G. Hypoadrenocorticism:
Renal sodium wasting leads to decreased medullary hypertonicity.

H. Pyometra:
(1) E. coli endotoxins interfere with sodium reabsorption and damage ADH receptors and (2) may result in an immune-complex glomerulonephritis.

I. Hypokalemia:
(1) Degeneration of renal tubular cells, (2) decreased medullary hypertonicity, (3) stimulation of thirst, and (4) stimulation of renin release.

J. Polycythemia:
Mechanism unknown; may be related to sluggish blood flow in kidney or hypothalamus.

K. Medications:
Exogenous steroids, diuretics, salt supplementation, primidone, phenobarbital, KBr and vitamin D.

L. Pituitary or central diabetes insipidus (CDI):
Due to inadequate production, storage or release of ADH. May occur as a congenital defect or secondary to trauma, mass lesions, infection or infarction of the pituitary or hypothalamus.

M. Nephrogenic diabetes insipidus (NDI):
Congenital structural or functional defects in ADH receptor. Rare in dogs and cats.

N. Primary polydipsia or psychogenic polydipsia:
Underlying cause unknown (possible CNS lesion); results in increased renal blood flow and a decrease in medullary hypertonicity. Extremely uncommon in dogs and cats and is largely a diagnosis of exclusion.

Diagnostic Approach to PU / PD

A. Document PU/PD actually exists. Recommend assessment of water consumption in the home environment. Hospitalized animals frequently do not drink as much as they would in their natural surroundings.

B. Quick evaluation of urine specific gravity and glucose is cheap, easy, and very helpful in evaluating animals for possible pathologic PU/PD. If the urine specific gravity of a non-glycosuric sample, obtained from a dog or cat without signs of dehydration, is greater than 1.030 (dog) or 1.035 (cat), the likelihood of pathologic PU/PD is small and further work-up may not be required.
C. Most causes of PU/PD will be identified following a good history, physical examination, and an initial data base consisting of a CBC, chemistry profile, and urinalysis with bacteriologic culture.

D. If a cause has not been discovered after step C, the most likely diagnoses are hyperadrenocorticism (dog only, cats with Cushing's are usually overtly diabetic), central and nephrogenic diabetes insipidus, and primary polydipsia. As hyperadrenocorticism is far more common than either of the other causes, an ACTH stimulation test, urine cortisol/creatinine ratio or low-dose dexamethasone suppression test should be performed before proceeding to the modified water deprivation test (See Canine Hyperadrenocorticism).

Trial Therapy with DDAVP

1. Aqueous vasopressin 2 - 3 units (dog) or 0.25 U/# (cat) is given SQ. Alternatively, DDAVP may administered into the conjunctival sac (1 – 2 drops for dogs and 1 drop for cats). Administer for 2-3 days.

Interpretation

A. CDI: After ADH administration, urine specific gravity should increase to greater than 1.012

C. NDI: No change in sp gravity following ADH injection.

Treatment of Polyuria and Polydipsia

A. Treat the underlying disorder!

B. Treatment of CDI

1. DDAVP (Desmopressin acetate) 1-2 drops into the conjunctival sac or 0.01 to 0.05 mls subcutaneously SID or BID. May also dose orally with 0.1 to 0.2 mg once or twice a day.
   a. 1 drop = 1.5 to 4.0 ug. Can use TB syringe to dose.
   b. Duration 8 - 24 hours.
   c. Redosed when polyuria returns.
   d. Most commonly used treatment today.
   e. Use the intranasal preparation.

2. Chlorpropamide (Diabenese)
   b. 25 - 40 mg once or twice a day (cat). Limited experience.

C. Treatment of NDI

1. Salt restriction
2. Thiazide diuretics:
   a. Natriuresis results in a decrease in blood volume and increased sodium reabsorption in the proximal tubule.
   b. Hydrochlorothiazide 12.5 - 25 mg once or twice a day (cat).
   c. Chlorthiazide 20 - 40 mg/kg BID (dogs).
   d. May also help with partial CDI.

D. Treatment of Primary Polydipsia

1. Treatment to restore hypertonic renal medullary interstitium.
2. Gradual water restriction over several days.
3. Behavioral modification or referral to a behaviorist may be needed.
Diagnosis and Treatment of Hyperadrenocorticism in Dogs
David Bruyette, DVM, DACVIM

1. Introduction

A. Cushing's syndrome refers to all causes of hyperadrenocorticism with overproduction of cortisol.

1. ACTH-dependent

A. Cushing's disease: Pituitary hypersecretion of ACTH which results in bilateral adrenal hyperplasia (90% of cases)

B. Ectopic ACTH production: Non-pituitary tumors secreting ACTH resulting in bilateral adrenal hyperplasia. Has not been completely documented in dogs or cats.

2. ACTH independent

A. Adrenocortical adenoma or carcinoma: Hypersecretion of cortisol with atrophy of normal adrenal and suppressed ACTH concentrations (10% of cases).

3. Iatrogenic

A. Excessive or prolonged administration of glucocorticoids. Clinically indistinguishable from natural disease. Results in adrenal atrophy and suppressed ACTH levels.

2. Signalment

A. Poodles, Dachshunds, Schnauzers, Boston Terriers, Boxers.

B. Middle to old age. Average 12 years; range 6 months to 17 years.

C. No sex predilection.

D. Rare in cats. Usually seen with insulin resistant diabetes mellitus and/or cats with severe dermal atrophy/ulceration.

3. Clinical Signs

A. PU / PD

B. Pendulous, "pot-bellied abdomen": Due to muscle catabolism by glucocorticoids and hepatomegaly.

C. Bilaterally symmetric alopecia: Head and extremities spared.

D. Thin skin

E. Muscle weakness and muscle atrophy; cruciate ruptures

F. Mineralization of skin (calcinosus cutis)

G. Hyperpigmentation: ACTH similar to MSH, co-existing hypothyroidism, chronic skin irritation.

H. Reproductive abnormalities
1. Anestrus
2. Clitoral hypertrophy
3. Testicular atrophy
4. Perianal adenomas in females and neutered males.

I. Respiratory signs

1. Panting: Pulmonary hypertension and decreased compliance, primary CNS disturbance, pulmonary mineralization.
2. Dyspnea: Rare; seen with pulmonary thromboembolism and concurrent congestive heart failure.

J. Central nervous system

1. Seen with large pituitary tumors (macroadenomas). Present at time of diagnosis or following therapy for Cushing’s disease as microscopic pituitary tumors enlarge into macroadenomas.
2. Signs due to compression/invasion of pituitary and/or hypothalamus:
   A. Seizures
   B. Pacing
   C. Lethargy
   D. Inappetence
   E. Behavior change
   F. Head pressing
   G. Circling

4. Diagnosis of Hyperadrenocorticism

A. History and clinical signs

B. R/O iatrogenic disease with questions concerning current or past medications. These medications can include oral, ophthalmic, otic, and topical medications. Make sure the owner tells you about everything and anything that went on or in their pet.

C. Laboratory data

1. Hemogram
   A. Polycythemia (PCV 45-55%)
   B. Stress leukogram
      1. Lymphopenia
2. Eosinopenia
3. Neutrophilia (mature)

2. Biochemistry profile
   A. Elevations in:
      1. Serum alkaline phosphatase (SAP)
      2. Cholesterol
      3. Serum alanine aminotransferase (ALT)
      4. Fasting blood glucose: Diabetes in 5-10%.

3. Thyroid function tests
   A. T3 and T4 basal levels are generally decreased.
   B. Response to TSH parallels normal.
   C. Secondary to negative feedback of cortisol on pituitary.
   D. 80% have a normal fT4ED
   D. Does not require thyroid supplementation.

4. Blood pressure: 50 – 80% are hypertensive, cause unknown.
   A. Recent study demonstrated normal or decreased levels of atrial natriuretic factor (ANF) in dogs with hyperadrenocorticism. Argues against hypervolemia as the etiology of the hypertension.

5. Urinalysis
   A. Decreased urine specific gravity.
   B. Proteinuria

D. Radiographic abnormalities
   1. Thoracic films
      A. Bronchial calcification
      B. Metastases from adrenal adenocarcinoma
   2. Abdominal films
      A. Hepatomegaly
      B. Osteopenia
      C. 50% of adrenal tumors are visualized as soft tissue or calcified masses.
      D. Subcutaneous calcification
E. Adrenal function tests

1. Three tests used to diagnose hyperadrenocorticism. They do not differentiate between PDH or AT.

   A. ACTH stimulation test
      1. Look for exaggerated cortisol response in response to ACTH.
      2. See protocols at the end of this discussion.
      3. Diagnostic in 85% of pituitary-dependent cases (PDH)
      4. Diagnostic in 70% of adrenal tumors (AT)
      5. Overall accuracy 80-85 %
      6. A suppressed response to ACTH in animals with clinical signs of hyperadrenocorticism suggests iatrogenic disease.

   B. Low-dose dexamethasone suppression test
      1. Low doses of dexamethasone inhibit ACTH release from the pituitary via negative feedback and decrease plasma cortisol concentrations in normal dogs.
      2. Dogs with Cushing's are more resistant to steroid suppression. Therefore, lack of suppression following dexamethasone = hyperadrenocorticism.
      3. Diagnostic in 95% of PDH
      4. Diagnostic in 100% of AT
      5. Overall 90-95%
      6. May also be used to distinguish PDH from AT (see below)
      7. See protocols

   C. Urine cortisol/creatinine ratio
      1. Assessment of cortisol production and excretion rate.
      2. Sensitivity of this test is greater than that of the LDDS (some animals with clinical signs of hyperadrenocorticism may have normal LDDS response tests but elevated urine cortisol to creatinine ratios). Used as a screening test.
      3. Test is easy to perform.
      4. As with all adrenal function tests, elevated results may occur in animals with non-adrenal disease.
      5. Positive tests confirmed with a LDDS.
      6. Must be performed on urine obtained at home, preferably in the AM
2. Tests to differentiate PDH from AT (performed after confirming diagnosis of hyperadrenocorticism).

   A. High-dose dexamethasone suppression test

      1. With PDH, a high dose of dexamethasone results in a decrease in ACTH release from the pituitary and a decrease in plasma cortisol.

      2. With AT, the tumor secretes cortisol autonomously thereby suppressing ACTH production. With low ACTH concentrations already present, dexamethasone has no effect on plasma cortisol.

      3. 70% of patients with PDH suppress plasma cortisol to less than 50% of the pre-treatment value.

      4. 100% of patients with AT do not suppress.

      5. Therefore: Suppression = PDH; Lack of suppression = Inconclusive

      6. See protocol

   B. Endogenous ACTH concentration

      1. PDH: Levels normal or high

      2. AT: Levels low to undetectable

      3. Contact lab regarding sample handling and collection. Use of the preservative (Aprotinin) allows for greater utilization of this test.

      4. Excellent method to differentiate PDH from AT.

**Testing Protocols**

These are suggested protocols that are used in the evaluation of patients with hyperadrenocorticism. You must use the protocol and normal values from the laboratory to whom you are submitting samples to properly evaluate endocrine tests.

1. ACTH Stimulation Test

   A. Synthetic ACTH (Cortrosyn) 5 ug/kg IV or IM; collect serum at 0 and 1 hour, or

   B. ACTH gel (Acthar) 2.2 U/kg IM; collect serum at 0 and 2 hours.

   C. Hyperadrenocorticism if post-cortisol > 20 ug/dl (530 nmol/L)

2. Low-Dose Dexamethasone Suppression Test

   A. 8 A.m: Baseline serum cortisol. Administer 0.01 mg/kg dexamethasone sodium phosphate (0.015 mg/kg dexamethasone) IV.

   B. 12 p.m: Collect 4-hour post-dexamethasone cortisol.

   C. 4 p.m: Collect 8-hour post-dexamethasone cortisol.

   D. In normal animals cortisol suppresses to less than 1.0 ug/dl (27.5 mmol/L) at 8 hours.
E. 50% or greater suppression at either 4 or 8 hours together with lack of suppression at 8 hours is diagnostic for PDH and additional tests are not necessary.

3. Urine Cortisol/Creatinine Ratio
   A. First morning urine sample is preferred. Sample should be obtained at home. Requires 1 – 2 mls.
   B. Stable at room temperature or refrigerated for 3 days.
   C. Normal range 2.8 - 4.8. A normal result effectively rules-out hyperadrenocorticism, an abnormal result should be confirmed with a LDDS or ACTH stimulation test.

Differentiating PDH from AT

1. Low-Dose Dexamethasone Suppression Test
   A. See above.

2. High-Dose Dexamethasone Suppression Test
   A. 8 a.m: Obtain serum cortisol. Administer 0.1 mg/kg dexamethasone sodium phosphate (0.15 mg/kg dexamethasone) IV.
   B. 4 p.m: Collect post-dexamethasone cortisol.
   C. Suppression defined as greater than a 50% reduction of cortisol.
   D. Suppression = PDH, non-suppression = Inconclusive

3. Endogenous ACTH Concentration
   A. Check with lab on sample collection and handling.
   B. Normal: 20-100 pg/ml (4.4-22.0 pmol/L)
   C. PDH: 40-500 pg /ml (8.8-110 pmol/L)
   D. AT: < 20 pg/ml (<4.4 pmol/L)

Treatment Options

A. Pituitary-dependent hyperadrenocorticism
   1. Surgical management
      A. Bilateral adrenalectomy
         1. Technically difficult
         2. Poor surgical/anesthetic risk
         3. Permanently hypoadrenal and require lifelong replacement therapy
B. Hypophysectomy

1. See discussion at the end of this section

2. Lifelong therapy with thyroid hormone and prednisone necessary.

2. Medical therapy

**Prognosis:** Most dogs with PDH live normal lives (average 2.2 years, but remember most are geriatric to begin with.)

1. Complications

   A. Recurrence of disease.
   
   B. CNS signs.
   
   C. Pulmonary thromboembolism.
   
   D. Infections.
   
   E. Hypertension.
   
   F. Congestive heart failure.

2. Adrenal tumors:

   1. Adenomas: Good if no evidence of local invasion.
   
   2. Carcinomas: Guarded to grave with metastases.

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**Trilostane Therapy of Canine Hyperadrenocorticism**

The efficacy and safety of trilostane in the treatment of canine PDH were evaluated in a multicentre study at the Royal Veterinary College in London, the Veterinary Teaching Hospital in Dublin and Small Animal Hospital in Glasgow. Seventy-eight dogs with confirmed PDH were treated with trilostane for up to 3 years. The starting dose varied from 1.8 to 20 mg/kg (mean = 5.9 mg/kg).

Trilostane appeared to be well tolerated by almost all dogs with only 2 dogs developing signs and biochemical evidence of hypoadrenocorticism. One of these dogs recovered with appropriate therapy. The other died despite withdrawal of trilostane and administration of appropriate therapy. A further two dogs died within one week of starting trilostane but in neither case could a direct link with the trilostane therapy be established. The low prevalence of side effects compared favourably to those reported with mitotane.

Trilostane was found to be nearly as effective as mitotane in resolving the signs of hyperadrenocorticism. Polyuria, polydipsia and polyphagia had dissipated in 40 dogs within 3 weeks after starting trilostane. Within 2 months, a further 20 dogs showed decreases in their water and food consumption. These improvements were maintained as long as the dogs remained on adequate doses of trilostane. Skin changes resolved in 24 out of 39 (62%) of dogs that initially presented with dermatological signs. All of these improvements were maintained as long as the dogs remained on adequate doses of trilostane. Only 8 dogs that were treated with trilostane for more than 2 months showed poor control of clinical signs. In contrast, mitotane is effective in about 80% of cases of pituitary dependent hyperadrenocorticism (PDH).

Trilostane caused a significant (p<0.001) reduction in both the mean basal and post-ACTH stimulation cortisol concentrations after 10 days of treatment. The post ACTH cortisol concentration decreased to less than 250
nmol/l (9 µg/dl) in 81% of dogs within one month and in another 15% at some time whilst on treatment. These improvements were also maintained in the study population for the duration of the trial.

Thirty-five dogs had at least one dose adjustment over the treatment period. The dose was increased in 23 dogs up to four times the starting dose. In one dog the dose was increased nine-fold over a period of six months. The dose was decreased in nine dogs to as low as a quarter of the starting dose.

The mean survival of all trilostane treated dogs was 661 days. Direct comparison with mitotane was difficult as 65% of the dogs were still alive at the time of censor and therefore the mean survival may still increase. By comparison, the mean survival of mitotane treated dogs has been reported to be 810 to 900 days.

Dosage and administration

The current suggested initial starting dose range for dogs with PDH is 1-2 mg/kg once daily. This needs to be adjusted according to clinical signs and serum cortisol values (see below). Doses up to 40-50 mg/kg (divided twice daily) have been given with no unwanted side effects. In some dogs twice daily dosing may be necessary. The drug is given with food.

**TRANSSPHENOIDAL HYPOPHYSECTOMY**

A variety of treatments are available for PDH. Medical treatment options include drugs that chemically destroy the adrenals (lysodren or op-DDD) inhibit enzymes in the adrenal leading to the synthesis of cortisol (ketoconazole, trilostane) or inhibit the release of ACTH from the pituitary gland (Anipryl or selegiline). While these treatments can improve the clinical signs in 40-80% of patients they need to be chronically administered, necessitate frequent monitoring and do not cure or address the primary cause of the disease (the pituitary tumor). In humans, surgery to remove the tumor is the most successful long-term therapy. The most common approach used is the transsphenoidal method, in which a passage way is made in the sphenoid sinus, an air space behind the back of the nose, which is just below the pituitary gland. Surgical cure rates for PDH are reported to be in the range of 65-85%, although more recent long-term follow up data suggest that the recurrence rate is as high as 25% within 5 years. When no discrete adenoma can be identified, remission of hypercortisolism is observed in only about 40%. Surgery has also been used to treat PDH in dogs. Several groups, most notably in the Netherlands have performed these surgeries with success rates paralleling those reported for humans. However, these surgeries have generally not been performed in the US. Veterinarians at VCAWLAAH, in collaboration with human neurosurgeons that regularly perform transsphenoidal surgery in humans have developed the methods to perform these surgeries in the US and are conducting a research study to determine how effectively these surgeries can be performed.

Given the survival times and the ability to cure the disease by removing the pituitary tumor we are conducting ongoing studies to evaluate the role of transsphenoidal hypophysectomy in the treatment of canine PDH. We will also be looking at the tumor tissue to investigate the pathogenesis of Cushing’s disease and evaluate novel medical therapies.
Canine Hypoadrenocorticism
David Bruyette, DVM, DACVIM

Canine Hypoadrenocorticism
1. Introduction

A. Etiology
   1. Primary adrenocortical failure (Addison's disease)
      A. Auto-immune disorder (Most common type)
      B. Destructive lesions
         1. Histoplasmosis
         2. Blastomycosis
         3. Metastatic neoplasia
      C. Infarction of adrenals
      D. Amyloidosis
      E. Secondary to o,p'-DDD
   2. Secondary (lack of ACTH secretion)
      A. Destructive lesions of pituitary/hypothalamus.
      B. Chronic, excessive steroid use.
      C. Hypopituitarism
      D. Idiopathic

2. Signalment
   A. Young to middle age.
   B. Females > Males (80% Female).
   C. May be a breed predilection in Standard Poodles.

3. Pathophysiology
   A. Requires 90% loss of adrenal cortex.
   B. Destruction is usually gradual and symptoms first appear during times of stress (trauma, surgery, infection). Ultimately, hormone secretion is inadequate even under normal conditions.
   C. Lack of glucocorticoids
      1. Gastrointestinal effects
         A. Anorexia
         B. Vomiting
         C. Abdominal Pain
         D. Weight loss
      2. Mental changes
         A. Lethargy
      3. Metabolic effects
         A. Decreased gluconeogenesis.
         B. Decreased fat metabolism and utilization.
         C. Hepatic glycogen depletion leading to fasting hypoglycemia.
   D. Lack of aldosterone
      1. Inability to conserve sodium
         A. Hypovolemia
         B. Weight loss
         C. Decreased blood pressure
         D. Decreased cardiac output
         E. Decreased renal blood flow; pre- renal azotemia
         F. Weakness
2. Inability to excrete potassium
   A. Due to reduced GFR
   B. Decreased excitability of heart
   C. Slows conduction (EKG abnormalities)
E. o,p'-DDD therapy can produce similar signs
   1. Sodium and potassium should be monitored during therapy.
F. Secondary adrenal insufficiency
   1. Aldosterone normal so no electrolyte abnormalities.
   2. Signs due to glucocorticoid insufficiency.

4. Clinical Signs

A. Waxing - waning course
B. Lethargy/depression
C. Weakness
D. Anorexia/weight loss
E. Vomiting/diarrhea/melena
F. Abdominal pain
G. PU / PD

5. Physical Examination

A. Weakness/unable to stand.
B. Weak, thready pulse.
C. Dehydration
D. Tachycardia (bradycardia with Hyperkalemia).
E. Abdominal pain

6. Laboratory Abnormalities

A. Hemogram
   1. Normocytic, normochromic anemia (may be masked by dehydration). Marked GI blood loss can also occur.
   2. Eosinophilia and lymphocytosis. Normal lymphocyte and eosinophil counts in a severely ill animal should make you suspicious of hypoadrenocorticism.
B. Biochemistry profile
   1. Uremia
      A. Usually pre-renal
      B. Specific gravity 1.010-1.025
      C. Responds to fluid therapy.
      D. Frequently Addison's misdiagnosed initially as renal failure.
   2. Hypoglycemia (rare)
   3. Hypercalcemia (25% of cases): Decreased renal calcium excretion.
   4. Metabolic acidosis
   5. Electrolyte abnormalities
      A. Classically hyponatremia, hypochloremia, hyperkalemia.
      B. May be normal if some aldosterone production is present. The disease can be slowly progressive and is not an "all-or-none" phenomenon.
      C. May be normal if treated elsewhere with fluids.
      D. Will be normal if atypical hypoadrenocorticism or ACTH deficiency (very rare).
      E. May be normal or have hypokalemia with severe intestinal loss (vomiting, diarrhea, GI hemorrhage).
      F. A Na/K ratio < 25:1 suggestive of Addison's.
G. A decreased Na/K ratio may also be seen with:
1. Renal failure
2. Severe acidosis
3. Primary gastrointestinal disease (esp. whipworms).
4. Severe liver disease.
5. Pleural and peritoneal effusions.

7. Radiographic Abnormalities
   A. Microcardia
   B. Decreased size of aorta and vena cava.
   C. Megaesophagus (rare)

8. EKG Abnormalities
   A. Useful in assessing hyperkalemia and following response to treatment. Not helpful in predicting initial K level. Most helpful if you can measure the potassium at the beginning and use the EKG to monitor response to therapy.
   B. Potassium > 5.5
      1. Spiked T wave
      2. Shortened Q-T interval
   C. Potassium > 6.5
      1. Prolonged QRS complex
   D. Potassium > 7.0
      1. Prolonged P wave, decreased amplitude
      2. Prolonged P-R interval
      3. Prolonged QRS complex, decreased amplitude
   E. Potassium > 8.5
      1. Lack of P waves
      2. Bradycardia
      3. Ventricular fibrillation

9. Diagnosis
   A. ACTH stimulation test
      1. To test adrenal reserve
      2. Same as protocol described under hyperadrenocorticism.
      3. Pre and post-cortisol below normal basal level.
      4. Test can be performed while instituting emergency therapy (see below).
   B. Endogenous ACTH concentration
      1. To distinguish primary versus secondary hypoadrenocorticism. Mainly of academic interest as the treatment is the same.
      2. Elevated with primary disease or post-Lysodren therapy.
      3. Decreased with secondary disease or following overdosage with steroids.

10. Treatment
    A. Correct hypovolemia
        1. 40-80 ml/kg normal saline (0.9%) IV over 1st hour.
        2. Decrease to maintenance needs.
        3. If concerned about hypoglycemia make solution 5% dextrose in saline.
        4. The most important aspect of therapy is fluid replacement.
B. Correct electrolyte abnormalities
   1. Fluid therapy is the best therapy.
      A. Saline restores sodium and chloride concentration.
      B. Promotes potassium excretion.
      C. Corrects hypovolemia
      D. Corrects pre-renal azotemia
      E. Promotes correction of acidosis by increasing tissue perfusion.
C. Glucocorticoid replacement (also see below)
   1. 30 mg/kg IV bolus Solu-Delta-Cortef.
   2. 1 mg/kg dexamethasone sodium phosphate IV (place in IV bottle).
D. Acidosis
   1. Use arterial blood gas or venous tCO2 to determine base deficit.
   2. Re-check following first hour of therapy. If tCO2 still <12, administer bicarbonate by adding it to the maintenance fluids. Calculate bicarb need: mEq bicarb = Body weight (kg) X 0.5 X Base deficit. Only add 25% of this total to the fluids and recheck in 4 hours. It is rarely necessary to use bicarbonate as most of these dogs will normalize or increase their tCO2 following fluid therapy.
E. Obtaining a diagnosis
   1. The ACTH stimulation test can be performed by taking the pre-ACTH cortisol sample when blood is obtained for other lab work (BUN, electrolytes, glucose, CO2, etc).
   2. The ACTH is injected and the post sample is obtained 1-2 hours later depending on your protocol.
   3. Treatment with Solu-Delta-Cortef, can be started following completion of the test. Dexamethasone can be used simultaneously as it will not interfere with the cortisol assay.

11. Maintenance Therapy
A. Mineralocorticoid therapy
   1. Start when animal is eating and drinking again.
   2. Fludrocortisone acetate (Florinef).
   3. 0.10 mg/10 lb starting dose.
   4. Maintain K concentration between 4.0-5.5 mEq/L.
   5. Monitor electrolytes and BUN every 1-2 weeks initially, then every 3-4 months when stable.
   6. If K is still high, increase Florinef by 0.10 mg/day and re-check in one week.
   7. Can also use desoxycorticosterone pivalate (DOCP) 2.2 mg/kg IM or SQ every 21-25 days.
B. Glucocorticoid therapy
   1. May not be needed in all dogs.
   2. May need replacement dose to prevent signs of glucocorticoid deficiency (0.2-0.4 mg/kg/day of prednisone).
   1. Should give owner 5 mg prednisone pills to give to the dog during illnesses or stress (hospitalization, boarding, etc.)
DEFINITION / OVERVIEW
Canine hypothyroidism, while a common endocrinopathy in the dog, may be over diagnosed due to confusion/inconsistencies in establishing a definitive diagnosis.

ETIOLOGY / PATHOPHYSIOLOGY
Hypothyroidism is due to decreased thyroidal production of the thyroid hormones thyroxine (T4) and triiodothyronine (T3). Greater than 90% of cases are primary and are due to acquired immune mediated destruction of the thyroid gland which is preceded by thyroiditis, idiopathic atrophy or less commonly neoplasia. Secondary forms of the disease include thyroid stimulating hormone (TSH) deficiency, pituitary neoplasia, and cystic Rathke’s pouch, are uncommon clinical entities. Tertiary hypothyroidism with thyrotropin releasing hormone (TRH) deficiency has not been documented in dogs. Congenital cases have been reported in both dogs and cats.

SIGNALMENT / HISTORY
Hypothyroidism most commonly occurs in young to middle aged dogs with an average age of 7 years. Dogs with autoimmune disease tend to develop hypothyroidism at a younger age. While thyroid values decrease within the reference range in senior dogs, hypothyroidism is very uncommon and other factors (see below) are likely responsible for the observed decreased thyroid concentrations in euthyroid older patients. Spayed females and neutered males are at an increased risk when compared to sexually intact animals. Breed predispositions have been reported for golden retrievers and Doberman pinschers. Thyroiditis is heritable in the beagle, Borzoi, golden retriever, great Dane, Irish setter, Doberman pinscher, and old English sheepdogs.

Risk Factors
No known environmental factors have been identified. Breed predispositions as outlined above.

Historical Findings
As thyroid hormone regulates the metabolic rate and influences the functions of many organs, clinical signs are often non-specific and insidious in onset. Many other diseases can have similar clinical signs to hypothyroidism, which may lead to an incorrect diagnosis. As such laboratory testing of thyroid function is often performed as part of the diagnostic work in animals with non-thyroidal illness.

CLINICAL FEATURES
Common clinical signs include lethargy, mental dullness, weight gain, exercise intolerance, alopecia, and obesity.

DIFFERENTIAL DIAGNOSIS
Many metabolic, infectious, neoplastic, congenital, degenerative, and inflammatory diseases can cause similar clinical signs and biochemical abnormalities seen with hypothyroidism.

DIAGNOSTICS
Laboratory Diagnosis
Thyroxine is the major secretory product of the thyroid while the majority of T3 is derived from extra-thyroidal sources. Both T4 and T3 are highly protein bound to serum carrier proteins such as thyroid binding globulin, transthyretin and albumin. Only unbound (free) hormone is able to penetrate cell membranes, bind to receptors and result in biologic activity. Protein bound hormone acts as a reservoir to maintain steady concentrations of free hormone in the plasma despite rapid alterations in release and metabolism of T3 and T4 and changes in the plasma protein concentrations.

Serum Total T4
Serum T4 is a sensitive (>90-95%), but not specific test (70-75%) for the diagnosis of canine hypothyroidism. The vast majority of dogs with hypothyroidism have a serum T4 below normal, but some normal dogs and those with a variety of other problems may have a low serum T4.
A diagnosis of hypothyroidism can be ruled out if the T4 is in the upper 50% of the reference range. Autoantibodies to T4 occur in about 15% of hypothyroid dogs, and these antibodies may falsely increase the serum T4 concentration from below normal into or above the normal range. In house testing of TT4 is not recommended.

**Serum Total T3**
Serum T3 concentration is an unreliable test for evaluation of thyroid function.

**Serum free T4 (fT4)**
Thyroxine is highly (99.9%) protein bound in the circulation. Protein binding can be altered by many nonthyroidal illnesses and by certain drugs. Measurement of the unbound or free hormone can provide a more accurate assessment of thyroid function in these cases (sensitivity > 95%, specificity > 97%). The sensitivity of fT4 is equivalent to or slightly better than total T4 in diagnosing hypothyroidism in routine cases. More importantly, fT4 is more specific, particularly when non-thyroidal factors that can influence total T4 are present. Free T4 is less affected by most non-thyroidal illness and drugs, but still can be altered in cases of moderate to severe illness. In addition, fT4 by equilibrium dialysis is not affected by the presence of T4 autoantibodies that will falsely elevate total T4. Measurement of fT4 by equilibrium dialysis should be performed when uncommon clinical signs of hypothyroidism are present, the dog is being treated with a drug that may affect thyroid function, when non-thyroidal illness is present, and if autoantibodies to T4 are detected.

**Serum TSH**
Primary hypothyroidism results in a decrease in T4 and thus decreased negative feedback on the pituitary gland. In response, the pituitary secretes more TSH and plasma TSH levels increase. In man, TSH is elevated prior to any decrease of T4 or fT4 outside the normal range. In the dog, TSH concentration is elevated in only 65-75% of cases of hypothyroidism, as such it lacks sensitivity for use as a screening test. The combination of decreased total T4 or fT4 with an elevated serum TSH is diagnostic of hypothyroidism (specificity > 95%). Therefore, a normal TSH does not rule out hypothyroidism, but an elevated TSH combined with a low T4 or fT4 provides a definitive diagnosis.

**Diagnosis of Thyroiditis**
Antibodies against either T4 or T3 or both are sometimes present in dogs with thyroiditis with or without hypothyroidism. The presence of these antibodies does not indicate that the dog is hypothyroid, but suggests that autoimmune thyroid disease is present. These antibodies frequently cause false elevation of T4 or T3 concentrations that can result in marked elevation of the hormones. Autoantibodies to T4 are present in about 10-15% of hypothyroid dogs.

Dogs with autoimmune thyroiditis may have circulating antibodies to thyroglobulin, the primary protein in the colloid of the thyroid gland. This is not a test of thyroid function, but rather a marker for the presence of autoimmune thyroiditis. In one long-term study at Michigan State University, 20% of asymptomatic, antithyroglobulin positive dogs with normal thyroid function progressed to hypothyroidism in 1 year. The presence of these antibodies in a dog with borderline laboratory evidence of hypothyroidism and clinical signs supports a diagnosis of hypothyroidism.

**Additional Considerations**

**Breeds**
Certain breeds have normal ranges of thyroid hormones that are different from most other breeds. Few have been evaluated, but greyhounds have serum total T4 and fT4 concentrations that are considerably lower than most other breeds. Scottish deerhounds, Saluki’s and whippets also have total T4 concentrations that are well below the mean concentration of dogs in general. Alaskan sled dogs have serum T4, T3, and fT4 concentrations that are below the reference range of most pet dogs, particularly during periods of intense training or racing.

**Time of Day**
In one study 50% of normal dogs had a low serum T4 concentration at some time during the day.
**Medications**

The drugs that are known to commonly alter thyroid function tests are glucocorticoids, phenobarbital, sulfonamides, clomipramine, aspirin, and some other NSAIDs. Glucocorticoids suppress total T4 and sometimes fT4 as well. Phenobarbital causes decreased total T4 and mild increases in TSH. Sulfonamides can induce overt primary hypothyroidism with clinical signs and thyroid function tests that support the diagnosis. The changes may be reversible when the medication is discontinued. There are dozens of drugs that affect thyroid function and thyroid function tests in man, so many others likely affect the dog as well.

**Nonthyroidal Illness**

Illness not involving the thyroid gland can alter thyroid function tests and has been labeled "non-thyroidal illness" or "euthyroid sick syndrome". Any illness can alter thyroid function tests, causing a fairly consistent decrease in total T4 and T3 concentrations in proportion to the severity of illness. Serum TSH concentration is increased in 8-10% of dogs with non-thyroidal illness. Serum fT4 measured by equilibrium dialysis is less likely to be affected, but can also be increased or decreased. However, in dogs with substantial non-thyroidal illness, the fT4 is likely to be decreased. It is recommended that testing of thyroid function be postponed until the non-thyroidal illness is resolved. If this is not possible, measurement of T4, TSH and fT4 are indicated.

**Ancillary Testing**

**Thyroid Gland Ultrasound**

Although rarely necessary, ultrasound of the thyroid glands (by an experienced ultrasonographer) can be used to aid in differentiating dogs with primary hypothyroidism from those with non-thyroidal illness. Thyroid glands of hypothyroid dogs tend to be smaller, less homogeneous, and hypoechoic than those of euthyroid dogs. There is considerable overlap with the ultrasonographic appearance and size of the thyroid glands of euthyroid and hypothyroid dogs. Thyroid ultrasound can only be used to help support a diagnosis of hypothyroidism if the thyroid glands are quite small.

**THERAPEUTICS**

**Drugs**

Levothyroxine is the only hormone that appears necessary for treatment of hypothyroidism. The frequency of levothyroxine dosing is controversial, and the only study to closely evaluate the response to treatment showed that once daily treatment is adequate. However, in clinical practice some dogs seem to respond better to twice-daily treatment.

The initial starting dose is 0.02 mg/kg PO q 24 h. In general you will never have to exceed 0.8 mg as an initial daily dosage even in very large dogs. If the dog has significant cardiovascular disease, diabetes mellitus, or hypoadrenocorticism, treatment should be instituted at 25% of the standard dose, with the dosage increased by 25% every 2 weeks based on clinical response and post-pill testing. Most dogs show improvement within the first 1-2 weeks, with increased activity, improved attitude, and partial or complete resolution of neurologic signs. The cutaneous manifestations of hypothyroidism may take several weeks to months to resolve.

Post treatment monitoring may be carried out but clinical response is the most important monitoring tool. Peak T4 concentrations generally occur 4-6 hours after administration of levothyroxine and should be in the high normal to slightly above normal range (40-70 nmol/L). However, the bioavailability of thyroxine ranges from 13 to 87% in the same dog from day to day bringing into the question the utility of random post pill monitoring of TT4. It is likely more meaningful (though more expensive) to measure TSH (especially if the TSH concentration was elevated pre- treatment) or fT4 concentrations after replacement therapy has been started, especially in animals that show a poor clinical response to therapy. Serum TSH concentrations should be in the normal range or undetectable and fT4 concentrations should be in the normal range. Serum concentrations of TSH and fT4 should not be performed until the patient has been on supplementation for at least 2 weeks. If the patient was initially started on twice daily therapy, treatment can be reduced to once daily treatment when a good clinical response has been obtained.
Hyperthyroidism is the most common complication of treatment with levothyroxine, but it is rare in dogs. Clinical signs are similar to those of hyperthyroidism in cats and the diagnosis is confirmed by documenting a substantial elevation of serum T4. Treatment consists of stopping levothyroxine treatment for 2-3 days, then instituting treatment at a lower dose.

COMMENTS

**Expected Course and Prognosis**
Response to therapy should be observed in the first 4-8 weeks post treatment. Improvements in mentation and physical activity may be noted within the first week though some abnormalities, especially dermatologic signs, may take several months to resolve. An absent or incomplete response to therapy may be due to an incorrect diagnosis, poor owner compliance, inadequate dosing, or poor absorption.
Diabetes Mellitus in Dogs and Cats
David Bruyette, DVM, DACVIM

Diabetes mellitus is a common endocrine disorder in dogs and cats. Recent data has shed light on the pathogenesis of the disorder in dogs and cats and has highlighted the role of diet, insulin and novel hypoglycemic therapies. In the majority of cases, the most appropriate therapy in both dog and cats includes the administration of insulin.

The key to successful management of the diabetic patient lies in close communication with the pet owner and prompt recognition and treatment of concurrent disorders.

**Key Facts:**

1. Insulin is still the mainstay of therapy in the majority of dogs and cats with diabetes mellitus.

2. Diet is an important part of diabetic management especially in obese patients and cats.

3. Auto-immune disease, pancreatitis and amyloidosis are the most common causes of diabetes in dogs and cats.

Successful management of the diabetic patient involves many factors. An understanding of dietary therapy, insulin preparations, oral and novel hypoglycemic agents and management of concurrent illness, are all required to optimize glycemic control. The goals of therapy are to control clinical signs, prevent or slow the progression of cataracts, avoid hypoglycemia and maintain ideal body weight. An additional goal in cats is to obtain remission. The challenge is to address these concerns while attempting to help the owners deal with what they may consider a time consuming, expensive and chronic medical condition.

Diabetes Mellitus in dogs and cats results from a decrease in insulin secretion from the beta cells of the pancreas and/or a decrease in insulin action. There are three classifications of diabetes:

**Type I** diabetes is comparable to insulin dependent diabetes mellitus (IDDM) in humans. It results in low basal insulin concentrations with impaired insulin secretion following a glucose load. Treatment requires insulin injections. It is the most common form of diabetes in dogs.

**Type II** diabetes is similar to non-insulin dependent diabetes (NIDDM) in humans and is managed with dietary therapy and oral hypoglycemics. It causes normal to increased basal insulin concentrations with decreased secretion following a glucose load. Insulin may or may not be required for animals with Type II diabetes.

**Type III** diabetes is seen most commonly in hormonally-induced diabetes in dogs and cats and is similar to impaired glucose tolerance (IGT) in humans. Diabetogenic hormones (epinephrine, cortisol, glucagon and growth hormone) or medications interfere with insulin action and cause glucose intolerance, which can lead to diabetes.

**Etiology and Signalment**

**Canine**

There are some distinct differences in the etiology of canine and feline diabetes. In dogs, it is generally thought to be an immune mediated disease with gradual destruction of beta cells. The progression from normal, to glucose intolerant, to diabetes, is generally slow so that most islets (over 90%) are lost before diabetes occurs. Other causes of diabetes in dogs include genetic predisposition, chronic pancreatitis and medication-induced diabetes (*glucocorticoids* and *megestrol acetate*).

Genetic predisposition to diabetes is most common in the following breeds: German Shepherd dogs, Schnauzers, Beagles, and Poodles. Golden Retrievers and Keeshonds are more prone to juvenile diabetes.

Gender is a factor in dogs with females being three times more likely to develop diabetes than males. Generally, diabetes occurs in dogs in middle age (6-9 years) but can also present earlier for specific breeds, particularly the Golden Retriever and Keeshond.
**Feline**

The most common causes of diabetes in cats are obesity, pancreatitis and most commonly, amyloidosis of the pancreatic beta cells. There appears to be very little gender predisposition to this disease in cats, although it is slightly more common in males than females. As with dogs, the onset of diabetes in cats occurs most often in middle age.

**Clinical Signs**

The clinical signs of diabetes include PU/PD (polyuria and polydipsia) from hyperglycemia, resulting in glycosuria and a resultant osmotic diuresis. Polyphagia and weight loss is common although many animals will still be obese upon presentation. In addition to the polyphagia, there may be variable degrees of dehydration especially in the cat. Cataract formation is very common in dogs with diabetes, but rare in cats. Cats often present with icterus as a result of concurrent hepatic lipidosis and/or pancreatitis. Icterus is not common in dogs unless they have pancreatitis. Cats may also exhibit a plantigrade stance (peripheral neuropathy) that is directly related to the severity and duration of hyperglycemia. Clinical neuropathies do occur in dogs, but are extremely rare.

**Differential diagnoses** include: hyperthyroidism (in cats), gastrointestinal lymphoma, hepatic disease, renal disease, pancreatitis, hyperadrenocorticism, and acromegaly.

**Diagnosis**

Diagnosis involves testing for persistent fasting hyperglycemia, with fasting blood glucose values greater than 200mg/dl. Clinicians also will need to rule out transient hyperglycemia that may be due to: post-prandial hyperglycemia; diabetogenic hormones (endogenous or exogenous); and stress hyperglycemia. Stress hyperglycemia can be a problem in cats due to the release of epinephrine when stressed or handled.

**Laboratory abnormalities** include:

- **Hemogram**
  1. non-specific
  2. signs of dehydration

- **Biochemistry profile**
  3. hyperglycemia
  4. increases in SAP and ALT
  5. increases in bilirubin (usually in cats)
     a) hepatic lipidosis
     b) pancreatitis

- **Urinalysis**
  1. glycosuria
     a) renal threshold for glucose
        - canine 180-220mg/dl
        - feline 240-300 mg/dl
  2. ketonuria
  3. up to 40% of patients will have positive urine cultures in the absence of an active urine sediment.

**Treatment**

The number one cause of death in diabetic dogs and cats is not the disease itself, rather, it is the owner’s frustration with the disease. This is an extremely important point to remember when treating diabetic animals. Good communication with the pet owner is perhaps the most important component of managing the disease.

It is recommended that clinicians schedule a 30-minute appointment with the client at the time of discharge before sending the diabetic patient home for the first time. During this appointment, clinicians should thoroughly discuss the care required for the patient. Include the following instructions in that discussion: how to give the animal injections; how to store insulin, what types of food to feed and how often; how to recognize the signs of hypoglycemia and how to react to this condition. Also include information on what clinical signs to look for in terms of monitoring water intake and urine production.
The client should be given written instructions for use as a reference once they are caring for the patient at home. It is essential that the clinician and veterinary staff strive to educate the caregiver and motivate them to get involved in the care of their diabetic pet.

The goals of treatment include elimination of the clinical signs of diabetes, prevention or slowing of cataract formation and resulting blindness, prevention of potentially dangerous hypoglycemia, and prevention and/or treatment of concurrent illness.

Therapy for diabetes centers on three main areas: Treatment of concurrent illness (i.e., urinary tract infections, pyoderma, etc.), insulin therapy, and dietary management.

**Concurrent illness.** Monitoring for concurrent illness is very important in effectively managing diabetic dogs and cats. Clinicians must effectively recognize and treat the other disorders because the concurrent illness will impact the diabetic regulation and many common diseases have similar clinical signs to diabetes mellitus. Even simple problems such as UTI's and pyodermas can result in activation of stress hormones and result in insulin resistance.

**Insulin Therapy.** There has been a considerable amount of confusion over the various insulin preparations that are available. In general, animal origin insulins are being discontinued as the desire and ability to treat people with human derived insulin preparations has progressed.

There is concern that animals receiving human insulin will develop antibodies resulting in decreased insulin activity and/or effectiveness. Dogs receiving any insulin product that is not derived from pork may make antibodies. However, studies have shown that those antibodies do not interfere with the glucose control. In fact, dogs that made antibodies against insulin had a longer duration of insulin action, which actually enhanced the effect of the insulin rather than decreased its efficacy. A recent study in cats should that 13% developed anti-insulin antibodies. None of the cats should signs of insulin resistance.

The options with human insulin include ultra-short acting, short acting, intermediate acting, and long-acting insulins. The short acting insulins are primarily used for ketoacidosis, and therefore, are not covered in this article. The intermediate acting insulins are classified as either NPH or Lente. It is important to note however, that even though they are classified as intermediate, they do not behave the same way in the dog or cat. Lente is actually a mixture of two different insulin preparations, which results in a bimodal onset of actions. This is helpful in some patients because it helps block post-prandial hyperglycemia. Conversely, a lente insulin is not recommended for use in an animal that does not develop post-prandial hyperglycemia. It is recommended that NPH be used in the majority of dogs and cats with diabetes and it is also understood that most patients will require two injections a day to achieve glycemic control.

**Canine Patients:**

**Newly Diagnosed Patients:**

1. Vetsulin (porcine origin lente): A zinc, porcine, intermediate acting insulin. Canine and porcine insulin have an identical amino acid sequence thereby eliminating the theoretical complication of anti-insulin antibodies and their effect on glycemic control. The suggested, initial starting dose is 0.5 units/kg BID. This insulin is only available at a concentration of 40 iu/ml (U-40) so please make sure that proper insulin syringes are provided to the owner. Re-assessment of clinical signs and a serial blood glucose curve should be performed 1 week after starting therapy. This insulin must be thoroughly shaken before administration. For additional information see: [www.vetsulin.com](http://www.vetsulin.com).

2. Humulin N or Novolin N; These are both intermediate acting, human origin insulins. Suggested starting doses are 0.5 units/kg BID. Re-assessment of clinical signs and a serial blood glucose curve should be performed 1 week after starting therapy. I would avoid NPH insulins from Wal Mart due to product inconsistencies.

3. Glargine:
Transitioning Canine Patients:

If you have canine patients currently taking Humulin L lente insulin, I would switch them to either Vetsulin or Humulin N. The initial dose of Vetsulin or Humulin N will remain the same with re-assessment of clinical signs and a serial blood glucose curve performed 1 week after changing insulin preparations.

Feline Patients:

Newly Diagnosed Patients:

1. Insulin glargine (Lantus): Glargine is a modified, recombinant, long acting insulin analog. A study presented at ACVIM in 2005 showed a very high rate of remission (8/8 in remission within 4 months with 6/7 still in remission at 1 year) in feline diabetics with the use of glargine and a low carbohydrate-high protein diet. The recommended starting dose is 0.5 units/kg BID if the fasting blood sugar is greater than 360 mg/dl and 0.25 units/kg BID if the initial fasting blood glucose is less than 360 mg/dl. For additional product information see: www.lantus.com. Glargine highlights:
   1. Should not be diluted or mixed as this will affect pH
   2. Should be kept refrigerated. Once open the vial has a shelf life of 4 weeks at room temperature. I would discard any remaining insulin after 8 weeks of refrigeration pending further clinical data.

2. PZI: As with dogs we only recommend the use of PZIR from BI.

3. Humulin N and Novolin N: Similar to PZI with remission rates of 40-50 % when used with a low carbohydrate-high protein diet. Starting doses are generally 1-3 units/cat once a day.

4. Vetsulin: Again, similar to PZI and Humulin N with remission rates of 40-50 % when used with a low carbohydrate-high protein diet. Starting doses are generally 1-3 units/cat once a day.

Transitioning Feline Patients:

If you have patients currently taking either Humulin L or Humulin U, I would switch them to either Vetsulin or Humulin N. The initial starting dose will remain the same with re-assessment of clinical signs and a serial blood glucose curve performed 1 week after changing insulin preparations. If you wish to transition them to glargine, I would follow the dosage recommendations as outlined above under newly diagnosed patients. It is important to note that remission rates will be much lower with glargine and a low carbohydrate-high protein diet in long standing diabetic patients (cats with diabetes for more than 6 months) than in newly diagnosed patients.

With the recent introduction of the AlphaTrak Blood Glucose Monitoring System (Abbott) we have the ability to very accurately measure blood glucose concentrations in both dogs and cats using very small quantities of blood. This will allow both veterinarians and pet owners to obtain very reliable results in both the hospital and home setting. This information can then be used to make informed decisions regarding the management of diabetic patients. These decisions impact the type and dose of insulin selected, the frequency of insulin administration, aid in the assessment of glycemic control, help in preventing hypoglycemic episodes and monitor for remission of diabetes especially in feline patients.

Glycemic control can be evaluated in a numbers of ways. Owner assessment of clinical signs (polyuria, polydipsia, weight gain or loss), progression of diabetic cataracts (dogs), presence of peripheral neuropathy (cats), and episodes of hypoglycemia are often the best indicators of glycemic control.
Changes in insulin dosage or documenting remission of diabetes, is best determined by blood glucose measurement. Recognizing that the measurement of blood glucose concentrations can be problematic in the hospital setting (especially in cats as a result of stress induced hyperglycemia) recent work has evaluated the practicality and value of at home blood glucose monitoring in dogs and cats. At home blood glucose monitoring is essential in the management of human patients with diabetes given that a number of the complications associated with long term diabetes are directly related to persistent hyperglycemia. While diabetic retinopathy, nephropathy, painful neuropathies and cardiovascular disease are rare in our veterinary patients, adequate glycemic control is required to eliminate clinical signs and decrease morbidity and mortality in dogs and cats. Control of clinical signs does not require the restoration of euglycemia but rather involves keeping the blood glucose levels below renal threshold for the majority of the day. Renal threshold for glucose is 180 mg/dl in the dog and approximately 280 mg/dl in the cat. It is very important that we remember the owners of diabetic dogs and cats are being asked to do a great deal to help in the management of their pet’s chronic illness and we need to do whatever we can to make the clients job easier while at the same time taking steps to assure maximal diabetic control.

Using the Information Derived Using at Home or In Hospital Glucose Monitoring

The data obtained with at home blood glucose monitoring in conjunction with clinical signs is used to adjust the dose of insulin and to monitor for remission of diabetes. We will look at scenarios for both cats and dogs. The recommendations for cats are based on our experience as well as the data generated by Dr Jacque Rand at the University of Queensland.

Cats

1. Cats on Glargine and PZI Insulins
   a. If the preinsulin blood glucose concentration is > 360 mg/dl and/or the nadir blood glucose (PZI) or 4-hour (glargine) post blood glucose concentration is > 180 mg/dl the dose of insulin is increased by 0.5 to 1 unit BID.
   b. If the preinsulin blood glucose concentration is 270 to 360 mg/dl and/or the nadir glucose (PZI) or 4-hour (glargine) post blood glucose blood glucose concentration is 90 - 180 mg/dl the dose of insulin is maintained.
   c. If the preinsulin blood glucose concentration is 190 - 270 mg/dl and/or the nadir glucose (PZI) or 4-hour (glargine) post blood glucose blood glucose concentration is 54 - 90 mg/dl use the nadir, clinical signs and the next preinsulin glucose concentration to determine if the dose is decreased or maintained.
   d. If the preinsulin blood glucose concentration is < 180 mg/dl and/or the nadir blood glucose (PZI) or 4-hour (glargine) post blood glucose glucose concentration is < 54 mg/dl the dose of insulin is decreased by 0.5 to 1 unit BID. If the total insulin dose is already 0.5 – 1 unit BID, stop the insulin and check for diabetic remission.

2. Cats on NPH, Lente or Ultralente Insulins
   a. If preinsulin blood glucose is < 210 mg/dl withhold insulin and check for diabetic remission.
   b. If preinsulin blood glucose is 234 - 288 mg/dl total insulin dose should not be higher than 1 unit BID.
   c. If nadir blood glucose is < 54 mg/dl insulin dose should be reduced by 50%.
   d. If nadir blood glucose is 54 - 90 mg/dl dose should be reduced by 1 unit BID.
   e. If nadir blood glucose is 91 - 162 mg/dl insulin dose should remain the same.
   f. If nadir blood glucose is > 180 mg/dl insulin dose should be increased by 1 unit BID.

Dogs

3. Dogs on NPH or Lente Insulins
   a. If the preinsulin blood glucose concentration is > 360 mg/dl and/or the nadir blood glucose concentration is > 180 mg/dl the dose of insulin is increased by 25%.
   b. If the preinsulin blood glucose concentration is 270 to 360 mg/dl and/or the nadir blood glucose concentration is 90 - 180 mg/dl the dose of insulin is maintained.
c. If the preinsulin blood glucose concentration is 190 - 270 mg/dl and/or the nadir blood glucose concentration is 54 - 90 mg/dl use the nadir, clinical signs and the next preinsulin glucose concentration to determine if the dose is decreased (50%) or maintained.

d. If the preinsulin blood glucose concentration is < 180 mg/dl and/or the nadir blood glucose concentration is < 54 mg/dl the dose of insulin is decreased by 50%.

The use of the AlphaTrak Blood Glucose Monitoring System both in the clinic and at home will greatly improve our ability to assess glycemic control and improve insulin therapy. In conjunction with close observation of clinical signs, at home glucose monitoring should go a long way towards improving the quality of life of diabetic pets and their owners.

**Diet.** There is a considerable amount of reliable research data showing that diets high in carbohydrates, low in fat and high in fiber are helpful in regulating diabetic dogs. These types of diets lower the average insulin dose, the average blood sugar, the amount of urine being produced and glycosolated hemoglobins and fructosamine levels.

The carbohydrates in these diets are complex carbohydrates. It is important to avoid diets high in simple sugars, which includes any commercial semi-moist food, primarily those packaged in foil packets. Diets high in simple sugars are absorbed very rapidly before the insulin has time to work. The goal with diet is to balance the absorption of sugar with the onset of action of the insulin. A high carbohydrate/low fat diets also decreases plasma free fatty acid and cholesterol concentrations, and increases the number and activity of insulin receptors.

High fiber diets reduce insulin resistance. The fiber acts to decrease post prandial hyperglycemia, primarily because it delays gastric emptying. A high fiber diet also decreases absorption of glucose and increases insulin action at the receptor.

It has recently been suggested that diabetic cats be fed a high protein/low carbohydrate diet. This can be accomplished with several commercially available canned diets (Hill’s M/D, IVD Development, Purina DM, many other canned kitten diets). These diets may result in remission of the diabetes and elimination of the need for exogenous insulin and/or oral hypoglycemic agents. High protein/low carbohydrate diets more closely resemble the diet of felines in the wild and may help reduce glucose intolerance, insulin resistance and obesity.

**Feeding.** Ideally, the feeding schedule should be coordinated with the onset of action of the insulin. With dogs, this is fairly easy to regulate, but with cats, it is nearly impossible due to their "grazing" style of eating. For cat owners who may not be able to follow a strict feeding schedule or those with multiple pet households, insulin therapy will have to be adjusted to meet the owner's needs. The most important component of the dietary plan is to stress consistency in the diet. The following feeding schedule can be used for dogs and some cats. With insulin given once a day, feed three meals a day (of equal calories) at six-hour internals. Give the first meal at the time of the insulin injection. For animals receiving insulin twice a day, feed four meals a day. Schedule them to coincide with the insulin injections and feed mid-afternoon and late evening.

If the owner is unable to follow this schedule, advise them to feed twice a day, at the time of injection and 8-10 hours later (for once a day insulin patients); or at the times of insulin injections (for twice a day insulin patients).

**Home Management**
1. Instruct owner on proper injection techniques, injection locations, storage and handling of insulin.
2. Instruct owner on how to monitor clinical signs.
3. Continue feeding schedule and dietary therapy.
4. Instruct owners to initially monitor urine glucose/ketone levels daily, usually in the morning or evening prior to feeding. If persistent glycosuria or ketonuria is observed, ask owner to contact the veterinary hospital.
5. Advise owners of the signs of and treatment for hypoglycemia. Have owners keep a bottle of Karo syrup on hand if signs occur (i.e., weakness, ataxia, seizures) so they can rub syrup on the gums immediately. Instruct them to call the veterinary hospital.
6. Home monitoring of a diabetic cat is frequently based on observance of clinical signs only.
7. Serial sugars after the first week of home management.
Re-check Evaluations
1. Obtain owner assessment of clinical signs.
2. Serial blood sugars are helpful due to:
   Variability of insulin action in a given patient.
   Inaccuracy of random blood or urine sugars in monitoring the degree of glycemic control.
   Not particularly helpful as a routine procedure in animals that are well controlled clinically.
3. Body weight
4. Physical examination/ophthalmic exam
5. Discuss urine log book with owner
6. Laboratory work as clinically indicated
7. Role of glycosylated hemoglobin and fructosamine:
   Fructosamine may be helpful in distinguishing stress-induced hyperglycemia from diabetes in cats.
   These tests can be used every 3 – 4 months as an indicator of long term (2-3 weeks fructosamine; 4-6 weeks glycosylated hemoglobin) glucose control. Rising values indicate the need for further evaluation.

Problems with Insulin Therapy
1. Insulin induced hyperglycemia (Somogyi phenomenon)
   - Hypoglycemia (<65mg/dl) followed by hyperglycemia (>300mg/dl) within 24 hours of insulin injection.
   - Suspect when insulin requirements exceed 2 U/kg and clinical signs persist.
   - Suspect when animal has signs of hypoglycemia in afternoon.
   - Diagnosis with serial sugars.
   - Treat by decreasing insulin dose 25-50% and review insulin administration with the owner to rule out management problems.
   - Re-check serial sugars in one week.
2. Rapid insulin metabolism
   - Duration of insulin less than 18 hours.
   - Signs return in the evening.
   - Diagnosis is with serial sugars. Hyperglycemia (>250) within 18 hours of insulin injection without previous hypoglycemia.
   - Treatment:
     - Review management with owner
     - Switch to twice daily insulin administration. Most dogs and cats require insulin twice a day to achieve adequate glycemic control. Consider switching to PZI in cats.
3. Insulin Resistance
   - Hyperglycemia (>300) throughout the day, despite insulin dosages > 2 U/kg.
   - Diagnosis based on serial sugars.
   - Potential causes of insulin resistance:
     Management problems
     Hyperadrenocoticism
     Steroid or Ovaban administration
     Diestrus or pregnancy
     Acromegaly
     Concurrent illness, infection
     Anti-insulin antibodies
     Hypothyroidism (dogs), hyperthyroidism (cats)
   - If insulin dose exceeds 2U/kg, the animal should be evaluated for one of these causes of resistance.
4. Hypoglycemia
   - Insulin overdosage
   - Suspect if animal shows weakness, shaking, ataxia, seizures at time of insulin's peak effect.
   - Therapy (instructions for owners)
     Mild signs - give food and call veterinarian
     Moderate signs - apply Karo syrup to the mouth, offer food when alert and then notify veterinarian.
     Comatose - apply Karo syrup to mouth and take animal to hospital.
   - When hypoglycemia occurs, serial sugars should be performed to re-assess insulin dose
Acromegaly

Feline acromegaly is a disease characterized by excessive growth hormone secretion leading to a wide array of clinical signs caused by the hormones effects on multiple organ systems. These effects can be divided into two major classes. The first are the catabolic actions of growth hormone that include: insulin antagonism and lipolysis with the net effect of promoting hyperglycemia. The second are the slow anabolic (or hypertrophic) effects of growth hormone which are mediated by insulin like growth factors. Growth hormone stimulates production of insulin like growth factors in several different tissues throughout the body. Insulin like growth factor-1 (IGF-1) which is produced in the liver is thought to be the key factor that facilitates the anabolic effects of growth hormone that are responsible for the characteristic appearance of acromegalic people, dogs, and cats. Similar to its etiology in humans, acromegaly in cats is the result of a functional adenoma of the pituitary that releases excessive growth hormone despite negative feedback.

Growth hormone is produced in anterior lobe of the pituitary gland, specifically by cells called somatotrophs. The regulation of growth hormone is very complex and many factors, both environmental and endogenous, are responsible for its control. The two most important regulators of growth hormone are the hypothalamic hormones, growth hormone releasing hormone (GHRH) and somatostatin. While growth hormone release is stimulated by GHRH, it is inhibited by somatostatin as well as by negative feedback from itself and IGF-1.

Feline acromegaly is an uncommon disease, although thought to be under diagnosed, it most commonly affects middle aged to older, male castrated cats. In one study, 13 of 14 cats with acromegaly were males with an average age of 10.2 years. This association may be biased, however, as most cats that are diagnosed with acromegaly present for complications associated with diabetes mellitus which is also more common in older, male castrated cats. Based on available data there is no known breed association for feline acromegaly.

Commonly patients with feline acromegaly present for insulin resistant diabetes mellitus (insulin doses dependant on insulin type) with concurrent weight gain rather than weight loss. Other clinical signs vary due to the wide range of effects the disease has on the body. Physical changes associated with feline acromegaly include increased body weight, a broadened face, enlarged feet, protrusion of the mandible (prognathia inferior), increased interdental spacing, organomegaly, and a poor haircoat. Respiratory disease may result from excessive growth of the soft palate and laryngeal tissues leading to stertorous breathing and even upper airway obstruction. Cardiovascular signs include the presence of a heart murmur, hypertension, arrhythmia, and congestive heart failure associated with hypertrophic cardiomyopathy. Neurologic disease associated with feline acromegaly is uncommon but can occur with larger pituitary adenomas. Neurologic signs that have been observed with acromegaly include dullness, lethargy, abnormal behavior, circling, and blindness. Glomerulopathy and secondary renal failure has also been associated with feline acromegaly. Histopathologic evaluation of the kidneys from acromegalic cats has revealed thickening of the glomerular basement membrane and Bowman’s capsule, periglomerular fibrosis, and degeneration of the renal tubules. Lameness has also been noted in acromegalic cats due to an associated degenerative arthropathy and peripheral (diabetic) neuropathy.

Diagnosis of feline acromegaly starts with clinical suspicion based on a thorough history, signalment, and clinical signs. Many of the abnormalities in the complete blood count, serum chemistry, and urinalysis of affected cats reflect concurrent diabetes mellitus which stresses the need to carefully evaluate the patient’s clinical history. Common abnormalities associated with diabetes mellitus include hyperglycemia, increased liver enzymes (ALT, ALP), hypercholesterolemia, glucosuria, and isosthenuria. Additionally, as many acromegals present for uncontrolled diabetes mellitus, azotemia and ketonuria are also common. Other common findings include erythrocytosis due to anabolic effects of growth hormone and IGF-1 and proteinuria secondary to glomerulonephropathy. Unexplained hyperphosphatemia and hyperglobulinemia have also been noted.

Measurement of serum growth hormone is commonly used to diagnose acromegaly in humans, however, assays specifically for feline growth hormone are not widely available. An assay using ovine GH as the antigen has been validated for use in cats, but is only available in Europe at this time.
However, even if an assay was available, growth hormone concentrations alone may not be a reliable diagnostic test for acromegaly as growth hormone production is cyclic and levels may vary throughout the day. As a result of this, relying on a single growth hormone measurement can be misleading. Additionally, it has been shown that growth hormone may be elevated in non-acromegalic diabetic cats. This elevation in growth hormone may be due to the fact that portal insulin is required for the liver to produce IGF-1. In diabetics that are being treated with insulin subcutaneously, portal insulin concentrations will remain low resulting in decreased IGF-1 production and theoretically decreased inhibition of GH release. In addition, growth hormone levels may not be elevated early in the course of the disease, but later increase significantly.

Serum IGF-1 is the most commonly used test to diagnose feline acromegaly and is readily available in the United States. Unlike growth hormone, IGF-1 concentrations are less likely to fluctuate over the course of the day as the majority of IGF-1 is protein bound giving it a longer half-life in the body. In addition, insulin like growth factor-1 increases in response to chronically elevated growth hormone concentrations and is thought to be a reflection of growth hormone levels over the last 24 hours. A recent study evaluating IGF-1 levels in confirmed acromegalics diabetics, diabetics, and healthy cats found that acromegalic diabetics had significantly higher levels of IGF-1 than diabetics and non-diabetics. This study concluded that using serum IGF-1 concentration for diagnosing feline acromegaly is 84% sensitive and 92% specific. However, just as with growth hormone, elevations in IGF-1 concentration alone may not be diagnostic for acromegaly. One study found that IGF-1 levels in non-acromegalic diabetic cats on long-term insulin treatment (>14 months) had higher levels of IGF-1 than non-diabetics. It was proposed that insulin treatment allowed for beta cell regeneration and increased portal insulin leading to elevations in IGF-1. In addition, another study revealed that untreated diabetic acromegalics can have low to normal IGF-1 concentrations that increase after starting insulin therapy. The results of this study indicate that retesting IGF-1 concentrations in patients with suspected acromegaly that had low or normal IGF-1 levels may be warranted a few weeks after starting insulin therapy or even after insulin dosage increases.

Radiographic findings associated with feline acromegaly are related to the hypertrophic or anabolic effects of excessive growth hormone. Hyperostosis of the calvarium, spondylosis of the spine, and protrusion of the mandible are common findings. Periosteal reaction, osteophyte production, soft tissue swelling, and collapse of joint spaces are signs associated with the degenerative arthropathy linked to feline acromegaly. Thoracic radiographs may reveal cardiomegaly and congestive heart failure. Non-specific signs such as abdominal organomegaly (hepatic, renal, and adrenal) may be revealed by abdominal radiographs and ultrasound.

Advanced imaging is needed to document the presence of a pituitary adenoma. Computed tomography (CT) and magnetic resonance imaging (MRI) are both useful for identifying pituitary masses. However, MRI is thought to be the more sensitive imaging modality. The presence of a pituitary tumor alone is not diagnostic for feline acromegaly as other functional tumors of the pituitary may also result in insulin resistant diabetes such as ACTH producing tumors in patients with Cushing’s disease. Conversely, the absence of a pituitary mass does not rule out acromegaly as there has been a reported case where a patient had a negative MRI but a pituitary mass was identified at necropsy and histopathology confirmed feline acromegaly.

Histopathology is needed for definitive diagnosis which makes ante-mortem diagnosis challenging. However, with advancements in surgical procedures such as the transsphenoidal hypophysectomy, surgical excisional biopsy is possible. The main histopathologic change associated with acromegaly is somatotrophic proliferation.

There is no single test for the diagnosis of feline acromegaly. Clinical suspicion based on a thorough history and physical examination is essential. As earlier stated a common presenting complaint for patients with acromegaly is insulin resistance with weight gain. Although rare, hyperadrenocorticism, can be mistaken for feline acromegaly as both of these diseases can be associated with insulin resistant diabetes mellitus (and associated clinical signs), a pituitary mass and bilateral adrenomegaly. As such, hyperadrenocorticism is an important differential to keep in mind should testing for feline acromegaly produce vague or unequivocal results.

**Primary Hyperaldosteronism**

Feline primary hyperaldosteronism is diagnosed based on clinical signs, serum biochemistry, plasma aldosterone concentration, adrenal imaging and histopathology of adrenal tissue.
Cats may present with blindness caused by systemic hypertension. Many will also present with weakness resulting from hypokalaemic polymyopathy. Elevated concentrations of plasma aldosterone and adrenocortical neoplasia have been documented in all cases. Seven cases had adrenal adenomas (unilateral in five and bilateral in two) and six had unilateral adrenal carcinomas. Three cases underwent medical treatment only with amlodipine, spironolactone and potassium gluconate; two cases survived for 304 and 984 days until they were euthanized because of chronic renal failure, while the third case was euthanized at 50 days following failure of the owner to medicate the cat. Ten cases underwent surgical adrenalectomy following a successful stabilization period on medical management. Five cases remain alive at the time of writing with follow-up periods of between 240 and 1803 days. Three cases were euthanized during or immediately following surgery because of surgical-induced hemorrhage. One cat was euthanized 14 days after surgery because of generalized sepsis, whilst the remaining cat was euthanized 1045 days after surgery because of anorexia and the development of a cranial abdominal mass. It is recommended that primary hyperaldosteronism should be considered as a differential diagnosis in middle-aged and older cats with hypokalaemic polymyopathy and/or systemic hypertension and this disease should no longer be considered a rare condition.

In recent years, there has been renewed interest in primary hyperaldosteronism, particularly because of its possible role in the progression of kidney disease. While most studies have concerned humans and experimental animal models, a recent paper highlighted the occurrence of a spontaneous form of (non-tumorous) primary hyperaldosteronism in cats. At presentation, the main physical features of 11 elderly cats were hypokalemic paroxysmal flaccid paresis and loss of vision due to retinal detachment with hemorrhages. Primary hyperaldosteronism was diagnosed on the basis of plasma concentrations of aldosterone (PAC) and plasma renin activity (PRA), and the calculation of the PAC:PRA ratio. In all animals, PACs were at the upper end or higher than the reference range. The PRAs were at the lower end of the reference range, and the PAC:PRA ratios exceeded the reference range. Diagnostic imaging by ultrasonography and computed tomography revealed no or only very minor changes in the adrenals compatible with nodular hyperplasia. Adrenal gland histopathology revealed extensive micronodular hyperplasia extending from zona glomerulosa into the zona fasciculata and reticularis. In three cats, plasma urea and creatinine concentrations were normal when hyperaldosteronism was diagnosed but thereafter increased to above the upper limit of the respective reference range. In the other eight cats, urea and creatinine concentrations were raised at first examination and gradually further increased. Even in end-stage renal insufficiency, there was a tendency to hypophosphatemia rather than to hyperphosphatemia. The histopathological changes in the kidneys mimicked those of humans with hyperaldosteronism: hyaline arteriolar sclerosis, glomerular sclerosis, tubular atrophy and interstitial fibrosis. The non-tumorous form of primary hyperaldosteronism in cats has many similarities with "idiopathic" primary hyperaldosteronism in humans. The condition is associated with progressive renal disease, which may in part be due to the often incompletely suppressed plasma renin activity.
Hyperthyroidism in Cats: Diagnosis and Treatment
David Bruyette, DVM, DACVIM

Hyperthyroidism is recognized as the most common endocrinopathy of older cats. Despite worldwide occurrence, the pathogenesis of feline hyperthyroidism remains unclear. Traditional methods of managing feline hyperthyroidism include thyroidectomy, anti-thyroid medications, and radioactive iodine. Recent studies document that another option now exists for hyperthyroid cats; feeding a limited-iodine food normalizes thyroid hormone concentrations and alleviates clinical signs of hyperthyroidism. Surgery and radioactive iodine are designed to provide permanent solutions, whereas, oral anti-thyroid drugs and nutritional management control hyperthyroidism and are needed daily to achieve/maintain their effect. All management options are effective and each has its pros and cons. It’s important to discuss all options with pet owners so the appropriate management can be selected for each hyperthyroid cat.

Diagnosis
Diagnosis most often is based on the presence of one or more typical clinical signs and increased serum total thyroxine (T₄) concentration. However, up to 10% of all hyperthyroid cats and 40% of those with mild disease have serum T₄ values within reference range. The diagnosis of hyperthyroidism should not be excluded on the basis of a single normal serum T₄ value, especially in a cat with typical clinical signs, a palpable thyroid nodule and serum T₄ in the upper half of the normal range. In these cases, serum free T₄ (fT₄), measured by equilibrium dialysis, may provide an alternative means of diagnosing hyperthyroidism in cats with normal serum total T₄ values. Studies document that up to 20% of sick euthyroid cats can have increased fT₄ concentration. Therefore, it is most appropriate and reliable to interpret the two values together. Mid-to-high reference range total T₄ and increased fT₄ concentration is consistent with hyperthyroidism. In contrast, low total T₄ and increased fT₄ values are usually associated with non-thyroidal illness.

Management Options
Once hyperthyroidism has been diagnosed, all management options (thyroidectomy, radioactive iodine, anti-thyroid drugs, nutritional management) should be discussed with pet owners. All options can be ≥ 90% effective for controlling hyperthyroidism when used appropriately. The selected management option will differ for each cat based on several considerations (Table 1). Radioactive iodine therapy is considered the gold standard for treatment of hyperthyroidism; however, most pet owners currently opt for medical management. Until recently, this included oral or transdermal anti-thyroid drugs. Now nutritional management using a limited-iodine food is another option for cats with hyperthyroidism.

Radioiodine Iodine
Radioiodine treatment is often considered the best option for many hyperthyroid cats because:
- It has the potential to eliminate a benign thyroid tumor or abnormal thyroid tissue with a single treatment
- It treats extra-thyroidal thyroid tissue, which may occur in 10 to 20% of hyperthyroid cats
- No general anesthesia is required
- Reported side effects are minimal

Cats should be stable prior to radioiodine therapy; those with clinically significant cardiovascular, renal, gastrointestinal, or endocrine (e.g., diabetes mellitus) disease may not be very good candidates, especially because of the time necessary for boarding after treatment.

After administration, radioactive iodine is actively concentrated by the thyroid gland and has a half-life of 8 days. It emits both β-particles and γ-radiation; the β-particles are responsible for the majority of tissue destruction, but are only locally destructive, traveling a maximum of 2 mm. Therefore, no significant damage to adjacent parathyroid tissue, atrophied thyroid tissue, or other cervical structures is expected. The main limitation to widespread use of radioactive iodine is the requirement for special licensing and the isolation of the cat for variable periods after treatment. This can range from several days to several weeks depending on state or local radiation regulations and the dose administered.

The goal of treatment is to restore euthyroidism with the smallest possible single dose of radioactive iodine, while avoiding development of hypothyroidism. Controversy exists as to the best method of calculating the optimum dose for individual cats.
Based on the majority of reported cases, post-treatment hypothyroidism is transient and generally uncommon (2 to 7% of cases); even fewer cats have clinical signs or appear to require thyroid hormone replacement. However, up to 30% (50 of 165 cats) were hypothyroid 3 months after radioactive iodine therapy in one study; of these, 56% (19 of 34 hypothyroid cats with available information) had clinical signs of hypothyroidism and 52% (23 of 44 cats) were given thyroid hormone supplementation. Thyroid hormone replacement may be needed in some cats, especially those with concurrent kidney disease, since hypothyroidism has been associated with azotemia and decreased survival time in previously hyperthyroid cats. Owners should be advised of this possibility, particularly if their motivation is to avoid long-term oral medication.

**Anti-Thyroid Drugs**

Anti-thyroid drugs (e.g., methimazole, carbimazole) are commonly used for treatment of hyperthyroidism in cats. If administered appropriately, they reliably inhibit the synthesis of thyroid hormones and thereby lower serum thyroid hormone concentrations. These drugs do not affect the thyroid gland’s ability to trap inorganic iodide or release preformed hormones. They are widely recommended to stabilize hyperthyroid cats prior to surgery and are the only drugs that can be used chronically for management of hyperthyroidism. Almost all cats are potential candidates unless thyroid carcinoma is suspected. Anti-thyroid drugs used most often in cats include methimazole and carbimazole; both can be given orally or formulated for transdermal application. Custom formulation of transdermal products may increase expense of therapy and stability of the product is not guaranteed.

Results of a recent prospective study conducted in New Zealand showed that once daily treatment for 12 weeks with transdermal methimazole in a novel lipophilic vehicle was as effective as twice-daily carbimazole administered orally.

While many cats have been successfully managed long-term with anti-thyroid drugs, it’s important to monitor for potential side effects that have been associated with their use. In the study with the largest number of cats, 18% had side effects associated with methimazole; a more recent study revealed that 44% of 39 cats had side effects. In 44 cats receiving carbimazole for 1 year, 44% had associated side effects with gastrointestinal signs (decreased appetite, vomiting, diarrhea) being most common. In another study, 13% of 39 cats treated with carbimazole experienced side effects. It’s difficult to determine what % of side effects are caused by the drug versus something else such as concurrent disease.

Most adverse reactions occur within the first few weeks to months after beginning therapy and include depression, inappetence, vomiting, and self-induced excoriations of the head and neck (facial pruritus). Gastrointestinal signs are less common with transdermal administration of methimazole. Mild to serious hematological complications, including agranulocytosis and thrombocytopenia either alone or concurrently, and more rarely immune-mediated hemolytic anemia may also occur. Hepatic toxicity with marked increases in bilirubin concentration and hepatic enzyme activities has been described in less than 2% of cats treated with methimazole. Cessation of therapy is required if either serious hematologic or hepatic reactions develop. Serum antinuclear antibodies develop in approximately 50% of cats treated with methimazole for longer than 6 months, usually in cats on high-dose therapy (> 15 mg/day). Although clinical signs of a lupus-like syndrome have not been reported, decreasing the daily dosage is recommended.

**Nutritional Management**

Production of thyroid hormone requires uptake by the thyroid gland of sufficient amounts of iodine, which is provided by dietary intake. The only function for ingested iodine is for thyroid hormone synthesis. This observation led to the hypothesis that limiting dietary iodine intake could be used to control thyroid hormone production and potentially manage hyperthyroidism in cats. After more than a decade of research and development, a limited-iodine therapeutic food (Hill's® Prescription Diet® y/d™ Feline) containing < 0.3 ppm (mg/kg) iodine on a dry matter basis (DMB), is now available as an option for managing cats with hyperthyroidism.
**Iodine Content of Commercial Cat Foods**

Iodine occurs naturally in many ingredients typically used in the manufacture of commercial pet foods (particularly fish, shellfish and fresh meats) and unless steps are taken to strictly control the iodine content of ingredients, the final iodine concentration in pet foods varies widely.

Commercial cat foods in New Zealand had iodine amounts ranging from 0.19 to 21.2 ppm in one study whereas in Germany a range of 0.22 to 6.4 ppm was reported. Evaluation of 28 canned cat foods in the US revealed an iodine content ranging from 1.09 to 52.3 ppm (mean = 7.83) and 14 dry cat foods contained iodine amounts ranging from 1.34 to 5.94 ppm (mean = 2.77). Based on these studies, the amount of iodine is much higher in many canned foods compared with dry foods and variability of iodine content is much greater in canned food.

Multiple feeding trials have been conducted in a research colony using over 100 cats with naturally occurring hyperthyroidism to determine the safety and effectiveness of limited dietary iodine in the management of the disease. The results of all studies support that a therapeutic food with dietary iodine ≤ 0.3 ppm iodine (dry matter basis) provides a safe and effective management option for cats with naturally occurring hyperthyroidism. Serum total thyroxine concentrations return to the normal range within 4 to 12 weeks of initiating nutritional management and 90% hyperthyroid cats maintained on the limited-iodine food as the sole source of nutrition become euthyroid.

Three studies were designed to determine the magnitude of iodine control necessary to return newly diagnosed cats to a euthyroid state; the maximum level of dietary iodine that maintains cats in a euthyroid state; and the effectiveness of a therapeutic food formulated based on the previous studies to control naturally occurring hyperthyroidism in cats. In summary, results of these studies demonstrated that a food with 0.17 or 0.32 ppm iodine (DMB) maintained normal thyroid hormone concentrations in hyperthyroid cats, helping to further define the range of iodine effective for managing hyperthyroidism.

We have treated 22 cats to date with feline y/d with follow-up data for at least 6 months. All of the cats found at least one form of the diet (dry or canned) to be palatable. Nineteen of 22 (86%) cats experienced clinical improvement with normalization of their TT4 concentrations. Of the three cats that failed to achieve remission, 2 cats were discovered to be eating foods other than y/d and when the owners switched them to y/d exclusively remission of hyperthyroidism was achieved. One cat (5%) failed to respond to dietary therapy and was subsequently treated with 131-I.

We are currently conducting a prospective study evaluating the efficacy of feline y/d in managing feline hyperthyroidism to include monitoring of thyroid function (TT4, fT4ED, TSH), clinical signs, body weight, renal function and blood pressure pre and post-treatment. The study should be completed in 2015.

**Newly Diagnosed Patients**

After confirming the diagnosis and performing a thorough patient evaluation, nutritional management should be discussed along with other options for managing hyperthyroidism. If selected as the management option, gradual transition to the limited-iodine food (Hill’s® Prescription Diet® y/d™ Feline) over at least 7 days is recommended. It is very important to counsel owners so they understand that success of nutritional management depends on the limited-iodine food being the sole source of nutrition for their cat.

The first recheck evaluation should be done 4 weeks after completing the transition to y/d Feline (i.e., once the cat has eaten y/d exclusively for 4 weeks) and as a minimum should include physical examination and measurement of T4, BUN, serum creatinine, and urine specific gravity. All cats should have decreased T4 concentrations compared with baseline and many will have returned to normal by the 4-week evaluation. Clinical improvement including weight gain, improved hair coat and decreased tachycardia/cardiac murmur also may be noted by the first evaluation. Clinical signs should continue improving by the next re-evaluation at 8 weeks and most cats will be euthyroid. Some cats require slightly longer to become euthyroid; however, it’s expected that 90% will have normal T4 concentrations if the limited-iodine food is their sole source of nutrition. If euthyroidism is not achieved within 4 to 12 weeks, a thorough history is indicated to confirm that only the limited-iodine food is being fed.
Managing Hyperthyroid Cats with Concurrent Kidney Disease

Chronic kidney disease (CKD) and hyperthyroidism are more likely to be diagnosed in older cats so it’s not surprising that many hyperthyroid cats have CKD. Untreated hyperthyroidism complicates the diagnosis of CKD because it’s associated with increased glomerular filtration rate (GFR) and therefore often masks biochemical markers of CKD. Regardless of the therapeutic modality (methimazole, surgical thyroidectomy, or radioiodine), decreased GFR, increased serum urea and creatinine concentrations and development of overt clinical signs of kidney disease have been reported after successful treatment of hyperthyroidism. The presence of underlying CKD may affect the prognosis - one study documented a shorter survival time in hyperthyroid cats with azotemia. However, two recent studies comparing survival of cats that developed azotemia with those that did not after treatment of hyperthyroidism found no significant difference between the two groups if cats did not become hypothyroid post-treatment.

The reported occurrence of azotemia after treatment of hyperthyroidism ranges from 15 to 49%. Iatrogenic hypothyroidism has been reported to decrease GFR in human patients. Post-treatment iatrogenic hypothyroidism has been reported in cats after radioiodine therapy and bilateral thyroidectomy, which constituted the predominant therapeutic modalities in previous studies. In one recent study, cats with iatrogenic biochemical hypothyroidism were almost twice as likely to develop azotemia post-treatment as euthyroid cats. The hypothyroid cats with azotemia had shorter survival times than cats without azotemia, whereas, consistent with previous reports, there was no difference in survival times of euthyroid cats with or without azotemia.

It’s not possible to consistently predict which cats will develop overt CKD after treatment of hyperthyroidism or have progression of their kidney disease. This should be considered when deciding on treatment options, particularly those that are irreversible (thyroidectomy, radioactive iodine). Regardless of the option selected for managing hyperthyroidism, it’s important to remember that the only intervention shown to improve quality of life and prolong survival time in cats with naturally occurring CKD is feeding a therapeutic renal food. Until recent availability of limited-iodine food, nutritional recommendations have not generally been considered for hyperthyroid cats without azotemia. In cats with compromised renal function, but without azotemia (IRIS Stage 1), the decrease in GFR associated with normalizing serum T4 levels may be sufficient to prevent effective clearing of protein metabolic by-products (BUN and creatinine) when dietary intake of protein and phosphorus is high. This could contribute to the occurrence of post-therapy azotemia in hyperthyroid cats.

In our work with 22 cats with hyperthyroidism treated with feline y/d, 4/22 cats (18%) were azotemic (IRIS Stage 1 and 2 CKD) prior to starting the diet. All 4 cats experienced normalization of their BUN and creatinine within 30-150 days along with normalization of their TT4’s. One potential explanation is that the expected decrease in GFR associated with normalizing serum T4 may be offset by the nutrient profile of the limited-iodine food which is similar foods for mature adult cats or cats with early CKD. Additional study is needed to better understand the effects of using limited-iodine food on hyperthyroid cats with concurrent kidney disease.

Conclusions/Summary

Hyperthyroidism is the most common endocrine disease of older cats worldwide. While the pathogenesis is unclear, several effective management options are available. All should be discussed with pet owners, including pros/cons, so that the best option can be selected for individual patients and their owners. Feeding a limited-iodine food is now available as an option for effective management of hyperthyroid patients. When fed as the sole source of nutrition, approximately 90% of hyperthyroid cats become euthyroid within 4 to 12 weeks. To date, over 150 cats with naturally occurring hyperthyroidism have been managed successfully by feeding a limited-iodine food, most for 2-3 years and some cats for as long as 6 years.
Table 1. Advantages/disadvantages of options for managing cats with hyperthyroidism

<table>
<thead>
<tr>
<th>Option</th>
<th>Advantages</th>
<th>Disadvantages</th>
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<tbody>
<tr>
<td>Thyroidectomy</td>
<td>Cures current tumor</td>
<td>High initial costs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Requires anesthesia</td>
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<td></td>
<td></td>
<td>Hospitalization required</td>
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<tr>
<td></td>
<td></td>
<td>Risk of post-operative hypocalcaemia</td>
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<tr>
<td></td>
<td></td>
<td>Irreversible*</td>
</tr>
<tr>
<td>Radioactive Iodine</td>
<td>Cures current tumor</td>
<td>High initial costs</td>
</tr>
<tr>
<td></td>
<td>Single treatment</td>
<td>Limited availability</td>
</tr>
<tr>
<td></td>
<td>Effective for ectopic tissue</td>
<td>Hospitalization required</td>
</tr>
<tr>
<td></td>
<td>Side effects uncommon</td>
<td>Irreversible*</td>
</tr>
<tr>
<td>Anti-Thyroid Drugs</td>
<td>Routinely available</td>
<td>Not curative (controls T₄ and signs)</td>
</tr>
<tr>
<td></td>
<td>Reversible</td>
<td>Daily administration needed</td>
</tr>
<tr>
<td></td>
<td>Costs spread over time</td>
<td>Drug side effects</td>
</tr>
<tr>
<td>Limited-Iodine Food</td>
<td>Routinely available</td>
<td>Not curative (controls T₄ and signs)</td>
</tr>
<tr>
<td></td>
<td>Reversible</td>
<td>Cat can only eat a single food</td>
</tr>
<tr>
<td></td>
<td>Costs spread over time</td>
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*Important consideration due to potential for worsening renal function in cats with kidney disease
ASSESSING AND MANAGING PAIN IN EXOTIC COMPANION MAMMALS

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Introduction
Small mammals in this document refers to exotic animal species commonly kept as pets such as rodents (rats, mice, chinchillas, guinea pigs), rabbits, and ferrets. A part of managing pain in an animal also includes managing stress in that patient, including making sure they are well hydrated, have normal GI sounds, are at a comfortable temperature and humidity, they have familiar and favorite foods available, any concurrent diseases are being treated, non-stressful sounds and smells, appropriate hiding place, appropriate dry and clean substrate is available, handling is kept to a minimum, and the patient is weighed daily.

Research
Because of the advanced field of laboratory animal medicine, there is more scientific information available regarding analgesia in small mammals than what is known about birds and reptiles combined. Remember that there is no generic small mammal. Some differences in drug metabolism that are known include: after tramadol IV or PO rabbits make over 5 different metabolites and levels of the active drug drops precipitously after just a few minutes, rabbits can seizure and die after being given fipronyl (Frontline®), and rabbits’ GI flora is disrupted by certain antibiotics including oral penicillins, macrolides and cephalosporins. There are probably some unknown differences in metabolism within each species and, as some researchers are finding out, there are even subtle differences between strains.

The most is known about the rat, including published studies of pharmacokinetics, pharmacodynamics (using the tail flick test) and established, standard methods of assessing pain. Remember that laboratory animals are usually young, healthy, and all one strain when tested, and these doses may be too high for a sick patient in your hospital, especially if used in combination with other drugs. A study was performed determining the magnitude and duration of analgesia using the standard hot plate test and the tail flick test, showing (n=61 ea. spp.). In this study, butorphanol SC (2.0 mg/kg for rat; 5.0 mg/kg for mouse) provided mild pain relief for the shortest amount of time (1-2 hours) compared to morphine SC (10 mg/kg in wither rat or mouse) that provided intermediate pain relief for the longest amount of time (6-8 hours for rat; 3-5 hours for mouse) and buprenorphine SC (0.5 mg/kg rat; 2.0 mg/kg mouse) provided the highest degree of pain relief for an intermediate amount of time (2-3 hours).

Assessing Pain
It is very difficult to assess pain in small mammals. They seem very stoic and do not cry out in pain unless they are in extreme pain. Small mammals are prey animals, so they do not want to be conspicuous if they are in pain or injured. So, try to observe your patient before they are aware you are observing them. Dr. Flecknell has available a training video with an objective scoring system to evaluate pain in rats. By counting the number of times a rat “flinches” the skin on its back, stretches like a cat or falters in its gait during a set period of time, a numerical score is assigned as to how much pain that rat is experiencing. It is my opinion that all practitioners should learn how to assess pain in their rat patients from this video. There is much laboratory animal medicine information available in the literature and from colleagues. Small mammal practitioners would do well to consult and use this large body of information. Less is known about objectively assessing pain in the mouse and rabbit, but recently a facial grimace scale has been developed for both these species (and the rat) and practitioners should familiarize themselves with these signs of pain: half closed eyes (orbital tightening), pointed or bunched up nose, and whiskers pulled back.

Opioids
Opioids include full agonists such as morphine, oxymorphone, hydromorphone, fentanyl, and alfentanil, and partial agonists such as buprenorphine, and agonist/antagonists such as butorphanol. Tramadol is a weak μ opioid agonist, but can have significant analgesic efficacy due to its inhibition of the reuptake of norepinephrine and serotonin.

Advantages of opioids are potent pain relief, well studied in rats, and choices available of varying levels or types of pain relief. Disadvantages of opioids are respiratory depression, nausea, GI Stasis, sedation, and pica with buprenorphine in rats. In rabbits, it is difficult to evaluate if the pain or the analgesic being given is causing the GI stasis. Care should be taken to not give too much opioid and cause too much sedation, or induce too much respiratory depression, because of the possible inability to quickly intubate small mammals.

Overall, butorphanol is recommended for mild pain of short duration, morphine for mild to moderate pain of increased duration, and buprenorphine for severe pain of short to medium duration. Remember to always reassess the patient to determine degree of pain, degree of sedation, and maintenance of normal GI motility. A survey of diplomats of the American College of Laboratory Animal Medicine showed opioids as the analgesic used most often for relief of pain in biomedical studies.

Unfortunately for rabbits, a PK study showed that after oral administration (11 mg/kg) tramadol and its major active metabolite, M1, decreased very quickly to what is considered non-therapeutic levels in a human. (Souza) In addition, an ISOMAC study (evaluating the isoflurane sparing effects of tramadol) using tramadol IV (4.4 mg/kg) there was a significant, but clinically unimportant, reduction of ISOMAC. Based in this PK study and the ISOMAC study, tramadol should not be used alone in rabbits for pain relief despite anecdotal reports to the contrary. Another note on rabbits and opioids: when using fentanyl patches watch for quick (24h) hair regrowth impeding absorption.

I use butorphanol at 0.2 – 0.3 mg/kg SC in most rabbits for mild pain of short duration and I use buprenorphine at 0.02-0.05 mg/kg SC in most rabbits for moderate to severe pain of longer duration. I use less if the animal is ill or is sensitive to the drug.

NSAIDS
Non-steroidal anti-inflammatory drugs (NSAIDs) inhibit cyclooxygenase (COX) enzymes that help produce prostaglandins which induce inflammation. Two isoforms are of COX (COX-1 and COX-2) are generally targeted. NSAIDS include meloxicam, ketoprofen, carprofen and flunixin meglumine.

Pros – good for mild pain and provides anti-inflammatory action as well
Cons – gastric upset, renal, may lower blood pressure under anesthesia???
Recommendations – meloxicam currently seems to be the most commonly used of all the NSAIDs. I tend to use a dose of 0.2-0.3 mg/kg in most ECM’s ; less if they are ill, and maybe not at all if they have renal disease. With any NSAID used long term (>5 days), I use a gastric protectant like omeprazole, famotidine, or sucralfate, and SQ or IV fluids, and check BUN and creatinine especially in geriatric animals (most rats get chronic progressive nephropathy starting at 6 months of age and progressing to significant loss by 1.5 years of age).

Local analgesics
Local analgesics include various routes such as topical, local infiltration, or nerve block (peripheral or as an epidural). The literature has many descriptions of how to place epidural catheters in various laboratory animals including guinea pigs, rabbits and ferrets, using a variety of analgesics. A study in pregnant guinea pigs described placing 27 gauge epidural catheter in the L3-4 intervertebral disc space and administering bupivacaine. Histopathological examination afterwards showed most catheters were properly placed, but some were inadvertently placed in the subdural space resulting in severe depression, and even penetrated the spinal cord. Several articles give detailed descriptions of anatomy and epidural catheter placement and use in ferrets. One study used morphine at 0.1 mg/kg via epidural for OHE and showed attenuation of pain compared to saline epidural. Epidural use in rabbits has been described using 0.1 mg/kg morphine, with care (Barter VCNA)

Note – The low therapeutic index of local analgesics is a good reason to calculate the dose every time prior to use. Dr. Darryl Heard in the Ferrets, Rabbits, and Rodents book states, “For example, if the toxic dose of lidocaine in a rabbit were 10 to 20 mg/kg, the equivalent volume of a 2% lidocaine solution would be 0.5 to 1.0 mL/kg.” That is a very small volume! What we do in the clinic is calculate the dose, dilute 1:2 or 1:10 with saline and then administer no more than what we drew up in the syringe.

Pros – few side effects, preemptive, does not affect gut motility
Cons – narrow therapeutic index when consider comparably small size of animal
Recommendation – use more frequently

New methods of providing local pain relief are currently being studied. A recent study showed better pain relief in ferrets undergoing acute, experimentally induced hernia repair with bupivacaine impregnated SIS (small intestinal submucosa) incorporated for repair compared to those whose hernias were repaired with non-impregnated SIS. Also, there is bupivacaine impregnated foam infiltrated into acute hernia incisions in rabbits that showed no adverse effects at necropsy that may be used in the future.

References
OHE, OVE, and Castration of Exotic Companion Mammals

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Introduction

Exotic companion mammals can include rodents (rats, mice, chinchillas, guinea pigs and prairie dogs), rabbits, ferrets, and other (sugar gliders, hedgehogs, kinkajous, etc.). The term “spay” as a verb refers to an ovariohysterectomy; removing the uterus and ovaries in a female. In rabbits oftentimes an ovariohysterovaginectomy is performed; removing the uterus, ovaries, and part of the vagina. Alternatively, an ovariectomy can be performed; removing just the ovaries. The term “neuter” as a verb refers to removing the testicles in a male, but the term is also used to refer to removing the uterus and/or ovaries in a female, and as an adjective refers to one that has had the sexual organs removed. Synonyms for neuter include to sterilize, castrate, spay, geld, fix, desex, alter, doctor, or emasculate. The term “castrate” can be a verb (to remove the testicles) or a noun (referring to one whose testicles have been removed). Vasectomy refers to the surgical cutting and sealing of part of each vas deferens, typically as a means of sterilization.

Castration: Anatomy and Reproductive Surgery by Species

Rabbits
Rabbits have external testicles and each lies with a separate hemiscrotal sac that is cranial to the penis, which is not typical for placental mammals. Rabbits have an open inguinal ring, therefore the testicles can move between the scrotum and the abdomen freely. The testicle is elongated in shape with peritesticular fat and a prominent epididymis. A closed technique with an incision in the scrotum over each testicle is typically used, or if an open technique is performed then care should be taken to close the inguinal ring to prevent herniation of abdominal contents into the hemiscrotum. A prescrotal incision can be performed since the penis is caudal to the scrotum, and is usually an open castration and closing the inguinal rings. There are descriptions in the literature of an open technique without closing the inguinal ring, citing that the fat pad will prevent herniation of abdominal contents into the scrotum, but this author disagrees with taking that risk. The scrotal incisional tissue can simply be re-apposed (author preference), or tissue glue can be applied, or suture can be used (subcuticular, simple interrupted, or simple continuous). Suture may be associated with a suture reaction, or the animal may be irritated by it and chew the area. If a prescrotal incision is done, then suture is necessary. Rabbit testicles descend at about 12 weeks of age.

Guinea pigs
Guinea pigs have external testicles that lie within the perianal sac very near to and cranial to the anus, but due to the open inguinal ring the testes can easily move into the abdomen. The penis is cranial to the testicles. Because of the open inguinal ring, a closed castration technique is done with an incision over each perianal sac, or if an open technique is performed, then care should be taken to close the inguinal ring. Alternatively, a prescrotal approach using two separate incisions, or an abdominal castration can be performed. Guinea pigs have a well-developed seminal vesicle that is about 10 cm long, and should not be confused for a uterus if performing an intraabdominal incision. This author has first-hand experience of three separate guinea pig cases that had been castrated elsewhere and presented to our hospital on emergency with herniation of intestine into the scrotum, with subsequent peritonitis, sepsis, and death in all cases because an open castration technique had been performed without closure of the inguinal ring. The author prefers to cover the anus with a sterile adhesive drape to prevent inadvertent contamination of the sterile surgical field. The scrotal incisional tissue can simply be re-apposed and tissue glue applied, or suture can be used (subcuticular is preferred, but simple interrupted, or simple continuous can also be used), Some animals may experience a suture reaction, they may be irritated by the suture, or chew the
area. The author prefers to close the guinea pig scrotal skin with a subcuticular (aka interdermal) continuous suture with 4-0 polydioxanone, the least reactive suture material. Due to the possibility of post-operative abscesses in guinea pigs due to *Leptotrichia* sp. anaerobic bacteria, the author may give chloramphenicol for approximately 5 days post-operatively.

Chinchillas and Prairie Dogs
Technically chinchillas do not have a scrotum therefore their testes lie within the inguinal canal or abdomen. Therefore, an abdominal or prescrotal incision is recommended. Prairie dogs less than 2 years of age will have intra-abdominal testes and even if older than two may have testes descended only during the breeding season. Therefore, prairie dogs commonly require an abdominal incision for castration. Otherwise, if the testes have descended then, a prescrotal incision is preferred.

Rodents
Rodents such as mice, rats, gerbils, and hamsters have relatively large testicles surrounded by fat that lie within a scrotum. The epididymis is also large. A prescrotal, or bilateral scrotal, incision can be performed. Because of the comparatively large size of the incision the author prefers to close the incision with suture in a subcuticular pattern. Castration techniques include scrotal, prescrotal, or abdominal approaches.

Hedgehogs and Kinkajous
Since technically hedghogs do not have a scrotum and their testes lie just under the perianal skin, an abdominal castration is usually performed. Kinkajous are similar to a dog castration using either open or closed technique.

Ferrets
Castration in ferrets is performed to prevent unwanted breeding, fighting, and to decrease the very strong musky smell. Descending alone does not deter the musky smell since it is hormonally driven and comes from all skin glands. Most ferrets are sold already spayed and castrated, and descended signified by two dot tattoos in the right ear. Castration is performed similarly to a dog castration in an open fashion. The testicles are similar to a cat, and the penis is similar to a dog with a ventrally placed prepuce with an os penis. One study showed that 2.5 years after spay or castration of a ferret the incidence of adrenal disease greatly increases.

Sugar gliders
Castration of sugar gliders is performed to prevent breeding, decrease male smell, or to reduce social tension. Sugar gliders are marsupials with anatomy that is different than other mammals. The pendulous scrotum is cranial to the penis. A scrotal or prescrotal incision can be made. The prescrotal technique is performed by either transecting the skin and spermatic cord between the body wall and the scrotum at the same time with bipolar electrocautery or CO2 laser, or the skin alone can be transected in the same place, the skin retracted proximally, and the spermatic cord ligated, and then the skin brought back distally over the ligated stump. Both techniques can be finished with a drop of tissue glue. It is recommended to leave very little residual skin so the sugar glider is not as tempted self-mutilate. The penis is forked in sugar gliders with the urethral opening at the base of the fork. To prevent self-mutilation in sugar gliders, our practice uses acepromazine to sedate them post-operatively.

Ovariohysterectomy (OHE, OHVE, and OVE)
Rabbits - Ovariohysterectomy of female rabbits is recommended before two years of age to prevent the incidence of uterine adenocarcinoma. Besides preventing uterine neoplasia, reasons to perform an OHE include to prevent pyometra, cystic endometrial hyperplasia, pseudopregnancy, breeding, urine spraying, hormone related aggression especially with other female rabbits, and the rare post-parturient urinary bladder prolapse. Rabbits are induced ovulators, like ferrets. Rabbits, rats, and mice have a duplex reproductive anatomy with a double cervix and a relatively long vagina. Rabbits have two cervices, therefore the proximal ligature can be at the level of the distal vagina, meaning just proximal to both cervices (ovariovaginohysterectomy). Care should be taken not to traumatize the nearby thin walled
cecum, and to also avoid the nearby ureters. There is also a thick (1 cm sometimes), fat filled broad ligament (mesometrium) making visualization of uterine vessels difficult, especially in overweight rabbits. Remember that rabbits are prone to adhesions, so flush any blood from the abdomen. Some advocate performing an OVE in young rabbits, but due to the high incidence of uterine adenocarcinoma in the rabbit this author still performs OHE. There are varying reports from Europe regarding OVE only in rabbits and the risk of adenocarcinoma, some say if the rabbit is less than 1 year of age there is no risk of uterine adenocarcinoma while others are saying no, they are seeing adenocarcinoma in rabbits as young as 8 months of age.

Guinea pigs - OHE of female guinea pigs is similar to rabbits and is used to prevent the high incidence of cystic ovary(ies) at about 5 years of age. Currently, our practice prefers to perform ovariectomy (OVE) in guinea pigs since there is less trauma than a traditional spay and the most common problem in guinea pigs is cystic ovaries. Other rodent surgeries are similar to the guinea pig. OHE or OVE of pet female rodents is becoming more common, and is usually done to prevent reproduction in multiply housed animals, cystic ovaries, or to prevent neoplasia of reproductive or mammary tissue. The surgery is similar, albeit smaller, than any other OHE. In dorsal recumbancy, an approximate 6 cm or smaller incision is made from about 1 cm cranial to the umbilicus caudally to expose the uterus. Some prefer to use intracinsional lidocaine and/or bupivacaine. The ovarian pedicle is ligated as well as uterine vessels with either 4-0 PDS or medium metal clips. The uterus is ligated with 4-0 PDS. The linea is closed with 3-0 or 4-0 PDS in a simple continuous pattern, and the skin is closed with the same suture in a subcuticular pattern. It is recommended to perform an ovariecotmy in rats, guinea pigs and other rodents as long as the uterus is normal since an OVE is less traumatic than an OHE and the placement of the ovary in these animals is so dorsal. If performing an ovarioectomy only, then the ovaries can be accessed dorsally on either side of the spine just caudal to the last rib. I prefer two separate skin incisions. In most animals the incision is approximately one cm long, one cm caudal to the last rib, one cm ventral to the lateral vertebral processes made at a 45 degree angle to follow the angle of the last rib. The ovary is usually in the fat just in this incision. The ovarian pedicle is ligated and removed. Some veterinarians even remove some of the uterus as well. If a cystic ovary is present in a guinea pig, then the incision might need to be larger than one cm. Take care not to spill cyst contents as they may contain 3-10 more hormone than in plasma or serum. In older animals, a radiograph and ultrasound may be needed to determine if any concurrent uterine disease is present that might necessitate the uterus being removed via an OHE. The author commonly encounters uterine leiomyoma in older guinea pigs. If a guinea pig is bred for the first time after 8 months of age there is a very high risk dystocia because of pelvic bone fusing. If the guinea pig has been straining and is not responding to conservative treatment or the pubic symphysis is less than 2.5 cm then a C-section should be considered. Rats, mice, chinchillas, and degus have a duplex reproductive anatomy with a double cervix and a relatively long vagina. The OHE is similar to that performed in a rabbit. The author prefers OVE in rodents.

Ferrets - The OHE is similar to a cat. It is recommended to spay female ferrets to prevent prolonged heat induced aplastic anemia. Ferrets are induced ovulators and will stay in heat until bred. After approximately 1 month of being in heat hyperestrogenism occurs and leads to bone marrow suppression and aplastic anemia. By 3 months of being in heat a ferret can die.

Sugar gliders and other masupials - Sugar gliders are described as having an advanced duplex reproductive anatomy with two uteruses, two cervices, and two vaginas (a median and two lateral vaginas). They give birth through the median vaginal canal. The ovaries and uterine horns (OHE) can be removed with or without also removing the lateral vaginas (ovario-vaginal-hysterectomy). Care must be taken to not disturb or traumatize the ureters which pass from the ventral urinary bladder to dorsal towards the lateral vaginas. If the uterus and lateral vaginas are removed they are best removed in two
separate sections in marsupials in order to prevent trauma to the ureters; first, an ovariohysterectomy is performed and then a bilateral lateral vaginectomy. In marsupials, it is impossible to remove both the uterus and both lateral vaginas at the same time due to the position of the ureters. Do not remove the median vagina (aka central vaginal canal) as the ureters empty into this structure. The skin incision is on ventral midline through the pouch.

Hedgehog

Ovariohysterectomy is similar to the procedure in other mammals, although substantial fat surrounds the ovaries and mesosalpinx. Hedgehog females should be at least 6 months of age prior to OHE.
Feline maxillofacial trauma: Appropriate steps to make a difference

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Oral and maxillofacial (OMF) trauma in cats poses multiple challenges for the clinician. Primarily, understanding the complex oral and maxillofacial anatomy, the innate nature of the cat and the type of trauma are critical to formulating an appropriate treatment plan. Importantly, computed tomography and dental radiography is the cornerstone of diagnostic imaging for OMFS trauma. In addition, therapeutic approach is patient/situation specific and may require a specialized intervention. This lecture will describe the approach to multiple OMFS trauma situations in cats.

Diagnostic imaging of maxillofacial trauma in cats
The skull is a difficult area to study radiographically because the bone structure is very complex. In recent years, computed tomography (CT) has become more available to veterinarians and more affordable. A study performed at UC Davis prospectively compared the diagnostic yield and contribution of conventional radiographs and CT in cats with OMF. It was found that CT-images are superior to conventional radiographs both in an ability to identify the various anatomic components of the skull but also in the ability to detect OMF injuries. The average number of OMF injuries per cat by radiographs and CT-scan was 3.8 and 7.7, respectively. Separation of the mandibular symphysis, fractures of the pterygoid bones and fractures and dislocations involving the temporomandibular joint were particularly common in this species. CT allows for accurate assessment, diagnosis and treatment planning of OMF in cats. Contiguous thin slices, as well as a tridimensional reconstruction, are recommended.

Multidisciplinary approach to OMFS trauma in cats
The OMF region is rich in diverse functionality and tissue types. Tissues such as the brain, eyes, bones, teeth, for example, require special considerations. Therefore, OMF trauma may require the involvement of several specialists to optimize the outcome.

Management of common maxillofacial injuries in cats

Mandibular symphysis separation
Symphyseal separation is common in the cat. Most symphyseal separations can be managed by the very simple but effective cerclage wiring technique. Variations on the simple cerclage wire technique have been described for cases where incisors or canine teeth were avulsed in the traumatic incident.
Fractures of the body of the mandible
A wide variety of surgical and non-surgical methods have been described for the treatment of fractures of the body of the mandible in the cat.
Tape or nylon muzzle: A tape muzzle is very useful in the first aid treatment of mandibular fractures, and is also indicated for the definitive treatment of stable and minimally displaced fractures, especially in patients with a deciduous dentition. The use of a tape muzzle can be considered as means of additional support in cases where internal fixation did not achieve optimal stabilization. When using a tape muzzle in cats, an additional strip of tape from the dorsum of the nose over the frontal region is helpful. A commercially available nylon muzzle may be an acceptable substitute provided it fits snugly.
Maxillomandibular fixation: Composite bonding of the canine teeth is commonly used for long-term mouth closure. This method aims to achieve reduction and stabilization of the fracture fragments by restoring and maintaining the normal dental interlock. The advantages of this technique are that no further damage is caused to the teeth or other tissues of the oral cavity, the complications associated with tape muzzling are avoided, and the technique can be applied in animals with poor bone quality. MMF is non-invasive and may achieve good restoration of occlusion. However, once the MMF is placed, reduction of the fracture fragments is completely dependent on the MMF and may not achieve ideal bone union. The main disadvantages are that a rapid return to normal function is not achieved and that the caudal fragment may displace in a dorsomedial direction. Vomiting and postoperative respiratory complications are particularly hazardous.
Interdental wiring and intraoral composite splint: Interdental wiring and an intraoral composite splint is occasionally used in the cat and may be difficult because of the small size and conical shape of teeth of the cat.
Miniplates and screws: Bone plating is becoming more and more common as allows excellent fracture reduction and quick return to normal function. Maxillofacial mini plates and screws offer more versatility and have been used with success as neutralization or buttress fixation of mandibular, maxillary, and other maxillofacial fractures.

Fractures of the ramus of the mandible
Fractures of the mid-part of the ramus: Fractures of this part of the mandible are commonly seen in the cat. The surrounding muscle mass usually prevents gross displacement of the fragments and provides sufficient stabilization, in combination with MMF. The distal fragment may be displaced dorsomedially due to the muscle pull of the pterygoid muscles. Internal fixation is difficult due to the small size of the bones.
Fractures of the condylar process: Fractures involving the condylar process are commonly seen in the cat and are the most common TMJ disorder in cats. Fractures of the condylar process treated conservatively may heal by a bony union or as a pain-free and functional non-union. Conservative treatment of minimally-displaced sub-condylar and pericondylar fractures without joint surface involvement is therefore justifiable. Comminuted and intra-articular fractures may result in TMJ arthrosis and possible ankylosis but the occurrence if ankylosis is rare. TMJ ankylosis is characterized by a progressive inability to open the mouth.
We recommend no rushing into a condylectomy procedure and perform this surgery only if TMJ ankylosis is developing.

**Fractures of the facial bones and maxilla**

The principles of management of maxillary fractures are similar to mandibular fractures. Fractures of the facial bones and maxilla are usually not severely displaced and often do not require surgical repair, especially in younger animals. Digital re-alignment and, if indicated, soft tissue débridement and closure are adequate in most cases. A tape or nylon muzzle or MMF is useful in maintaining fragment alignment and preserving normal occlusion. Fractures involving the dental arch can be stabilized using an intra-oral composite splint, provided enough stable anchoring teeth are present mesial and distal to the fracture fragment(s). Severely comminuted and unstable fractures involving the lateral and dorsal part of the maxilla may require surgical intervention. Miniplates are very versatile and can easily be contoured over the maxillofacial bones.

**Midline palatal fracture / separation**

Isolated palatal fractures mainly occur as midline clefts often seen as part of the high-rise syndrome in cats. This injury can easily and effectively be managed by soft tissue débridement and approximating the displaced bony structures by gentle digital pressure, followed by primary closure of the torn palatal soft tissues in a simple interrupted pattern. If the separation of the interincisive suture is associated with considerable displacement, a cerclage wire may be placed interproximal between the third incisors and canine teeth.

**References**


Is your patient having difficulties in opening or closing the mouth?

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Introduction

Difficulties in opening or closing the mouth is a relatively common presenting complaint that may be the presenting feature of a broad array of disorders and organic diseases. In some patients, the disorder can be nondescript, chronic and without clinically identifiable source. These dogs are often diagnosed with ‘pain of unknown origin’ – a term that has unfortunately taken on the projective connotation and become a clinical diagnosis unto itself instead of one of exclusion. The differential diagnosis list for the clinical presentation of difficulties in 'opening/closing the mouth' or ‘pain of unknown origin’ is extensive. A class of disorders involving the TMJ exists and is generically termed TMD. The term TMD is used to describe problems and concerns associated with the TMJ that produce orofacial pain and mandibular dysfunction. TMD in dogs manifests as mandibular pain, difficulties in opening or closing the mouth, ‘head shyness’, vocalizing while yawning or eating, drooling and pawing at the face.

The TMJ is a synovial joint where the head of the mandible on the condylar process articulates with the mandibular fossa of the squamous part of the temporal bone. Unlike other synovial joints that are covered by hyaline cartilage, the TMJ is covered by a unique fibrocartilagenous layer. Furthermore, an articular fibrocartilaginous disc separates the mandibular fossa and the articular surface of the head of the mandible into dorsal and ventral compartments. Essentially, the disk fills the void between the condylar process and the mandibular fossa, promoting congruity of the joint. A lateral ligament strengthens the lateral aspect of the joint capsule. It is important to emphasize that the mandibles are the major moving bones of the TMJ in comparison to the mandibular fossa of the squamous temporal bone that remains stationary with respect to the cranium. The TMJ structure – function relationship have similarities but also several differences across the mammalian species. For example, dogs can open and close the mouth as a result of the TMJ hinge movement and a slight laterotrusion movement is possible. In comparison, the cat TMJ has a closer congruity, and the structure of the feline mandibular symphysis allows only a hinge movement and less independent movements of the mandible. These functionality restrictions should be considered during diagnosis and treatment.

Temporomandibular joint disorders mostly include arthritis, fractures due to trauma, dysplasia and ankylosis and rarely neoplasia. When present, TMJ disorders may be debilitating necessitating medical or surgical treatments. TMJ arthritis can be crippling, leading to a variety of morphological and functional abnormalities. In veterinary medicine computed tomography (CT) is a major imaging tool to evaluate the TMJ as it is valuable not only for evaluation of osseous pathosis but also for spatial position of the TMJ bones.
Moreover, three-dimensional CT reconstructions images may improve the understanding of the lesions in selected cases. Also, CT has been found superior to conventional skull radiographs for identification of anatomic structures and pathologies at the maxillofacial regions in dogs and cats.

The following list encompasses common TMJ disorders:

**Temporomandibular joint dysplasia**
Temporomandibular joint dysplasia typically results in subluxation and lateral and/or rostral displacement of the mandible. In turn, the coronoid process contacts the adjacent zygomatic arch and results in intermittent episodes of inability to close the mouth or joint pain without locking episodes. The diagnosis of TMJ dysplasia should heavily rely on clinical findings of ‘locking’ of the coronoid process on the zygomatic arch. This can be done safely with the patient under general anesthesia. A CT scan should complement the clinical findings. Long-term TMJ dysplasia should be managed surgically. The main aim of the surgery is to eliminate the potential for ‘locking’ of the coronoid process on the zygomatic arch. Therefore, coronoidectomy with or without zygomaticectomy is the current surgical methods of choice. As TMJ dysplasia can occur bilaterally, clinical and CT evaluation should include both joints. Management of TMJ dysplasia using condylectomy is inappropriate and will result in an even greater displacement of the mandible and higher potential for open-mouth jaw-locking.

**Temporomandibular joint luxation**
Temporomandibular joint luxation typically occurs in a rostrodorsal direction. Due to the presence of the retroarticular process, luxation in a caudal direction rarely occurs and if it does, the retroarticular process is likely to be fractured. Regardless, the direction of the luxation and the TMJ should be imaged utilizing CT to confirm the disorder, direction, and potentially associated disorders. In most cases, management of TMJ luxation is done by closed reduction. Under general anesthesia, a fulcrum is obtained by placing a pencil (but not too rigid) transversely across the mandible as caudal as possible. While the mouth is gently closed the pencil is rotated in counterclockwise to result in the mandible moving in a ventral and caudal direction and the TMJ to rearticulate. Rigid fixation utilizing maxillomandibular fixation is recommended for 7-14 days. Recurrence of luxation is likely to occur if no fixation is done. A muzzle can be placed but may not have a positive outcome as the TMJ can reluxate spontaneously. Feeding tube placement is necessary if rigid maxillomandibular fixation is placed.

In difficult situations, an open reduction may be needed. In that case, it is advised to examine the reason for the difficult reduction. Folding of the TMJ disc, foreign body or preexisting TMJ arthritis are possible reasons for difficult TMJ reduction. Reconstruction of the TMJ capsule and lateral ligament are necessary to prevent luxation.
Temporomandibular joint fractures
Temporomandibular joint fractures typically occur as a result of trauma and are often seen in combination with other maxillofacial injuries. We can classify TMJ fractures to be intra-articular and extra-articular. Moreover, the fracture can involve the condylar process and the mandibular head as a solitary lesion or cross the joint and involve the mandibular fossa as well. Furthermore, the fracture segments can be non-displaced or have displacement to various extents and may have fracture fragments within the joint space. Hence, it is crucial to have a full understanding of the fracture configuration before formulating a treatment plan. As in other TMJ disorders, fracture characterization should be made based on CT imaging. The goal of managing TMJ fractures is to restore mandibular symmetry, occlusion, and function and to prevent long-term complications. In young dogs and cats as well as in most adult dogs (and based on the fracture configuration), non-surgical (i.e., conservative) therapy is the method of choice. In fact, there is an excellent chance for fracture healing and regeneration of the damaged tissues as well as the continuation of normal development in young dogs and cats with TMJ fractures.

Maxillomandibular fixation (MMF) can be done in either one of two methods: rigid or elastic therapy. The disadvantage of rigid therapy is delayed return to normal function, maintaining feeding tube, poor oral hygiene, difficulties in thermoregulation and potential aspiration.

However, if the fracture is non-displaced and there is mild or no malocclusion, then elastic (functional) therapy is recommended for a period of 14 days. This will allow a more rapid return to normal function as compared to rigid therapy, allow the fracture area to receive more blood supply (due to the movement of the joint and muscles surrounding it) and decrease the chance of complications due to aspiration or thermoregulation issues. Open reduction is only recommended if there is/are fracture fragments in the joint space preventing opening or closing the mouth. Condylectomy is not recommended and should be reserved as an extreme measure in case of complete destruction of the TMJ and for fragments that prevent the joint from regaining normal function.

Temporomandibular joint osteoarthritis
Diagnosis of TMJ osteoarthritis (TMJ-OA) can only be achieved by clinical combined with CT evaluations. It is important to remember that while TMJ-OA is the most common TMJ disorder, it is often combined with other TMJ disorder. Once TMJ-OA as a solitary disease is diagnosed, therapy is aimed at alleviating pain and regaining TMJ functionality. Non-steroidal anti-inflammatory medications for 2-4 weeks with or without opioid supplementation are recommended. Also, ‘jaw rest’ can be performed for 2-4 weeks by preventing rough chewing (i.e., chewing on raw hide, tug-of-war games, etc.). However, physical therapy utilizing opening and closing the mouth to exercise and stretch the joint in a controlled fashion is recommended. Also, soft food should be given for the first few days followed by a return to normal size kibble. Only in extreme situation where the pain cannot be controlled, or the TMJ exhibits signs of ankylosis, condylectomy should be performed.
**Temporomandibular joint ankylosis**

True ankylosis of the TMJ or intracapsular fusion of the joint can be osseous or fibrous. It typically results in a reduction of the range of motion up to the point of complete immobility. The most common cause of ankylosis is trauma associated with the condylar process and/or the mandibular head. Other causes are a previous surgical treatment that resulted in scarring and joint infection. In general, the incidence of TMJ ankylosis in dogs and cats has not been reported and in humans is about 0.4% following TMJ trauma.

The treatment of choice is condylectomy or ‘gap arthroplasty’. There is no need to fill the void following condylectomy with adipose tissue.

**Differential diagnosis**

Keeping in mind the plethora of TMJ disorders that may be present, the clinician needs to be aware of other possibilities, or differential diagnosis, that may be present, mimicking TMJ disorders. For example, pain on opening the mouth may occur due to retrobulbar disease (i.e., abscess or neoplasm) or ear disease. Trouble in opening the mouth may occur due to masticatory muscle myositis. Therefore, a thorough understanding of the TMJ and its structure-function properties is fundamental to comprehensive clinical approach to formulate an appropriate treatment plan.

This lecture will discuss in details the clinically relevant aspects of difficulties in opening or closing the mouth, including TMJ disorders and differential diagnoses as well as surgical and medical treatments.

**References**


Stem Cell Therapy for Feline Chronic Gingivostomatitis: What Have We Learned So Far?

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Feline chronic gingivostomatitis (FCGS) is a severe inflammatory oral disease of cats that is often refractory to treatment. The condition is characterized by oral inflammation affecting the gingival and non-gingival mucosa. Affected cats typically display clinical signs related to oral pain, including inappetence, anorexia and ptyalism. The etiopathogenesis of FCGS is partially understood. Current treatment modalities include full or near-full mouth dental extractions, corticosteroids, antibiotics and analgesia. Extraction therapy provides the highest success rate with approximately 30% of cats exhibiting complete remission, 40% exhibit improvement and 30% of cats do not respond to extraction therapy. The cats that do not respond to extractions will often need lifelong medical management to maintain adequate quality of life. Adipose-derived mesenchymal stem cells (adMSCs) have regenerative and immunomodulatory capabilities and can be easily obtained in large numbers from harvested fat.

Our studies in the past 7 years, as well as our ongoing clinical trials, focus on the safety and efficacy of adMSCs in treating refractory FCGS after full-mouth dental extractions has failed to achieve substantial improvement or complete remission. To date, we have found that approximately 70% of the cats with refractory FCGS demonstrated significant improvement 2-6 months after receiving adMSC treatment. A subset of cats that exhibited slow response to adMSC treatment demonstrated a delayed response with either substantial improvement or complete resolution within a year of treatment. Moreover, we demonstrated that adMSCs are capable of immunomodulatory effects in cats and those effects are seen in conjunction with clinical improvement. This lecture will focus on the work that we have completed since the beginning of the clinical trial and will outline the difficulties encountered and the future direction of adMSCs therapy in cats and humans. Finally, a path from current clinical-trial work to vast clinical practice and commercialization will be described.

References


Pain management in cats can present unique challenges, particularly considering the concerns for adverse effects of nonsteroidal anti-inflammatory drugs in the species. These proceedings focus on management of acute pain in hospitalized cats with an emphasis on non-opioid choices due to their limited availability.

**PAIN PHYSIOLOGY**

Pain in people is an unpleasant sensory and emotional experience associated with potential or actual tissue damage; it is assumed that other mammals experience pain in a similar way. The pain pathway consists of at least 4 distinct steps, each of which is a potential target for analgesic therapy; in other words, a medication can impact the perception of pain by interrupting the pain pathway at any (or many) of these 4 steps.

*Transduction:* The pain pathway is initiated when nerve endings are affected by a *noxious stimulus,* which can be mechanical, chemical, or thermal in nature. The first step of the pathway is *transduction* of the noxious stimulus into an electrical (neural) signal. Nonsteroidal anti-inflammatory drugs, opioids, and local anesthetics can interfere with transduction of noxious stimulus to neural signal, so are all effective at this step in the pain pathway.

*Transmission:* The second step in the pain pathway is *transmission* of the neural impulse from the site of injury up the sensory nerve and spinal cord. Local anesthetics (introduced locally or regionally; systemic effects debated) and alpha-2 agonists (administered regionally or systemically) interfere with signal transmission, so are effective at this step in the pain pathway.

*Modulation:* The third step of the pain pathway is *modulation.* As afferent nerves transmitting the painful signal ascend the dorsal horn of the spinal cord, these signals are modulated by neurons that project from the midbrain to the cord. These inhibitory signals help attenuate and ultimately lessen the perception of pain at the level of the cortex. Lack of ability to mitigate these nociceptive (painful) signals by modulation is the basis of the “central sensitization” or “wind-up” phenomenon that contributes to chronic and neuropathic pain in people. Drugs that provide analgesia by affecting the modulatory step include opioids (administered systemically or regionally), nonsteroidal anti-inflammatory drugs, alpha-2 agonists (administered regionally or systemically), NMDA antagonists, and local anesthetics (introduced regionally; systemic effects debated) all inhibit pain at the modulatory step.

*Perception:* The final step in the pain pathway is *perception,* which is the cortical / conscious awareness of pain. Prior to the point of awareness of pain, the signal is considered only nociceptive – it is a message of a noxious stimulus. It is not until the perceptive stage that this neural information is perceived as pain by the animal. Opioids and alpha-2 agonists (as well as general anesthetics) administered systemically inhibit pain perception.

**DETECTING PAIN IN HOSPITALIZED CATS**

It can be challenging to detect pain in caged, nonverbal animals. While lameness or certain guarding behaviors can provide clues in the home setting, these behaviors are harder to appreciate and interpret in (often fearful) hospitalized cats. However, recent studies and clinical reviews provide some guidance for diagnosing pain in cats. In the 2015 AAHA/AAFP Pain Management Guidelines for Dogs and Cats, Epstein and others suggest to monitor for normal behaviors (grooming, eating, postures, sleeping positions), which may be absent in painful animals, and for new behaviors (lack of eating and grooming; withdrawal or unusual aggression) that may indicate pain.

*Facial expression:* Holden and others described in 2014 facial changes that appear to indicate acute pain in cats, namely the flattening of the pinnae from an erect position and the forward positioning of muzzle and whiskers.

*Pain scoring:* Two pain scales developed at Colorado State University are used commonly to score acute pain in cats and kittens. These scales include scoring sections for psychological and behavioral, response to stimulus (palpation), and overall body tension. Both scales (as well as a canine version) are freely available on the Internet at [http://csu-cvmbs.colostate.edu/Documents/anesthesia-pain-management-pain-score-feline.pdf](http://csu-cvmbs.colostate.edu/Documents/anesthesia-pain-management-pain-score-feline.pdf) (adult cat) and [http://www.vasg.org/pdfs/CSU_Acute_Pain_Scale_Kitten.pdf](http://www.vasg.org/pdfs/CSU_Acute_Pain_Scale_Kitten.pdf) (kittens; both accessed Sept. 26, 2019). Pain scoring can be helpful to standardize practice within a hospital, though inter- and intra-observer scoring can vary.
Physiologic parameters: increased heart or respiratory rate can be seen in very painful animals, but absence of these abnormalities does not rule out pain.

Index of Suspicion – In general, when we are uncertain about a hospitalized cat’s comfort, it makes sense to maintain an index of suspicion for pain. In the presence of disease or trauma (natural or surgical) that would cause pain in a person, paired with the absence of normal behavior, treating for pain is an appropriate strategy. If behavior or physiologic parameters improve with analgesics, pain may have been a problem and continued treatment can benefit the cat.

TREATING PAIN IN HOSPITALIZED CATS

Local anesthetics: Local anesthetics such as lidocaine and bupivacaine can be used in cats, though their doses are lower than those used in dogs. Drugs in this class must be administered parenterally and can be given by local infiltration as a subcutaneous / intramuscular infiltrate, regionally (at the root or proximal region of a peripheral nerve), intracavitary, or epidurally. These drugs cause analgesia by blocking sodium channels in the nervous system; they can thus affect the pain pathway at the transduction, transmission, and modulation steps. Along with analgesia, these drugs also cause loss of function (paresis or paralysis).

Lidocaine

- Feline dose for local infiltration: 1 – 2.5 mg/kg of a 0.5% solution (total dose per cat, not per site) for a procedure; unlikely to provide analgesia beyond 2-4 hours. Epidurally, up to 4.4 mg/kg as a 2% solution.

Bupivicaine

- Feline dose for local infiltration: 1 mg/kg of 0.25% or 0.5% bupivacaine solution (total dose per cat, not per site) as often as every 8 to 12 hours.

A newer, longer-acting liposomal bupivacaine product (Nocita® Aratana Therapeutics) is labeled for use in cats for SC infiltration prior to onychectomy at a total dose of 10.6 mg/kg per cat (5.3 mg/kg per forelimb). This drug cannot be used systemically nor directly into joints due to risk of chondrolysis. The therapeutic index of Nocita® is narrow in cats, so care should be taken with the dose. A single infiltration can provide up to 72 hours of analgesia to the blocked region.

Nonsteroidal anti-inflammatory drugs: Some NSAIDs can be used in cats, though concerns exist that the species may be less tolerant of their kidney-associated adverse effects than dogs and people. Drugs in this class can be administered enterally (orally) and some have parenteral formulations. Pharmacologic and adverse effects of NSAIDs are systemic; the drugs inhibit inflammation and pain by inhibiting prostaglandin synthesis by blocking cyclo-oxygenase. This drug class affects the pain pathway at the transduction and modulation steps.

Robenacoxib (Onsior) is reasonable to use in hospitalized cats that are well-hydrated, have stable kidney function, and (for oral administration) can tolerate oral medications. Per the product label, the cat should be at least 4 months old and weigh ≥ 2.5 kg to receive robenacoxib, and doses are 6mg PO once daily for cats 2.6 – 6 kg and 12mg PO once daily for cats 6.1 – 12 kg. For parenteral administration, the dose is 2 mg/kg SC once daily for up to 3 days.

Meloxicam can be used in cats with caution. It is labeled for one-time use at 0.3mg/kg SC for acute pain relief. Off-label in the USA, doses have also been reported to be 0.1 – 0.2 mg/kg once on the first day of treatment followed by 0.05 mg/kg every 24 hours for the duration of acute pain and inflammation.

Carprofen can be used with caution. Use in cats is off-label in the USA. One published dose is 4 mg/kg SC or IV once as pre-emptive analgesia before a painful procedure.

Alpha-2 agonists: The alpha-2 agonist dexmedetomidine is used frequently at a low dose as a constant rate infusion (CRI) for hospitalized cats with acute pain. This drug class affects the transmission, modulation, and perception steps of the pain pathway, and can be given SC, IM, IV, or epidurally. For acute pain management, the low-dose CRI, often in combination with other medication(s), is the most common regimen. Recommended dose for a dexmedetomidine CRI is loading dose of 1 mcg/kg IV followed by a CRI 0.5 – 2 mcg/kg/hr. At the CRI dose, the adverse effects of blood pressure and heart rate changes are usually not seen beyond the initial loading dose.

NMDA receptor antagonists: Ketamine is the most commonly used NMDA receptor antagonist in veterinary medicine in the USA. This class inhibits pain at the modulation step of the pain pathway. For analgesia, ketamine is usually administered as a CRI in conjunction with another analgesic. The loading dose is 0.5 mg/kg IV followed by 2 – 4 mcg/kg/minute of ketamine. Amantadine is another NMDA receptor antagonist for oral use in chronic pain, but little information is available in small animals.
**Veterinary-specific opioids**: While in general opioids are currently difficult to source, synthetic opioids made specifically for the veterinary industry are still readily available and remain an attractive option for treatment of acute pain in hospitalized cats. Opioids affect the transduction, modulation, and perception steps within the pain pathway.

**Butorphanol** is a fair choice for analgesia in hospitalized cats with only mild to moderate pain. It is sedative at analgesic doses, and a single injection is limited in analgesic effects. Butorphanol is better used as a CRI for analgesia, and can be given by loading with 0.2 – 0.4 mg/kg IV followed by a CRI of 0.1 – 0.4 mg/kg/hr IV. It is a mixed opioid agonist-antagonist drug with short duration of action.

**Long-acting buprenorphine** is an attractive choice for hospitalized cats with mild to moderate acute pain because it can be given SC once daily without the need for repeated injections or CRI. The feline dose is 0.24 mg/kg SC every 24 hours. It is a partial µ-opioid receptor agonist.

**Tramadol**, though available only in an oral formulation that cats generally detest, appears to be an effective analgesic in cats, who make the morphine-like metabolite from the drug. Dose is unknown, but formularies suggest 1 – 2 mg/kg q12h.

**Other analgesics to consider for hospitalized cats**

**Gabapentin**: Early data about analgesic effects of gabapentin in cats are encouraging. The drug is probably more appropriate for use in chronic pain in cats as it is in other species, but it can be considered an adjunctive medication in acute pain. Doses are unknown, but extrapolated dose is 5 – 10 mg/kg every 8–12 hours.

**Maropitant**: This drug blocks the binding of substance P (a neurotransmitter involved in the pain pathway), but efficacy as an analgesic is unknown at this time. Some experimental canine and rodent data suggest maropitant may have analgesic properties. No feline data are available to this author’s knowledge.

**Nursing and preventative care** are also paramount in treatment and prevention of pain in hospitalized cats. Appropriate padding, turning, oral care, eye care, wound management, and definitive diagnosis and treatment of underlying conditions are all vital. Therapeutic laser may be helpful, and the veterinary community eagerly awaits further information about this modality in cats. Because pain is an emotional experience, anxiolysis and as-soon-as-appropriate hospital discharge are also likely helpful in mitigating painful experiences for cats.

**REFERENCES AND SUGGESTED READINGS**

- CSU pain scale scoring system (kittens): [http://www.vasg.org/pdfs/CSU_Acute_Pain_Scale_Kitten.pdf](http://www.vasg.org/pdfs/CSU_Acute_Pain_Scale_Kitten.pdf)
Cats are not chew toys: Managing thoracic trauma in the emergency room
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Thoracic trauma is common in cats and can result from penetrating injuries such as from dog bites, or from blunt force trauma such as motor vehicle accidents and falls. Thoracic trauma can be complicated to manage because the respiratory compromise can arise from a combination of different problems including thoracic wall defects, pulmonary contusions, and pleural space problems like pneumothorax or hemothorax. These proceedings will discuss initial management strategies for cats with respiratory compromise from thoracic trauma.

HOW THORACIC TRAUMA CAN AFFECT RESPIRATION
An intact thoracic wall is required to generate the pressure changes needed for breathing. Penetrating chest wounds and diaphragmatic tears caused by dog bites, projectiles, or impalement compromise breathing by impairing negative pressure generation, and also by causing gas or blood to enter the thorax. Pneumothorax and pleural effusion allow the lung to collapse away from the chest wall and thereby impair oxygenation. Pulmonary contusion from blunt trauma or crushing injury is almost always present if chest wall trauma is present and causes significant compromise in lung function.

Disruption of the nerves and muscles that drive breathing impairs ventilation and can therefore impair oxygenation if adequate fresh gas isn’t introduced to gas exchange areas of the lung. Primary inspiratory muscles include the diaphragm, which is innervated by the phrenic nerves that arise bilaterally from C3–C5 spinal cord segments, and the intercostal muscles that are innervated by individual spinal nerves from the caudal cervical and the thoracic nerve roots. Accessory muscles aid in generating extra respiratory effort during exercise or disease, and include some cervical and back muscles, scalene muscles, and muscles of the upper airway that open the pharynx and larynx. Exhalation is passive during low-effort breathing; increased expiratory effort is generated by abdominal musculature, intercostals, and neck and back muscles. Injury to the nerves and muscles that control breathing can cause hypoventilation and hypoxemia. Pain from injuries may also inhibit the increased tidal volumes that would help a non-painful animal increase ventilation to compensate for lung and pleural space problems; this lack of hyperventilation can confound the respiratory compromise.

INITIAL TRIAGE AND INTAKE
Respiratory support
Supplemental oxygen should be provided to all cats in respiratory distress. If you are using flow-by oxygen from a circle system, the pop-off valve should be closed, the circuit should be flushed of anesthetic gas, and the rebreathing bag should be filled with fresh oxygen so the fresh gas flow to the patient will be what is dialed into the flowmeter from the start; otherwise, it takes time for the bag to fill and less oxygen flows from the Y-piece. A makeshift oxygen tent can also be made by placing the cat inside a carrier, then placing the whole carrier into a large trash bag with high-flow oxygen (via non-rebreathing system or very high flow from a rebreathing system configured as above). Small holes can be made in the bag for some temperature regulation and to allow gas to escape.

Some cats with dog bite wounds present with a flail chest, in which multiple adjacent ribs have ≥ 2 fractures and the affected segment of the thorax (the “flail segment”) moves in opposition to the rest of the thorax during breathing. The best strategy in such cases may be to place the cat with the flail segment toward the treatment table; the table provides some “splint” effect for the segment and may minimize the cat’s effort. If the cat doesn’t prefer lying with the flail segment dependently, it should be allowed to assume its preferred position instead.

Treating shock
All cats that have sustained acute trauma should have perfusion parameters evaluated immediately upon presentation. These 6 parameters include mental status (normal versus quiet/obtunded), mucous membrane color, capillary refill time, heart rate, pulse quality, and extremity (paw) temperature. Traumatized cats with dull or quiet mental status, pale or other abnormal gum color, prolonged capillary refill time, with a heart rate higher or lower than expected for their signalment and current activity level, poor pulse quality, and/or cool extremities are likely hypovolemic. Cats with hypovolemia should receive shock fluid therapy in the form of a relatively large volume of isotonic crystalloid fluid given IV over a short time period.
The volume is based on the estimated blood volume of the animal (not by a % of dehydration). Feline blood volume is 45 – 60 mL/kg. At least ¼ of this dose should be given IV over ~15 minutes to any cat suspected to have hypovolemia; up to the full blood volume can be expected to be required, depending on degree of hypovolemia. Some caution should be taken when administering large fluid volumes to cats with thoracic trauma because excess fluid can exacerbate pulmonary contusions and thus lung function. Please see the proceedings for Shock Fluid Therapy for Cats for further information.

**THORACOCENTESIS**

Pleural space disease should be ruled out in all animals with thoracic trauma. If pneumothorax or pleural effusion is present, physical examination may reveal breath sounds that are quieter than expected for the degree of respiratory effort. Bedside thoracic ultrasound can be helpful to definitively rule in or out clinically important pleural space problems when it is available (see below).

Therapeutic thoracocentesis should be performed in cats suspected to have clinically significant pneumothorax or pleural effusion. Thoracocentesis should be performed before radiographs in cases where delay or radiographic positioning could compromise the animal. Ultrasound guidance should be used when a machine is available. If no machine is available, blind chest tap should be performed between rib spaces 7 and 9, generally in the middle third of the chest when considered from spine to sternum. The appropriate site should be clipped of fur and aseptically prepared. An 18- or 20-gauge over-the-needle catheter may be used so that only soft, flexible material remains in the chest cavity during the tap; alternately, a standard butterfly needle can be used, though depending on the cat’s chest wall thickness the needle may either not reach the pleural space or may result in lung laceration when fully inserted. The needle should be introduced at an approximately 45 degree angle toward the sternum with the bevel of the needle facing the lung (dorsally), and as much as possible it should be inserted just cranial to the rib to avoid the vessels and nerves that run along the caudal rib aspect. Tubing should be attached to the catheter; a 3-way stopcock between the tubing and the aspirating syringe helps minimize disconnections during the procedure.

**THORACIC IMAGING**

**Bedside thoracic ultrasound:** Bedside ultrasound is sensitive for detecting clinically-relevant volume pleural effusion, and identification of “glide sign,” the appearance sliding at the parietal / visceral pleural interface, rules out pneumothorax at the imaged site.

**Thoracic radiographs:** Ideally 3-view thoracic radiographs are acquired once the cat is adequately stable. However, some cats may not tolerate positioning for many hours while the team attempts to stabilize the cat. Radiographs may reveal rib fractures or luxations, pneumothorax and/or pleural effusion, pulmonary contusions, and subcutaneous emphysema in cases of penetrating trauma.

**Computed tomography:** Occasionally CT is used to determine the full extent of thoracic injury in traumatized cats and to help with surgical planning, if indicated.

**WOUND MANAGEMENT**

Bite wounds should be clipped, cleaned, and explored carefully. Occasionally exploration will reveal a full-thickness chest wall defect, which in the absence of an endotracheally intubated patient should be immediately closed (even temporarily) until the cat can be fully anesthetized, intubated, and ventilated so the defect can be debrided and closed. Open chest wounds should be covered with sterile jelly or adherent dressing and a light bandage until they can be addressed surgically, which should ideally happen after stabilization and within the initial 24 hours if possible.

**MEDICATIONS**

**Antimicrobials:** Broad-spectrum antimicrobials should be given at presentation in cats with penetrating wounds. Ampicillin/sulbactam (30 mg/kg IV q 6-8 hours) or ampicillin (22 mg/kg IV q 8 hours) / enrofloxacin (5 mg/kg IV q 24 hours) are good choices. Antimicrobials are not warranted to “cover” pulmonary contusions alone if no external wounds are present.

**Analgesia** should be given to all acutely traumatized cats at presentation unless there is a distinct contraindication. Pure mu agonist opioids such as methadone (0.2 – 0.3 mg/kg q 4 – 6 hours) or hydromorphone (0.05 – 0.1 mg/kg q 4 – 6 hours) are probably both the safest and most effective choice in these cases. Once hospitalized, other adjunct medications such as dexmedetomidine or ketamine as low-dose CRIs may be considered. Please see the proceedings for “Analgesia for hospitalized cats: Life without methadone” for further information.
REFERRAL
If you plan to refer the cat, they should be stabilized prior to transport, if possible. Animals for which anxiety appears to be exacerbating the respiratory distress may benefit from sedation (for instance, butorphanol 0.2 mg/kg IM or IV once) in an attempt to stabilize them for transport. Animals known or suspected to have pleural effusion or pneumothorax contributing to distress should undergo therapeutic thoracocentesis prior to being transported. Those that cannot be stabilized prior to referral should be sent by pet ambulance if possible; tracheal intubation with manual ventilation prior to referral may be required in the most severe cases. Any IV catheter should be aseptically flushed and wrapped, and can be left in place for use at the referral facility.

REFERENCES AND FURTHER READING
- Haskins SC. Hypoxemia. In Silverstein DC, Hopper K: Small Animal Critical Care Medicine, St. Louis, 2015, Elsevier Saunders, p 81.
Diabetic ketoacidosis (DKA) is a common complication of diabetes mellitus in cats. Diabetic ketoacidosis occurs when a patient has either inadequate endogenous insulin or insulin resistance (either of these is diabetes mellitus) in combination with another predisposing condition or illness. The exact mechanism is debated, but the concurrent illness may lead to an increase in inflammatory cytokines and/or an increase in counter-regulatory ("diabetogenic") hormones such as glucagon, cortisol, and epinephrine. Increases in such mediators lead to the overproduction of ketone bodies in the liver. The main ketoacids produced in DKA are β-hydroxybutyrate, acetoacetate, and acetone. Animals with DKA are made sick by the acidemia, which causes malaise, poor appetite, vomiting, diarrhea, and subsequent dehydration, and can lead to more severe problems like cardiovascular collapse. The concurrent disease often contributes to the sickness, as does the diabetes. Common concurrent diseases in cats include but are not limited to pancreatitis, pyelonephritis, and hepatic lipidosis.

**MANAGEMENT OF DKA**

Diabetic ketoacidosis is diagnosed by establishing hyperglycemia, glucosuria, ketonemia or ketonuria, and a metabolic acidosis (low blood HCO₃⁻ and a more negative base deficit). The mainstays of treatment are aggressive fluid therapy tailored to the animal’s electrolyte imbalances and insulin. Despite the fact that people are affected by DKA and the fact DKA is seen frequently in general and emergency veterinary practices, controversies still exist regarding some aspects of management, such as whether to delay insulin therapy, how to administer insulin and what type to use during the crisis, and whether or not to treat severe acidemia with sodium bicarbonate.

**Fluid and targeted electrolyte therapy**

At presentation, fluid therapy is a higher priority than insulin in cats with DKA. Patients that present hypovolemic should be adequately fluid resuscitated with a balanced isotonic crystalloid such as Plasmalyte 148 (or A), Normosol-R, or lactated Ringer’s solution. Plasmalyte or Normosol-R may be superior choices because they contain magnesium, which is often depleted in patients with DKA. After resuscitation, fluid therapy should continue to replace any remaining deficits and maintenance requirements, as well as to account for the expected excessive ongoing losses in the polyuric diabetic.

Patients often require aggressive potassium and phosphorous supplementation in the form of KCl (2 mEq/mL K⁺) and K₂HPO₄ (4.4 mEq/mL K⁺ and 3 mmol/mL PO₄³⁻) to counteract the hypokalemia and hypophosphatemia that often accompany DKA; both also reliably worsen with insulin therapy. Standard potassium supplementation charts are available in many clinical veterinary handbooks, and potassium supplementation may need to be more aggressive than standard. The maximum safe rate of potassium supplementation without ECG monitoring is 0.5 mEq/kg/hour. Additionally, solutions containing more than ~50-60 mEq/L potassium should ideally be delivered through a central rather than peripheral vein due to hyperosmolality and thus risk for phlebitis. Dedicated phosphorous supplementation may be required if serum phosphorous drops below 2.0 mg/dL; common dose range for phosphorous supplementation in DKA is 0.01 – 0.1 mmol/kg/hr delivered as a CRI into a calcium-free fluid.

Magnesium therapy is indicated if the patient has a low ionized magnesium concentration. This is particularly true if hypomagnesemia is accompanied by persistent or severe hypokalemia because hypokalemia can be refractory to treatment in patients with hypomagnesemia. **Magnesium replacement can be administered at 0.75 mEq/kg/day as a continuous rate infusion**; some authors recommend loading the first ¼ to ½ of the dose over 10–20 minutes, followed by a slower infusion for the rest of the daily requirement. Norm-R (or P-148) run at a twice-maintenance rate supplies approximately ½ the daily Mg needed for replacement, which is another reason these fluids may be particularly desirable for treating DKA. Patients receiving full-dose Mg supplementation should be monitored closely with ECG for dysrhythmias, and for hypotension or gastrointestinal disturbances.
Insulin therapy
Insulin therapy is the second mainstay of treatment for DKA. Recommendations regarding the timing and method of insulin therapy vary. The primary goal of insulin therapy in the DKA patient is different than that for patients with stable diabetes mellitus. **The primary goal of insulin therapy in DKA is to stop ketone production, not to optimize blood glucose concentration.** Exactly how and when insulin should be administered, and how aggressively the acidemia should be managed, are debated.

Timing of Insulin Initiation
It is generally agreed that fluid resuscitation should commence as soon as possible at presentation. Some veterinary authors recommend delaying insulin therapy until the animal is rehydrated or has received IV fluids for at least 6 hours; others do not emphasize such a delay. The arguments for delay in insulin administration are generally that a) blood glucose concentration decreases somewhat with fluid therapy alone due to dilution and improved renal excretion; and that b) insulin administration along with initial fluid therapy may lead to overly-rapid electrolyte changes that could cause cerebral edema. The American Diabetes Association does not recommend a delay in insulin administration in its position and consensus statements on hyperglycemic crises unless the patient has moderate to severe hypokalemia, and there is not strong evidence to support waiting to start insulin. Furthermore, it is now believed that cerebral edema (seen particularly in human pediatric DKA) is not cytotoxic edema due to electrolyte shifts, but rather vasogenic edema due to vascular permeability changes from the ketoacidosis itself. Ketogenesis (and acid production) will not stop until patients with DKA receive exogenous insulin. Moreover, a retrospective study published in the Journal of Veterinary Emergency and Critical Care found that initiation of insulin therapy ≤ 6 hours after initiation of fluid therapy was associated with significantly more rapid resolution of DKA without adverse effects in a group of 60 dogs and cats.

Hyponatremia associated with hyperglycemia
In diabetes, glucose acts as an effective osmole because it accumulates in the extracellular fluid, unable to enter the cells. Being an “effective osmole” means that the molecule (glucose in this case) has the ability to “hold” water molecules nearby due to osmotic forces. When glucose molecules accumulate in the ECF, they therefore attract water molecules into the ECF. Increased water accumulation in the ECF drops the sodium concentration by an expected degree for each mg/dL increase in BG: a ~2 mEq/L drop in [Na+] is expected for every 100mg/dL increase in the BG. Thus, if a patient’s normal [Na+] is 150mEq/L and normal BG is 100mg/dL, if that patient develops a BG of 500, one could calculate the expected [Na+] drop as:

$\text{(400 mg/dL increase in BG) x (2 mEq/L decrease in [Na+] per 100 mg/dL increase in BG) = 4 x 2 mEq/L = 8 mEq/L expected drop in [Na+].}$

Thus, expected [Na+] = 142 meq/L. Conversely, as the BG drops with fluid therapy and insulin therapy, the [Na+] is expected to increase by ~2 mEq/L for every decrease in BG by ~100 mg/dL. Note that these fluctuations are nearly osmotically neutral and thus do not lead to significant fluid shifts into and out of cells.

Constant Rate Infusion of IV Insulin versus Intermittent IM (+/- SC) Insulin
Regular (“crystalline”) insulin CRIs are now commonplace in treatment of feline DKA and have largely replaced intermittent IM regular insulin protocols. Please see Table 1 and Figure 1 for examples of common insulin regimens used in DKA. Advantages of the continuous rate infusion method are ease of infusion after initial setup and reliability; disadvantages are the opportunity for insulin overdose with incorrect fluid rate or insulin concentration, the need for a second IV infusion and fluid pump, and the fact that 50 mL of insulin infusion must be wasted through the infusion line prior to administration. Advantages of the intermittent IM method are its simplicity, its reliability, and the lack of needing a second IV infusion setup; disadvantages are the opportunity to make dosing errors with each injection, repeated patient interactions, and increased technician time. Authors agree both CRI and intermittent IM methods are effective and reliable, so this is a decision based on resources and institutional preference.

One method involving the concomitant, intermittent SC and IM injection of glargine has been described in cats, as has a method combining intermittent SC glargine and IM regular insulin. Compared to regular insulin CRI, combination SC glargine and IM regular insulin performed at least as well in resolving feline DKA, though the study was very small. A very small recent study of cats with DKA suggested non-inferiority of lispro insulin CRI when compared to regular insulin CRI.
Table 1: One protocol for CRI of intravenous regular insulin in treatment of DKA. Other similar protocols exist.

Add 2.2 U regular insulin per kg body weight to a 250 mL bag of 0.9% NaCl. Mix well. Some protocols recommend half this concentration for cats, though 2.2 U/kg may be more effective in cats based on recent data. Run 50mL of solution through IV tubing before attaching administration set to patient IV line.

<table>
<thead>
<tr>
<th>Blood Glucose Concentration, mg/dL</th>
<th>Crystalloid fluid composition:</th>
<th>Rate of insulin CRI administration, mL/hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 250</td>
<td>No additives</td>
<td>10</td>
</tr>
<tr>
<td>200 – 250</td>
<td>2.5% dextrose</td>
<td>7</td>
</tr>
<tr>
<td>150 – 200</td>
<td>2.5% dextrose</td>
<td>5</td>
</tr>
<tr>
<td>100 – 150</td>
<td>5% dextrose</td>
<td>5</td>
</tr>
<tr>
<td>&lt; 100</td>
<td>5% dextrose</td>
<td>0 (Stop insulin)</td>
</tr>
</tbody>
</table>

Figure 1: Protocol for intermittent IM administration of regular insulin in treatment of DKA

Switching to long-acting insulin
Once the animal is adequately hydrated, eating, and (serum or urine) ketones are trace or absent, the switch is made to long-acting, SC insulin at an appropriate maintenance dose and interval for the insulin type.

Bicarbonate therapy
It is generally agreed that sodium bicarbonate therapy is not indicated in DKA unless severe acidemia persists after fluid resuscitation (pH ≤ 7.0 – 7.1, or blood [HCO₃⁻] ≤ 11 – 12 mEq/L, depending on author). Advocates of bicarbonate therapy recommend its use because severe acidemia causes significant problems, such as anorexia, vomiting, diarrhea, cardiovascular dysfunction including death, and electrolyte abnormalities. However, treatment of DKA with bicarbonate may be counter-productive, because ketone body metabolism (which occurs once insulin therapy begins) consumes hydrogen ions and thus treats acidemia on its own.
Additionally, some studies suggest that bicarbonate therapy doesn’t improve or even worsens prognosis in DKA. If one chooses to use bicarbonate therapy in animals with DKA, the animal should be monitored carefully for severe hypokalemia, ionized hypocalcemia, hypernatremia, signs of cerebral edema, and rebound alkalosis (which is very difficult to treat), all of which can be complications of sodium bicarbonate therapy.

Cats that experience a bout of DKA can still enter a state of remission from their diabetes mellitus. Indeed, long-term prognosis for cats having experienced DKA appears no worse than that of other diabetics, as long as they are supported adequately through the crisis.

REFERENCES AND SUGGESTED READINGS

Urethral obstruction in male cats is an immediately life-threatening problem that requires emergency treatment to save the cat. In the United States, approximately 70 – 75% of cats with urethral obstruction have feline idiopathic cystitis, a disease process associated with inflammation of the lower urinary tract. Whether this problem is confined to the lower urinary tract or whether the lower urinary tract is one of many organ systems affected in cats with obstructive feline idiopathic cystitis (OFIC) is a matter of active investigation. Fewer cats with urethral blockage have obstruction secondary to cystic calculi, neoplasia, foreign bodies, or other obstructive processes, or uncommonly in association with bacterial cystitis. Treatment of OFIC often includes intravenous catheterization for IV fluid therapy, sedation or anesthesia, a urethral catheterization procedure, hospitalization for a day or longer, medications, and in-home dietary, water provision, and environmental changes. These measures are emotionally and financially costly to clients, and stressful for the cat.

Unfortunately, cats that experience OFIC are at high risk for re-obstruction after the initial event, with recently published recurrence rates as high as 38% depending on the length of the follow-up period. Cats that experience their first OFIC event under the age of 4 years may be at higher risk of future OFIC events, though a single study found that older cats were at higher risk of recurrent OFIC. Since urethral obstruction in male cats is most commonly associated with FIC in the United States, long-term prevention of feline OFIC primarily focuses on decreasing incidence of FIC using diet and multimodal environmental modification. However, since many cats that re-obstruct do so within the first days or weeks following initial obstructive event, the veterinary emergency community has recently begun to investigate how we can prevent recurrence of OFIC within the first few weeks of the initial event. These proceedings focus on some recent investigations, the results of which may help the emergency clinician reduce the rate of short-term recurrent OFIC in male cats.

INITIAL PRESENTATION AND ACUTE CARE
Cats with OFIC often present with a history of lower urinary tract signs, which may include dysuria, pollakiuria, stranguria, hematuria, and peruria. Some clients confuse stranguria with tenesmus, so male cats presenting for “constipation” should be assessed for urethral obstruction. Depending on the length of time the obstruction has been present, cats may also be lethargic, hiding, inappetant, vomiting, or collapsed due to shock. Classic physical examination findings are a large, inexpressable and apparently painful urinary bladder, and a dark red or purple penile tip. The cat may also be dehydrated or in hypovolemic shock, or may have bradycardia or bradyarrhythmias due to hyperkalemia and acidemia.

Immediate treatment includes provision of oxygen to collapsed cats, IV catheterization with IV fluid therapy, and relief of the urethral obstruction as soon as reasonably possible. Alert cats require sedation for urethral catheterization, and anecdotally some people report easier urethral catheterization when cats are under general anesthesia. Patients should be fluid resuscitated prior to sedation or anesthesia, if possible. No study to this author’s knowledge has found a meaningful difference among different sedation or anesthetic protocols for this specific disease process, so best practices are those that consider the cardiovascular, respiratory, and neurologic systems of the individual. In severely obtunded cats, no sedation is required or recommended.

INITIAL RELIEF OF URETHRAL OBSTRUCTION
No evaluation has been done to compare catheter types for the initial relief of obstruction. Polypropylene, olive-tipped, stylet-reinforced, and other catheter types are available for this purpose, and some people prefer to use a 22-Ga intravenous catheter with the needle removed. Something with moderate firmness is generally required to dislodge plugs or stones, though softer materials may pass easily if the obstruction is strictly functional without a physically obstructive object. A recent investigation that was presented in abstract form at the 2017 International Veterinary Emergency and Critical Care Symposium found no increased risk or benefit associated with decompressive cystocentesis prior to initial catheterization. However, this study was designed such that every male cat presenting with OFIC was randomly allocated to either catheterization alone or to decompressive cystocentesis followed by catheterization. Thus, it is still unclear whether a subset of cats that are particularly difficult to unblock with catheterization alone may benefit from initial decompressive cystocentesis.

INDWELLING CATHETERIZATION
A recent prospective cohort study evaluated the benefit of placement of an indwelling catheter compared to single, temporary catheterization followed immediately by hospital discharge. This study found that cats were 3.7 times more likely to re-obstruct within 30 days if they were discharged immediately after unblocking (14/45, 31% re-obstructed) as opposed to being hospitalized with an indwelling urethral catheter for ≥ 24 hours (5/46, 11% re-obstructed). This finding was in agreement with another recent prospective cohort study that found that longer catheterization time was associated with lower risk of recurrent OFIC. The Seitz study also showed that among cats managed as inpatients with indwelling urethral catheters, almost all of which had a catheter in place for ≥ 24 hours, the length of catheter dwell time had no association with likelihood of recurrence. This finding is consistent with another study that found no effect of catheter dwell time on likelihood to re-obstruct when the catheterization time was long (in that study, mean ~36 hours). Yet another older study suggested that longer catheter dwell times may be associated with higher risk for recurrent OFIC. It appears that the common recommendation to hospitalize for a day following initial OFIC relief is appropriate, and the benefit beyond that time is unknown.

Ideally, cats should probably remain catheterized until their urine is grossly clear. In the study by Seitz and others, removal of the urethral catheter while the urine was still grossly bloody was a risk factor for re-obstruction within 30 days. To this author’s knowledge, no study has yet evaluated the effect or prognostic value of urine turbidity on re-obstruction in cats with OFIC.

If the cat can be hospitalized for indwelling urethral catheterization, it appears that a smaller diameter catheter may be beneficial. Hetrick and others found that male cats managed with a 3.5-Fr indwelling urethral catheter were significantly less likely to re-obstruct at 24 hours than those managed with a 5-Fr catheter. The Hetrick study was retrospective, and so more confounders may be at play (for instance, larger catheters may have been chosen for cats with more turbid urine at the outset, which could bias the results). However, a smaller catheter may be a safer choice.

When indwelling catheterization is not possible, repeated decompressive cystocentesis and pharmacologic management may be pursued. Such a protocol has been proposed by Cooper and others, and in that case series, 11 of 15 cats treated with repeated cystocentesis and anxiolytics urinated spontaneously within 72 hours. Two of 9 cats (22%) with follow-up at 3 weeks had experienced recurrent OFIC, while 7/9 had not.

**PHARMACOLOGIC TREATMENT**

Urethral anti-spasmodics are commonly used to treat cats following OFIC. One recent study found that cats treated with prazosin were less likely to re-obstruct than those treated with phenoxybenzamine. The retrospective nature of that study and the fact that the hospital's protocol for many portions of OFIC management changed at one time confound this finding, however. A more recent prospective, double-blinded, randomized clinical trial found no treatment effect of prazosin compared to placebo in cats treated for OFIC; however, the study was underpowered to detect the expected difference, and so the results are difficult to interpret. Since prazosin is an alpha receptor antagonist that inhibits smooth muscle contraction, and the distal portion of the feline urethra where most obstruction occurs is largely skeletal muscle, it does not make intuitive sense that prazosin would be effective at preventing OFIC. Other muscle relaxants proposed for use in OFIC are phenoxybenzamine and acepromazine, both of which are also alpha antagonists that tend to have longer onset of action and more sedative properties, respectively. Because of these properties of phenoxybenzamine and acepromazine, prazosin seems to be used more commonly in practice at this time.

Analgesics are routinely administered to cats with OFIC since FIC a painful, inflammatory process. While painful patients should be provided analgesics when clinically appropriate, there is no evidence that consistent provision of analgesia reduces rate of OFIC recurrence. Meloxicam has also been used to relieve pain in cats with OFIC and has been evaluated as a possible treatment to reduce recurrence. Unfortunately, no treatment effect has been found in either a retrospective cohort study or in two recent prospective, randomized clinical trials on recurrence when meloxicam was given to cats during or after hospitalization for OFIC. Similarly, volume of IV fluids administered has not been associated with recurrence, nor has length of hospitalization (separate from length of indwelling urethral catheterization).

Investigators have tried to prevent recurrent OFIC by instillation of a variety of medications into the bladder and urethra, including the paralytic atracurium, glycosaminoglycans, and pentosan polysulfate; none were found...
effective. These medications, topical benzodiazepines, and Walpole’s solution have all been reported
anecdotally to prevent recurrent OFIC, but evidence is lacking.

**LONG-TERM FACTORS AFFECTING INCIDENCE OF FIC**

From a long-term perspective, initiation of multimodal environmental modification (MEMO) is thought to be
instrumental in decreasing stress in cats with FIC and thus in reducing their clinical signs. Environmental
enrichment, appropriate placement and number of litter boxes, provision of adequate number and placement
of feeding and watering stations, and provision of adequate number and placement of resting places are all
part of the MEMO recommendations and can be included in discharge instructions. Similarly, increased water
intake has been associated with a decrease in even short-term OFIC recurrence and should thus be
encouraged immediately upon discharge. Excellent resources for client information on long-term management
of FIC include the Indoor Pet Initiative sponsored by the Ohio State University (https://indoorpet.osu.edu) and
the client site on the subject at VeterinaryPartner.com: (http://www.veterinarypartner.com/Content.plx?P=A&A=612), both accessed on September 24, 2019.

In the future, research into FIC and best prevention and treatment measures will likely be a multi-disciplinary
venture, to include basic scientists and physicians, as well as veterinarians from general and feline-only
practice, internists, emergency clinicians, and emergency / critical care specialists.

**REFERENCES & FURTHER READING**

- Bradley AM, Lappin MR. Intravesical glycosaminoglycans for obstructive feline idiopathic cystitis: a pilot
- Buffington CAT, Westropp JL, Chew DJ, Bolus RR. Clinical evaluation of multimodal environmental
  modification (MEMO) in the management of cats with idiopathic cystitis. J Fel Med Surg 2006;8:261–
  268.
- Buffington CAT, Westropp JL, Chew DJ. From FUS to Pandora Syndrome. Where are we, how did we
- Cooper ES, Owens TJ, Chew DJ, Buffington CAT. A protocol for managing urethral obstruction in male
  640.
- Cooper ES, Lasley E, Daniels JB, Chew DJ. Incidence of bacteriuria at presentation and resulting from
- DECYST Trial: a multicenter study evaluating decompressive cystocentesis for treatment of feline
  urethral obstruction. (Abstract) Presented at the 23rd International Veterinary Emergency and Critical
  Care Symposium, September 2017, Nashville, TN.
- Delille M Fröhlich L, Müller RS, et al. Efficacy of intravesical pentosan polysulfate sodium in cats with
  1146.
- Galluzzi F, De Rensis F, Menozzi A, Spattini G. Effect of intraurethral administration of atracurium
- Gerber B, Eichenberger S, Reusch CE. Guarded long-term prognosis in male cats with urethral
- Hetrick PF, Davidow EB. Initial treatment factors associated with feline urethral obstruction recurrence
- Lee JA, Drobatz KJ. Characterization of the clinical characteristics, electrolytes, acid-base, and renal


Pneumocat! Managing peri- and post-anesthetic complications
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Feline anesthesia can be challenging, and cats are twice as likely as dogs to experience a fatal peri-anesthetic complication. These proceedings focus on recognition and management of some common peri-anesthetic complications seen in cats in veterinary hospitals.

HYPOTENSION
Hypotension is one of the most common peri-anesthetic complications seen in cats; frustratingly, sometimes it seems that cats require hypotension-inducing levels of anesthesia to prevent movement. Cats under anesthesia should have blood pressure monitored frequently. Hypotension is prevented and treated by a combination of factors. **Balanced anesthetic technique:** Balanced anesthetic technique allows adequate anesthetic depth while minimizing the adverse effect of any single agent. Balanced anesthetic technique starts with pre-emptive analgesia for painful procedures; analgesia is usually provided as an opioid or a nonsteroidal anti-inflammatory drug for stable cats or as an opioid for sick animals. Methadone (0.2 – 0.3 mg/kg) or hydromorphone (0.05 mg/kg) are appropriate pre-medications for most procedures; buprenorphine (0.005 – 0.02 mg/kg) may be considered if post-operative pain is expected to be only moderate. Pre-emptive analgesia minimizes the amount of hypotension-inducing inhalant or IV anesthesia agent required to induce anesthesia and perform the procedure.

Induction with a combination of agents, such as propofol and a benzodiazepine, or alfaxalone and a benzodiazepine, helps minimize cardiovascular depressant effects. Inhalant should be titrated to the minimum-required dose to prevent patient movement in an effort to minimize hypotension. **Fluid therapy:** The cat should receive IV fluids during anesthesia unless there is a specific contraindication. Depending on the cat’s initial hydration status, comorbidities (such as kidney disease, diabetes mellitus, heart disease), and the procedure, common anesthetic fluid rates range from 3 – 10 mL/kg/hr IV of balanced isotonic crystalloid; 5 mL/kg/hr is used commonly for standard anesthetic conditions for non-diabetic cats with normal hearts and kidneys. **Vasopressors:** For cats that become hypotensive under anesthesia, first steps are usually to decrease the inhalant concentration and administer a 5 – 10 mL/kg bolus of isotonic crystalloid. If these measures fail to increase the blood pressure, vasopressor therapy may be required. **Dopamine** is used commonly to support blood pressure in cats under anesthesia, often started at a rate of 5 mcg/kg/minute and titrated as high as 10–15 mcg/kg/minute to achieve adequate blood pressure. Dopamine dilution can be calculated by the following formula:

\[
\text{(Desired \: mcg/kg/minute)} \times (\text{cat weight, kg}) \times (\text{total fluid volume, mL}) \div (\text{delivery rate, mL/minute}) \times 1000 \, \text{mcg/mg} = \text{mg to add to the fluid volume}
\]

Occasionally norepinephrine is required; common doses are 0.05 – 0.2 mcg/kg/minute started at the low end and titrated up to effect.

TRACHEAL TRAUMA and SUBCUTANEOUS EMPHYSEMA
Tracheal tears can occur during endotracheal intubation in cats. Anecdotally, many cats with tracheal tears associated with intubation suffered the tear during a dental procedure; this is probably because the head is moved during dental procedures and if the head is moved without first deflating the tube’s cuff, tracheal trauma can ensue. Tracheal tears are sometimes associated with respiratory distress, but often are discovered because the cat develops subcutaneous emphysema. Thoracic radiographs usually reveal a pneumomediastinum and SC emphysema; a pneumothorax may be present. Management options include conservative treatment and surgical intervention. Most cases do well with conservative management of monitoring in-hospital and oxygen therapy if there is any distress. The tear usually heals and signs improve within a few days with this plan. However, if signs worsen or persist, further imaging, such as by bronchoscopy, may be required. Surgical intervention can be pursued to repair the tear if indicated.
CARDIOPULMONARY ARREST

Cats are twice as likely as dogs to experience cardiopulmonary arrest (CPA) in the peri-anesthetic period. Many arrests occur in the post-anesthetic period so close patient monitoring should continue beyond extubation. Witnessed CPA allows for rapid institution of chest compressions, which is one of the most important determinants of survival from CPA.

**Chest compressions** should begin as soon as the cat is determined to be non-responsive and making no attempts to breathe. Compressions should be made just behind the elbow, over the heart, at a rate of 100 – 120 per minute. Adequate force should be applied to compress the thorax by ~1/3 its width in a 1:1 cycle of compression and complete decompression to allow for chambers to refill. Each CPR “cycle” lasts 2 minutes, and the person delivering compressions should trade at every cycle with no more than ~10 seconds between cycles.

**Airway and breathing** should be addressed after chest compressions are initiated. After an ET tube is placed, secured, and its cuff inflated, positive pressure ventilation should start at ~10 breaths / minute at an approximate tidal volume of 10 mL/kg. Ideally, to institute advanced life support, a capnometer should be attached to the ET tube and the PetCO2 monitored.

**End-tidal CO2 monitoring:** A PetCO2 of ≥ 15 mmHg is associated with improved survival. Once a capnometer is attached to the ET tube, the person delivering the breaths reports the PetCO2 to the compressor regularly. With a stable breathing pattern, the PetCO2 becomes a monitor of circulatory efficacy (it “reports” that fresh aliquots of venous blood are returning to the lung). Thus, if the PetCO2 is < 15 mmHg, the compressor should alter the compression strategy by switching compressors, altering hand position, or changing depth or rate of compression in an effort to improve the PetCO2.

**ECG** should be attached during the first 2-minute cycle if available. The ECG rhythm is evaluated every 2 minutes during the 10 second break between compressors. Ventricular fibrillation or pulseless ventricular tachycardia (a ventricular rhythm in excess of ~200 / minute in absence of a pulse or palpable apex beat) are both treated with electrical defibrillation. Asystole or pulseless electrical activity (pulseless complexes with rate < 200 / minute) are treated with continued 2-minute cycles of CPR.

**Epinephrine and atropine** should both be administered in most cases during the first cycle, and low-dose epinephrine repeated every 2d cycle throughout the CPR effort. Published guidelines state that epinephrine should not be given if electrical defibrillation is indicated, and so one should rule out ventricular fibrillation before giving epinephrine. However, newer literature suggests that epinephrine should be given every other 2-minute cycle regardless of ECG rhythm appearance. Therefore, it is clinician preference whether to administer epinephrine in the face of a shockable rhythm. At our institution, we administer low-dose epinephrine in the first cycle of CPR, even though the ECG rhythm is unknown at that time.

**Reversal agents** are particularly important in peri-anesthetic CPR, and are given following the initiation of basic life support, attachment of ECG and capnometer, and consideration of epinephrine (+/- atropine). Atipamezole, naloxone, and flumazenil should ideally be administered if the paired sedative / anesthetic drug has been given within the past few hours. Doses of these reversal drugs are listed on the CPR drug dosing chart produced by the Veterinary Emergency and Critical Care Society. This chart also includes doses for other important CPR drugs and defibrillation.

REFERENCES AND FURTHER READING

Delivery of oxygen to tissues is critical to life and is determined by arterial oxygen content and cardiac output. Cardiac output is the product of heart rate and stroke volume, which is the volume of blood ejected into the aorta during a single cardiac cycle. When stroke volume falls, whether due to decreased blood volume, cardiac disease, or another problem, cardiac output can be supported with an increase in heart rate. This compensatory sinus tachycardia is one of the 6 classic clinical signs of shock.

Preload is the stress placed on the myocardium at the end of diastole, and stroke volume increases when preload increases. One major determinant of preload is end-diastolic left ventricular volume, or the volume of blood within the left ventricle just before cardiac contraction. Thus, patients with poor cardiac output due to poor stroke volume often benefit from having their left ventricular volume increased by means of blood volume expansion with intravenous fluids. Patients that are fluid replete but remain hypotensive or poorly perfused may benefit from inotropic or vasoactive drugs. The purpose of these proceedings is to provide an overview of fluid types and appropriate volumes in hypovolemic patients, and to discuss basic monitoring of adequacy of fluid resuscitation.

FLUID TYPES
Fluids used for resuscitation in cats include isotonic and hypertonic crystalloids, synthetic colloids, and blood products. Crystalloid fluids are used most commonly and are fluids made of water, electrolytes, and organic anions, all of which freely pass between the vascular compartment and the interstitium that bathes the cells. Common isotonic crystalloids in the United States include Plasmalyte 148, Plasmalyte A, Normosol R, lactated Ringer’s solution, and 0.9% NaCl. A fluid is considered “isotonic” when its sodium concentration is close to plasma sodium concentration. Hypertonic saline (usually 7.2% or 7.5% NaCl) is the only hypertonic crystalloid in common use for resuscitation in cats, and is usually reserved for resuscitation of cats with acute traumatic brain injury. The sodium concentration in hypertonic saline is far higher than that in plasma. Table 1 contains information about some common crystalloid fluids.

Synthetic colloid fluids are isotonic crystalloids that contain dissolved synthetic colloidal molecules that do not freely pass between the vascular space and the interstitium. These fluids tend to stay within the intravascular space for a longer period of time than do non-colloidal fluids. Common synthetic colloids used in the United States are hydroxyethyl starches including 6% hetastarch solutions in 0.9% NaCl, 6% hetastarch solutions in balanced, buffered electrolyte solutions (such as Hextend), and tetrastarch solutions (such as Vetstarch). Table 2 contains information about these common synthetic colloidal fluids. Synthetic colloids have received attention recently because studies have found an increased risk for AKI and mortality in some people given bolus doses of synthetic colloids compared to those treated with crystalloids alone. The risk in cats is not known, but there is concern that cats could also be adversely affected by colloid fluid boluses, particularly considering their apparent predisposition to kidney injury. Two recent clinical studies in cats that received (relatively low daily doses of) tetrastarch solutions did not show an increased risk of acute kidney injury in cats that received tetrastarch compared to those that received only crystalloid fluids.

There has long been debate about the performance and superiority of one type of fluid over another in both human and veterinary medicine. Definitive results of this debate have been elusive, and thus many fluid choices still exist. Because of the association between kidney injury and mortality in some groups of people receiving bolus doses of synthetic colloids for resuscitation, synthetic colloids have fallen out of favor for resuscitation in some critically ill human populations. There is also recent literature in people that supports the superiority of balanced crystalloid fluids over 0.9% NaCl. However, because animals with many different underlying problems require fluid resuscitation, it is probably fortunate that we have the many choices we do when it comes to fluid therapy. Balanced isotonic crystalloid fluids are generally the fluid of choice for volume expansion in cats.

APPROPRIATE FLUID VOLUMES FOR RESUSCITATION
The fluid volume used for blood volume expansion in hypovolemic cats is based on blood volume rather than on percentage dehydration or maintenance requirements. Fluid resuscitation should be given intravenously and through a fairly low resistance system (as short a length and as wide a bore/lumen as practical).
There is not a single dose that is appropriate for all hypovolemic cats; rather, the dose should be chosen first based on an estimate of severity of hypovolemia, and additional doses administered based on careful, frequent, serial monitoring of cardiovascular status.

A full “shock dose” of isotonic crystalloid for a cat is a full blood volume of 45 – 60 mL/kg of body weight given over no more than 60 minutes. If a patient is deemed hypovolemic by physical examination or other means, an initial “bolus” should be at minimum 15 mL/kg of isotonic crystalloid over 10 – 20 minutes. For cats it is safest to use a syringe or fluid pump or a 250 mL bag of fluids, to avoid accidental severe fluid overload when using a pressure bag or gravity drip and a 1 L fluid bag.

**MONITORING FLUID RESUSCITATION**

**Physical Perfusion Parameters**
The 6 physical examination parameters that help assess perfusion are mental state, mucous membrane color, capillary refill time, heart rate, pulse quality, and extremity temperature compared to core temperature. While the physical examination is not necessarily “advanced,” it is cheap, easy, and requires no special equipment. These parameters should be assessed in all acutely ill animals, and frequently in all critically ill animals.

Palpable pulse pressure variation with respiration suggests hypovolemia, and a fluid bolus should be administered unless there is a specific, clear contraindication. A recent study in cats presenting to an emergency room revealed that pulse palpation by an experienced emergency clinician correlated with systolic blood pressure. Cats with strong metatarsal pulses had a median systolic blood pressure of 135mmHg, while absent metatarsal pulses correctly classified cats with a systolic pressure of ≤ 75mmHg in a majority of cases.

Unfortunately, physical perfusion assessment is an insensitive test for hypoperfusion: many animals with poor oxygen delivery or use have perfusion parameters that are normal or so mildly abnormal that they are missed or attributed to something else. Thus, cardiovascular compromise may be missed without further investigation and any animal at risk for cardiovascular compromise (excessive losses, lack of intake, systemic inflammation, others) should have more extensive cardiovascular evaluation.

**Non-Invasive Blood Pressure Measurement**
Doppler blood pressure measurement is one of the most common ways cats are assessed cardiovascularly. Many clinics use this measure as part of their triage examination, which is standard of care in human medicine. Blood pressure is the product of cardiac output and systemic vascular resistance (degree of systemic vasoconstriction). Thus, when cardiac output drops due to hypovolemia, systemic vasoconstriction helps support blood pressure. Systemic blood pressure is one of the most well-defended physiologic parameters because it determines cerebral, coronary, and renal blood flow. Therefore, hypotension indicates very seriously compromised circulation, and normotension (or even mild hypertension) does not rule out hypovolemia. It is appropriate to use improvements in blood pressure as a sign of fluid responsiveness, but a normal blood pressure cannot be used to determine that a patient is fluid replete.

**Blood Lactate Concentration and Base Excess**
Blood lactate concentration and base excess on an acid-base panel can be helpful in determining degree of anaerobic metabolism, which occurs when oxygen delivery to tissues is poor. Precise normal reference intervals vary, but in general, blood lactate concentration ≥ 2.5 mmol/L or a base excess more negative than -4 to -6 are associated with tissue hypoperfusion. Blood lactate concentration and acid-base status are now widely used in human and veterinary medicine as methods to track perfusion status over time. Serial values are more valuable than single time-point measures to determine adequacy of perfusion. Blood lactate and acid-base status must be evaluated immediately after the blood sample is drawn, as a delay of only 30 minutes can lead to lab error. Serial evaluation on an every 1 – 4 hour basis is reasonable in the initial resuscitative period.

Lactated Ringer’s solution contains sodium lactate, and it is safe to use as a resuscitation fluid in patients with lactic acidosis. However, the lactate anion in the fluid can complicate serial blood lactate measurements, particularly in patients with certain disease processes such as lymphoma and liver failure, in which lactate metabolism can be compromised. The lactate itself is not dangerous to the patient, but it can render serial blood lactate evaluation less useful as a marker of perfusion in these specific cases.
Normosol-R or Plasmalyte 148 (A) may be better choices if serial lactate concentration will be used as a guide in animals known to have problems metabolizing lactate (liver insufficiency, lymphoma), because these 2 fluids do not contain lactate.

**Bedside Echocardiography**

When an ultrasound machine is available, a brief echocardiogram may be helpful to assess volume status. Complete or near obliteration of the left ventricular lumen at end-systole is consistent with volume depletion in patients with otherwise normal hearts (i.e., without cardiomyopathy — this can be hard to guarantee in a cat at the bedside).

Echocardiography can also be helpful to determine whether a patient is likely to tolerate a fluid challenge, in the event that primary cardiac disease is suspected. Does a brief cardiac ultrasound reveal obvious atrial enlargement? When the ultrasound probe allows a good view at the level of the aortic valve, measure or estimate the left atrial-to-aortic (LA:Ao) ratio if possible. A ratio of > 1.5 is consistent with meaningful heart disease, and while this would not necessarily preclude fluid administration, it suggests that one should be cautious.

**Central Venous PO\textsubscript{2} (PcvO\textsubscript{2})**

Central venous catheterization with a jugular catheter allows measurement of central venous oxygenation status, either as central venous O\textsubscript{2} tension (PcvO\textsubscript{2}) or as central venous O\textsubscript{2} saturation (ScvO\textsubscript{2}). ScvO\textsubscript{2} measurement is the gold standard, because PcvO\textsubscript{2} and ScvO\textsubscript{2} can convey somewhat different information during critical illness due to alterations in hemoglobin binding of oxygen. However, most private practices have blood gas analyzers that calculate SO\textsubscript{2} (often from a human algorithm) from PO\textsubscript{2} rather than measuring SO\textsubscript{2} directly. Therefore, saturation information from these analyzers must be considered estimations rather than precise values.

Tissues with decreased oxygen delivery extract a higher percentage of oxygen from the capillary blood than tissues with normal oxygen delivery. Therefore, the partial pressure of oxygen in the venous blood that returns to the right side of the heart is decreased compared to normal. Normal ScvO\textsubscript{2} is > 70%; values below this suggest global tissue oxygen deficit and should prompt the clinician to initiate or continue resuscitation. ScvO\textsubscript{2} can be analyzed repeatedly during resuscitation to evaluate efficacy — this value will change rapidly with improved perfusion, so one can monitor every 15 – 30 minutes to monitor cardiovascular status during dynamic periods. Very small quantities of blood (< 0.5 mL per sample) are usually required.

**Fluid Challenge as a Diagnostic Tool**

Sometimes it is not possible to guess, even when monitoring the parameters above, whether or not a patient may benefit from rapid intravascular volume expansion. Sometimes even if these parameters could be helpful, they cannot be accomplished either due to time, availability, or cost. In such cases, a fluid challenge is a reasonable method to assess a patient’s fluid responsiveness: give the patient a little fluid and see what happens. A fluid challenge is usually performed by intravenous administration of a small percentage of blood volume, such as 5 – 10 mL/kg of isotonic crystalloid in a cat, over 10 – 20 minutes, and watching closely for improvement in clinical signs, lactate concentration, or other well-defined outcome measures. A successful fluid challenge would be expected to drop the heart rate, if not to normal, at least by a bit, for at least 10 – 20 minutes. If a patient responds to the fluid challenge but perfusion parameters (or other objective indices) remain abnormal, more intravenous fluids should be administered as clinically indicated. Objective endpoint goals such as a decrease in blood lactate concentration by X or a heart rate of Y can be helpful in deciding when a patient has received an adequate fluid volume. If the patient is clearly in need of intravenous volume expansion, a fluid challenge of these small volumes is not appropriate — such animals require larger volumes of fluid for support.

**REFERENCES AND SUGGESTED READINGS**


Cats scratch as prose and are hard-wired to do this, it’s a normal feline behavior. The reasons are:

- Olfactory communication depositing feline interdigital semiochemical
- Send a visual cue
- Replace worn nails sheaths
- Exercise/A good stretch/It feels good

As more cats were being kept indoors (in conjunction with the invention of kitty litter), the idea of declaw surgery was first presented in a letter to the editor in the Journal of the American Veterinary Medical Association in 1952 by a Chicago veterinarian without first studying short-term or long-term effects, it was just an idea.

“Onychectomy is an amputation and should be regarded as a major surgery”

A Declaw is removal of the third phalanx.

If this surgery was conducted on a human, the last section of each finger would be cut off at the knuckle.

Tendonectomy: The tendon that controls the claw in each toe is severed. The cats keeps the claws, but can't control them or extend them to scratch.

How common is declaw?

- 21% of cats at vet hospitals near Raleigh (2013)
- 24% Paternek study (2001)
- 45% of cats in Indiana telephone survey (1997)
- 24% study published in JAVMA (2005)

Reasons for declaw

- Destructive scratching: Keeping cats in homes
- Concerns about scratching children
- Immune suppressed or seniors in the home
- Autistic children

In regard to children, seniors or immune-suppressed individuals, the U.S. Centers for Disease Control does not suggest relinquishing the cat or declaw. Instead:

- Cats live inside (less prone to parasites)
- Cats have appropriate flea protection (Bartonella exposure)
- Play using interactive cat toys
- If declawed may be more likely to bite (potentially a more serious issue for this population).

What happens to cats? They land in a shelter:

1.4 million feline euthanasias in shelters annually, at least half are a result of behavior.

- Inappropriate elimination, 37 to 43%; Aggression, 10 to 18%; Destructive Scratching 12%
- Inappropriate elimination 33%; Biting 14%; Intolerant of Children 11%; Scratching People 11%; Destructive Scratching 8%

Conflicting evidence

- Onychectomy has long and short-term complications, including pain, hemorrhage, soft time swelling, nerve trauma, infection, lameness (Paternek 2001; Mission et al 2002)
- Some suggestion declawed cats are more likely to bite and eliminate inappropriately (Paternek 2001; Yeon et al 2001)
- Phantom pain experienced by people – we can’t ask cats. “Pain literature suggest it’s likely” (Downing, 2016).
- When pain is poorly managed at the outset…this means ongoing, perpetual, self-sustaining chronic maladaptive pain that constitutes life-long torture (WSAVA, 2014; Costigan, 2009; Dahl, 2011).
- Approximately 60% of a cat’s body weight is carried on front feet, altered biomechanics changes the way cats move. At the very least, development and progression of osteoarthritis (Downing 2016)
• Association between declaw and back pain, aggression and barbering, as well as inappropriate elimination. Odds of relinquishment likely greater than cats scratching. Biting aggressive cats more dangerous than cats who may scratch, usually by accident. “In view of these findings the on ongoing practice of declawing cats should be further questioned.” (Martell-Moran, Solano, et al, 201715)
• 18 declawed cats with a history of missing their litter boxes a two-week trial of a pain relief drug (Buprenorphine) and 90 percent begin to use their boxes regularly again. (Gaskin, 2017) 6
• Many declawed cats develop hyperflexion, or club-footedness. A callous on the hyperflexed digit paw pad is common and an abnormal condition. Walking on the amputated toe tips is painful, and this chronic pain worsens over time. Pain is so intense that there’s a relationship between cortisone levels heightened due to pain and increased diabetes in these cats, and inappropriate urination. Owners may not report pain but do report changes in cats’ personalities. Some bite. Some hide more. With a change of gait, arthritis more likely occurs. (Gaskin, 2017) 6

When clients have a cat that’s scratching in all the wrong places – what now?
• Behavior modification and / or
• Pheromone product that attracts cats to posts4, 20
• Punishment only destroys human-animal bond2,3,4
• What can help some is facial pheromone (Classic Feliway) 21
• for other stressors, catnip, frequent nail trips or plastic nail caps 2,3,4

So, why do cats scratch inappropriately? 2, 4, 5
• No posts or not enough (per number of cats in the home)
• Vertical and horizontal choices
• Poor location of posts
• Substrate choices
• Never encouraged or trained cats to use posts
• Poor posts (not sturdy, not tall)
• Once they done it – it’s theirs

How many posts? We don’t really know that….maybe the answer is the same for litter boxes, one per cat plus one. And we don’t even know for sure where that number derives. We do know, the more resources and options (vertical and horizontal) the better 2

Location of posts matters2
• Near where family is
• Near favorite napping location (good stretch when waking)
• Not next to another vertical scratcher (not side by side)
• As a part of feline aerobic center
• Near windows
• Near door most often used by family

Teaching cats to use posts2, 22
Tools
• Catnip
• Feliway Classic

Training
• Clicker training
• Observational learning
• Physical have the cat scratch (gently)
• Coax with a toy

It’s simple
Never punish ----makes places your clients you don’t want unattractive and places you do want more attractive (to scratch) 2,4,5, 22

Tips to deter
• Keep nails clipped 2
• Soft Paws (nail caps) 23
• Sticky Paws 24
• Rug runner upside down/car mat upside down2
• SSSCAT! 25
• ScatMat 26
Feliscratch by Feliway attracts cats to posts and contains: 27, 28
- Feline Interdigital Semiochemical (FIS)
- Blue dye
- Catnip

Pilot study indicates Feline Interdigital Semiochemical (FIS) pheromone does attract cats away from ‘target area,’ where they have been scratching. 29

In a test of FIS vs placebo, where each cat was test twice, the FIS post was scratched longer and more frequently. 20

Study combines catnip with blue dye and FIS with 166 cats in 117 homes.

All cats – unwanted scratching, no use of medications over the past three months, no use of pheromone products over the past four weeks, no more than two cats in the homes, health check and no declaw.

Families provided with new posts – and told to remove old ones. Directions about where to place posts.

Product provided in pipettes with instructions to apply it by drawing a single vertical line on the post once daily for 6 days and then again at day 14, 21, 28

And a 5-pt. Likert scale used for owner assessment

0-no; 1 yes 1x daily, 2 yes 2-3 x daily, 4 yes several times daily

Pheromone product with FIS, Catnip and blue dye: 79.9% success scratching within seven days, and 87 percent at day 28. With behavior modification, wonder if it could be 100 percent effective?

Supplemental materials

The current prevalence of onychectomy (declawing) in cats is unknown

Almost 21% of cats seen in veterinary hospitals near Raleigh, NC were declawed. Less than 50% of veterinary schools in the USA include a mandatory lecture or laboratory to teach the procedure.

Onychectomy in cats is a controversial elective procedure that involves the removal of the third phalanx using a guillotine-type nail clipper, surgical blade, or laser (Shwartz 2001; Swiderski 2002). The onychectomy procedure is legal and performed throughout most of the United States, except in eight California cities, where the procedure has been banned (Whitcomb 2010)

The most common indication for performing onychectomy is unwanted scratching behavior causing personal injury or property damage (Patronek 2001; Shwartz 2001).

Other indications for onychectomy include medical conditions of the patient, such as paronychia or nail bed neoplasms, and to prevent spread of zoonotic disease, as might occur in owners with immunodeficiency (Mison et al. 2002; Atwood-Harvey 2005).

Onychectomy has both long- and short-term complications including pain, hemorrhage, soft tissue swelling, incisional dehiscence, infection, draining tracts, nerve trauma and lameness (Patronek 2001; Mison et al. 2002). It has also been suggested that onychectomy may cause stress in cats due to their inability to perform natural behaviors (Patronek 2001). Some veterinarians consider elective onychectomy unethical and refuse to perform the procedure (Patronek 2001).

24.4% of owned cats were onychecutomized (declawed) (Patronek 2001). A local telephone survey conducted in Indiana in 1994 found that 45% of household cats had undergone onychectomy (Patronek et al 1997).

Force plate analysis of cats following onychectomy has demonstrated that cats have abnormal gaits for at least 12 days after surgery (Romans et al. 2005).

Not only is there a shortage of information regarding the prevalence of declawed cats in the USA, there is no compiled information regarding the instruction of veterinary students in the onychectomy procedure. Anecdotal information suggests that USA veterinary schools vary in the methods used to teach the onychectomy surgery and even whether the procedure is taught at all.

Table 2

<table>
<thead>
<tr>
<th>Practice type</th>
<th>Total number of cats</th>
<th>Onychectomized cats n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>General practice</td>
<td>410</td>
<td>123 (30.0)%</td>
</tr>
<tr>
<td>Cat-only practice</td>
<td>1170</td>
<td>211 (17.9)%</td>
</tr>
<tr>
<td>Tertiary practice</td>
<td>214</td>
<td>40 (18.7)%</td>
</tr>
</tbody>
</table>

The prevalence of onychectomy in cats presented during a 10-week period to five veterinary practices (categorized by practice type) near Raleigh, NC. Differing superscripts denote significant differences onychectomized practice type Total number of cats cats n (%) in the prevalence of onychectomy in cats apparently has not changed in the last decade.

It has been suggested that declawed cats are more likely to bite and eliminate outside of their litter box (Patronek 2001; Yeon et al. 2001) Almost 93% of USA veterinary schools responded to our survey, and all geographic areas were represented.

Of the veterinary schools that responded, more than half (54%) have no mandatory lectures or laboratories in their pre-clinical curriculum to teach the onychectomy procedure and 12% of the responding schools do not offer formal instruction or clinical opportunities to learn the procedure.
Since 20.8% of cats seen in this study were declawed and that the percentage of onychectomy was higher in younger cats, it is clear that onychectomy surgeries are still being performed rather frequently. A discrepancy exists between how commonly cats are undergoing onychectomy and the amount of emphasis placed on its instruction in veterinary schools.

The lack of formal instruction in onychectomy potentially has far-reaching effects. The obvious concern would be the potential for poor surgical technique that could increase the prevalence of short and long term complications. In addition to surgical technique, there would also be concern for the lack of instruction of anesthetic and analgesic techniques appropriate for what most veterinarians consider a procedure that results in severe pain.

**Nail care, cat healthy healthcare protocols**

It is also important for the veterinarian to help clients understand what is involved in the actual surgical procedure of declawing, explaining that the procedure involves amputation of the last bone (P3) of each digit.

In past years, the practice of tendonectomy had been recommended as an alternative to declawing. This procedure involves surgical severing and/or removal of a short length of the deep digital flexor tendon for each digit. This prevents the cat from being able to expose the claws and will prevent all scratching activities. However, the procedure leaves the cat without the ability to shed the cap of growing, healthy claws, leading to painful thick nails that are difficult to trim and that are predisposed to growing into the paw pads. Therefore, tendonectomy is NOT recommended.

It is important to note that laser declaw does not prevent surgical pain. In two studies examining post operative declaw pain in cats declawed by scalpel or laser, neither study concluded any long-term (greater than 2-5 days) reduction in post-operative pain when the procedure was performed by laser and laser declaw patients were never observed to be pain-free (Holmberg et al, 2006, Robinson et al, 2007, Wilson & Pascoe, 2016).

In some cases, declawing is requested in order to protect an immunocompromised person from scratching and related disease. The United States Center for Disease Control (CDC) does not recommend onychectomy as a means of disease control even in these instances. The CDC recommends an indoor lifestyle for cats living with immunocompromised individuals and regular flea prevention as a means of reducing the risk of exposure to Bartonella spp (Cat Scratch Disease).

It discourages immunocompromised individuals from playing directly with young cats. This recommendation should be extended to any type of play involving one’s hands or feet with any age of cat as it might lead to biting or scratching. Clients should always avoid aggressive play with any cat, as it can increase the incidence of play aggression and the risk of injury to humans living with the cat.

**Feline toe amputation statement (Robin Downing, DVM)**

Amputating the last phalanx (P3) of the toes of cats was once considered a “commodity” procedure, commonly performed by well-intentioned veterinarians. As time has passed and our understanding of feline pain, biomechanics, and quality of life has evolved, we now recognize many downsides to this procedure and truly NO upside.

Cats are sentient beings with moral agency who, it has been recently argued (Andrews 2011; Copp 2011; Downing 2016; Nussbaum 2015; Panskeep 2012), should be approached with the same consideration as nonverbal children. As beings with moral agency, it behooves us to consider them within the context of the foundational principles of clinical bioethics.

Nonmaleficence means “do no harm” or “avoid harm”. Considering feline toe amputation, the question then becomes, “Does amputating all of a cat’s front toes (P-3) cause harm?” Amputation is painful, potentially for the rest of the cat’s life, it forever alters the way a cat walks, it prevents natural (scratching) behavior, and it forever prevents the cat from being able to defend itself by escaping (climbing) or fighting. Clearly toe amputation causes harm.

Beneficence means to act in a being’s best interest. Can we truly argue that amputating all of any cat’s third phalanges of the front toes is ever in that cat’s best interest? It appears that the answer to this question is a self-evident ‘no’.

Finally, the fourth cornerstone principle of clinical bioethics is justice. Translating this for application in veterinary medicine focuses on fairness. The relevant question to ask is if amputating the third phalanx of each a cat’s front toes could ever constitute fairness to the cat within the context of its life and lifestyle. Considering all of the compromise that toe amputation creates, I respectfully submit this does not reflect fairness.
Pain perspective
Considering feline toe amputation from a pain perspective, multiple studies have demonstrated that most cats receive woefully inadequate pain prevention and management for procedures like spays and neuters -- procedures far less traumatic than multiple toe amputations. The pain literature clearly demonstrates that acute pain poorly managed at the time of the trauma often leads to the establishment of permanent pain states. This means ongoing, perpetual, self-sustaining chronic maladaptive pain that constitutes lifelong torture (AAHA/AAFP 2007; WSAVA 2014; Costigan 2009; Dahl 2011) The few studies that have evaluated either the presence of leftover bone fragments following toe amputation, or the regrowth of sharp bone spurs following amputation, demonstrate that an embarrassingly large number of cats suffer from this extra boney tissue. These sharp shards perpetually poke at the underside of the skin at the end of each toe stump, making every single step like walking on needles or nails.

Finally, we know from pain physiology that when we sever a nerve there is a very high risk of creating an ongoing, self-perpetuating pain state called “neuropathic pain”. Humans most commonly develop neuropathic pain as a result of conditions such as amputation, direct nerve trauma, shingles, and diabetes. People who develop neuropathic pain can describe how it feels, so we know quite well the unremitting torture they endure each and every day - - tingling, burning, electric-like pulsed pain, pins and needles. We also know quite well that once chronic, maladaptive, neuropathic pain is in place, these people report ongoing challenges to relieving pain (Sandkuhler 2006; Woolf 2006; Woolf 2004).

Biomechanical perspective
Because approximately 60% of the cat’s body weight is carried on the front feet, altered biomechanics changes the way the entire body moves. If we superimpose chronic, maladaptive, neuropathic pain in the feet onto altered front foot biomechanics, we amplify the downstream implications of the cat moving in an abnormal fashion. The altered biomechanics can significantly interfere with the cat’s ability to exhibit normal cat behaviors. We also know that the vast majority of cats 10 years of age and older suffer from degenerative osteoarthritis (OA) in at least one joint (Kerwin 2010; Lascelles 2010). The majority of cats who develop OA in later life have it occur in their equivalent of the human lower back - - where the spine and pelvis come together. When the biomechanics of movement are altered, so are the forces generated throughout the body’s joints - - in particular the joints of the spine. The repetition of ergonomically unsound movements creates over time micro-traumas to these joints which can contribute to the development and progression of OA. OA, then, provides these cats with ongoing chronic maladaptive pain.

Pain and adverse behavior in declawed cats (Nicole K Martell-Moran1, Mauricio Solano and Hugh GG Townsend)

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<th>Table 2 Multivariate models of factors significantly associated with back pain and adverse behavior in 274 declawed and non-declawed cats</th>
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<td>Factor</td>
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OR = odds ratio; CI = confidence interval

Among the 137 declawed cats, 86 (63%) showed radiographic evidence of residual P3 fragments. Of these, 31 (36%) had P3 fragments measuring <25% of the bones, 29 (34%) had fragments equivalent in size to 25–50% of the P3, and 26 (30%) showed evidence of having had only the ungual process removed. Four cats with externally visible nail regrowth had only the distal end of the ungual process removed when declawed. the 137 (24%) cats were declawed on all four limbs.
The current study shows a clear association between declawing and the presence of deleterious side effects after the typical postoperative period in a comparatively large sample population. The primary analysis of the cohort data comparing declawed cats and a non-declawed control group shows that the odds of the highly undesirable habits of elimination, periuria and/or perichezia were much greater in declawed cats than their controls. In addition, declaw surgery was associated with a significant increase in the odds of back pain, biting, aggression and barbering.

Many cats express pain with a behavioral change such as biting, aggression or inappropriate elimination. Clinically, we have observed that pain arising from the lower back is associated with inappropriate elimination. Similarly, if the source of pain is declaw phalanges, the act of walking on or digging in a gravel-type substrate may result in pain and aversion to use of the litter box. Many cats that eliminate outside of the litter box choose a soft substrate such as carpet, clothing or a location next to the litter box like a mat. With respect to aggression, following claw removal, a cat’s only defense when upset or fearful is biting. When touched, painful, fearful or stressed declawed cat may react by attempting to bite as it has few or no claws to scratch with.

During the physical examination of the cats in this study, many biting attempts occurred when cats were lifted, creating an arched back; when they were touched or petted caudal to the middle thoracic vertebrae; or in anticipation of pain when a handler was reaching to touch the lower back or tail.

The removal of a cat’s distal phalanges forces it to bear weight on the soft cartilaginous ends of the middle phalanges (P2) that were previously encapsulated within joint spaces. In this study, 11 declawed cats showed radiographic evidence of remodeling of the P2 bone. The significance of bone remodeling is unknown and was not explored in this study. There is currently no study that addresses the anatomic and pathologic changes affecting the P2 bone and cartilage that may incur over the declawed cat’s lifetime. The potential for effects on the rest of the musculoskeletal system such as weight bearing among other joints, arthritic changes, chronic pain elsewhere in the body or changes in bone density needs focused research. Based on the present study, a minority of cats showed remodeling of the middle phalanx.

The presence of P3 fragments in 63% of declawed cats is excessive and surprising. It reflects the use of poor or inappropriate surgical techniques, leading to increased odds of adverse outcomes in declawed cats. The primary analysis of the data related to all 274 cats in the study shows that declawed cats with P3 fragment retention are at greater odds of experiencing biting and inappropriate habits of elimination as compared with declawed cats without P3 fragment retention. To further explore the impact of P3 retention, a secondary analysis, limited to the 137 declawed cats, showed that cats with retained P3 fragments were at increased odds of back pain, periuria and/or perichezia and aggressive behavior when compared with declawed cats without fragments.

This study found that declaw surgery in cats was associated with a significant increase in the odds of developing adverse behaviors, including biting, barbering, aggression and inappropriate elimination, as well as signs of back pain. There was a high prevalence of P3 fragments in declawed animals in this study and this was associated with an increase in all adverse outcomes in these animals compared with the non-surgical controls. As well, declawed cats with retained P3 fragments had higher odds of back pain, inappropriate elimination and aggression when compared with declawed cats without retained fragments. Although cats receiving optimal surgical technique had fewer adverse outcomes and lower odds of these outcomes being present, these animals were still at increased odds of biting and undesirable habits of elimination as compared with non-surgical controls. We propose that persistent pain and discomfort subsequent to declaw surgery is an important risk factor for the development of behavioral changes such as biting, aggression, barbering and inappropriate elimination. These are common reasons for the relinquishment of cats to shelters. In view of these findings, the ongoing practice of declawing cats in North America should be further questioned.

**Attitudes of owners regarding tendonectomy and onychectomy in cats (Seong C. Yeon, DVM, PhD; James A. Flanders, DVM, DACVS; Janet M. Scarlett, DVM, MPH, PhD; Stacey Ayers; Katherine A. Houpt, VMD, PhD, DACVB)**

Recently, the merits of the 2 surgical techniques have been debated.10,15 Jankowski et al10 found that cats that underwent tendonectomy recovered more quickly according to the owner, and owner satisfaction was similar after onychectomy and tendonectomy. However, to our knowledge, a clinical behavioral study has not been performed to compare owners’ attitudes regarding tendonectomy and onychectomy. The purposes of the study reported here were to compare owners’ reasons for surgery and changes in the cat’s behavior (including use of the paws) following each procedure. We also sought to compare owners’ assessment of recovery time, benefits and concerns regarding each surgical procedure, and overall attitude.

Tendonectomy has been used as an alternative method to decrease destructive scratching; it is recommended to owners that have humane concerns or those who are worried about problems that may develop after onychectomy. 2,9 Results of 1 study revealed tendonectomy was associated with fewer signs of pain than onychectomy, but nail trimming must be performed at regular intervals after surgery.9

Results of another study revealed that although there was no significant difference, more owners of cats that underwent onychectomy were satisfied with the procedure, compared with owners of cats that underwent tendonectomy; the prevalence of lameness between cats undergoing onychectomy and those undergoing tendonectomy was similar following surgery.10

Although nail trimming of thick claws may be a problem, 17 of 18 (95%) owners of cats that underwent tendonectomy in our study had a positive attitude toward the surgery, compared with 34 of 39 (87%) owners whose cats underwent onychectomy. In our study, 1 cat that underwent tendonectomy had prolonged lameness, which was the same number as that described after onychectomy.
A common reason for considering tendonectomy or onychectomy was that owners wanted to prevent their cats from scratching household materials. When owners considered injury to humans, fewer owners of cats that underwent tendonectomy considered this problem than those whose cats underwent onychectomy. A larger proportion of owners selecting tendonectomy had the procedure recommended to them by a veterinarian or someone else. Tendonectomy was typically not performed at the time of neutering but later, when problems arose. Veterinarians or others rarely recommended onychectomy, but that is most likely because owners were already aware of that procedure. Tendonectomy is a newer procedure and as such maybe recommended by veterinarians or others from whom the owner seeks advice.

**Applied animal behavior science**

There are an estimated 1.4 million feline euthanasias in US shelters every year, half of which are a result of behavioral problems (ASPCA, 2015; Salman et al., 2000). The top three behavioral reasons for relinquishment of a cat to a shelter are inappropriate elimination (37–43%), aggression (10–18%), and inappropriate scratching (approximately 12%) (Patronek et al., 1996; Salman et al., 1998, 2000). If declawing, which addresses inappropriate scratching, actually causes an increase in inappropriate elimination or aggression, as is asserted by opponents, then the declawing of cats would put them at higher risk of surrender and euthanasia. If declawing does not cause behavior problems but is assumed to declawed cats could be at higher risk for lack of adoption at shelters (leading to euthanasia in shelters that cannot hold animals indefinitely).

There was no association between declawing and biting behavior in these 1013 shelter cats. This study supports the findings of Declawed cats were underrepresented in this shelter population. This could be the result of a protective effect offered by declawing, as has been hinted by trends in past studies (Patroneket al., 1996). Declawed cats are by definition incapable of destructive scratching and may not anger the owner as often as intact cats, leading to a better relationship (Landsberg, 1991a). Also, declawing is an expensive procedure and it may be the case that declawed cats already have desirable temperaments and behaviors other than destructive scratching. Given this, it is possible that the owner is willing to invest more money in the cat, and is therefore less likely to surrender the cat to a shelter (Bennett et al., 1988). Overall, owners tend to be pleased with the results of declawing (Landsberg, 1991a; Yeon et al., 2001). It is also possible that the underrepresentation of declawed cats in this shelter is a reflection of changes in the rate of declawing: the most recent estimate of the number of declawed cats in the US (24.4%) is 14 years old (Patronek, 2001).

However, one recent study looked at the prevalence of declawing in cats near Raleigh, NC and found that almost 21% of cats were declawed (Lockhart et al., 2014). Clearly declawing is still prevalent, though more information is necessary to make accurate estimates.

We found that only 2% of the cats in this shelter bit during their tenure, a significantly smaller number than that claimed by surrendering owners. There are many possible explanations for this finding. It is possible that biting is more rare in the shelter than in the home; rather than getting more aggressive when in the stressful shelter environment, it is likely that cats practice a “conservation withdrawal” strategy in response to the stress of the shelter, and offer fewer of their typical behaviors, including biting (Carlstead et al., 1993; Engel and Schmale, 1972; Koolhaas et al., 1999).

Declawing is not statistically associated with biting behavior in the shelter, nor has any other study to date found an association between declaw status and any problem behavior cited as a reason for relinquishment (Bennett et al., 1988; Landsberg, 1991a; Morgan and Houpt, 1989; Patronek, 2001; Yeon et al., 2001).

**Assessment of claims of short-and long-term complications associated with onychectomy in cats**

Patronek, Gary, VMD, PhD

JAVMA, Vol 219, No. 7, October 1, 2001

Others are concerned about the adverse effects of depriving a cat of the use of its claws and the ability to engage in hard-wired species-specific behaviors such as scratching, grooming, and defense. This frustration of natural behaviors has been proposed as a cause of chronic stress.

It has even been suggested that removal of the claws inhibits normal isometric exercise of the back muscles during scratching, and that changes in how the paw contacts the ground can cause back pain, similar to that which occurs in people wearing ill-fitting shoes .18

The AVMA and the Canadian Veterinary Medical Association both take a cautious approach, characterizing onychectomy as a justifiable option to prevent scratching when a cat cannot be retrained and is in danger of being removed from the home. Some veterinarians maintain that declawed cats even appear to have a normal behavioral repertoire (catching birds and climbing trees) when they are allowed to go outdoors .24,25

In the second study, 45 client-owned cats that underwent elective onychectomy performed by veterinary student surgeons were randomly assigned to receive either butorphanol or transdermal fentanyl patches .30

Vital signs, force applied by the forelimbs, signs of pain, lameness, and appetite were evaluated before and up to 40 hours after surgery. Cats treated with fentanyl had better recovery scores at 2 of 4 evaluation times, lower sedation scores at 2 of 8 evaluation
times, and lower pain scores at 6 of 8 evaluation times. Use of a pressure-sensitive mat did not reveal any differences in force applied by the forelimbs in the 2 groups. 30

The fourth of these 6 studies compared laser and blade onychectomy. Forty cats were randomly assigned (n = 10/group) to the following groups: no anesthesia (control), bandaged and anesthetized (control), blade onychectomy, or laser onychectomy. Complications, behavioral changes, plasma cortisol concentration, and urine cortisol-to-creatinine ratios were evaluated up to 48 hours after surgery. Complications (bleeding, limping, swelling, infection) were generally worse in the laser onychectomy group in the first 2 days after surgery but were equivalent thereafter. Negative behavioral changes were more pronounced in the blade onychectomy group for 2 days, with less play and willingness to use the paws. Blood and urine cortisol concentrations were increased to a greater degree in the blade onychectomy group for 24 hours, compared with the other groups.

In 1 study, 14 early postoperative (i.e., during the hospitalization period) complications in 160 cats included signs of pain (38.1%), hemorrhage (31.9%), lameness (26.9%), and non–weight-bearing lameness (5.6%). Follow-up information was available for 121 (75.6%) cats. After discharge, 24 of 121 (19.8%) cats developed complications. Lameness persisted for 1 to 54 days (median, 2 days), was correlated with occurrence of postoperative signs of pain, and was more common in cats that underwent blade onychectomy (21/40 [52.5%]) than in cats in which guillotine shears were used (17/101 [16.8%]); however, there was no difference in persistence of long-term lameness between these techniques (1/32 [3.1%] vs 2/75 [2.7%], respectively). One cat had persistent intermittent lameness 96 months after surgery, and 2 others had lameness that resolved within 4 months. The high overall rate of complications was attributed to the fact that surgeries were performed by veterinary students. 14

The surgeries also were performed during an 8-year period, and there was substantial variation in techniques used.

Biting was reported for approximately 12% of declawed cats and sexually intact cats, house-soiling was reported for approximately 25% of declawed cats versus approximately 15% of sexually intact cats, and jumping on counters was reported for approximately 75% of declawed cats versus approximately 53% of sexually intact cats. Statistically, only the latter comparison was significant. In another survey, 34 of 252 owners of declawed cats and 613 owners of non–declawed cats in 4 veterinary hospital waiting rooms, similar numbers of declawed and sexually intact cats (60/252 [23.8%] vs 168/613 [27.4%], respectively) were reported to have bitten family members; for each group of cats, 2.3% of these injuries were serious enough for medical attention.

(3.6%) cats were reported to have a possible increase in the frequency or severity of biting; however, all owners of these cats were reported to be satisfied with the procedure. In a study of owner attitudes after onychectomy, at least 1 behavioral problem began after surgery in 13 of 39 (33%) cats. Seven of 39 (17.9%) cats began biting or had an increase in intensity of biting, and 6 of 39 (15.4%) cats would not use the litterbox after onychectomy.

The evidence for adverse behavioral sequelae is somewhat more provocative, albeit equally inconclusive. In the retrospective studies, biting and house-soiling frequencies after onychectomy varied tremendously from 17 to 1%. In the study with the highest frequency (33%) of reported behavioral problems after surgery, sufficient data were not reported to determine the response and complication rates for onychectomy alone, compared with tendonectomy, so it is possible that this proportion is somewhat inflated.

Analgesic efficacy of preoperative administration of meloxicam or butorphanol in onychectomized cats (Gwendolyn L. Carroll, MS, DVM, DACVA; Lisa B. Howe, DVM, PhD, DACVS; Kurt D. Peterson, DVM/

Conservative estimates indicate that approximately 14.4 million cats (24% of owned cats) in the United States undergo declawing.1 Short-term and long-term complications of onychectomy are ill-defined, as are the adverse consequences of failure to perform this surgery in some cats. However, the potential development of acute postoperative pain after onychectomy is a generally accepted consequence. Heightened awareness and concern for animal pain have resulted in several studies3-7, a recent that examined the treatment of acute postoperative pain from onychectomy

American association of feline practitioners

The American Association of Feline Practitioners (AAFP) strongly believes it is the obligation of veterinarians to provide cats owners with alternatives to declawing (onychectomy). Declawing is an elective procedure that is highly controversial. If owners are considering declawing, they must be provided with complete education about feline declawing, including the anatomic details of what a declaw entails (ie, amputation of the third phalanx [P3]) and the importance of proper pain management. In addition, alternatives to surgery and the risks and benefits of surgery need to be discussed. It is important that owners understand that scratching is a normal feline behavior; it is both inherited and learned. The primary reason for scratching is to maintain the necessary claw motion used in hunting Declawing and climbing. In addition, it is done to re-establish claw sharpness via ‘husk’ (or ‘sheath’) removal and to stretch the body.

Finally, it is an important means of visual and olfactory communication. Scratching can be directed to areas that owners consider appropriate.

Consider using synthetic facial pheromone sprays and/or diffusers to help relieve anxiety or stress. A synthetic feline interdigital semiochemical (FIS) on the desired scratcher has been shown to induce scratching behavior on an appropriate target.

Provide appropriate feline environmental enrichment, which must be implemented for successful behavioral modification.
Repetitive or increases in scratching behavior of indoor cats may be related to anxiety, stress, attention seeking, or lack of perceived security in their environment. Anxiety can be exacerbated by owner punishment, thus driving the cat to increase scratching behavior in the same or other locations. Most instances. There are inherent risks stance, behavioral problems, and complications with this surgical chronic neuropathic pain. Fewer than procedure that increase with age. 10

Half of veterinary schools in the USA These include, but are not limited to, include a mandatory lecture or the following: acute pain, hemorrhage, laboratory to teach this surgery, swelling, infection and nerve trauma. 11

Lack of formal training in the procedure Long-term complications include could lead to inferior surgical lameness, chronic draining tracts, technique, thereby increasing retained P3 material leading to claw the likelihood of both long- and necessary procedure for the cat in regrowth, development of palmigrade short-term complications. 10

While it has been suggested that onychectomy is acceptable to prevent spread of zoonotic disease(s) to immune compromised people, 10 current research demonstrates the greater value of proper hygiene and parasite control in the prevention of most common zoonoses. In households where cats come into contact with immune-compromised individuals, extensive education about zoonotic disease potential should be discussed and documented in the medical record. Of note, the Centers for Disease Control and Prevention does not advise declawing cats owned by HIV-infected persons; rather, these individuals ‘should avoid rough play with cats and situations in which scratches are likely.’ 13

**Telephone survey to investigate relationships between onychectomy or onychectomy technique and house soiling in cats**

Amanda F. Gerard DVM  
Mandy Larson DVM, MS  
Claudia J. Baldwin DVM, MS  
Christine Petersen DVM, PhD

Onychectomy technique was identified as a risk factor for house soiling. Cats for which a non-CDL technique was used had a higher risk of house soiling than cats for which the CDL technique was used. Cats that had undergone onychectomy and that lived in a multi-cat (3 to 5 cats) household were more than 3 times as likely to have house soiled as were single-housed cats with intact claws.

Results of the study reported here supported the hypothesis that onychectomy is associated with an increase in house soiling behavior of cats.

**References**

2. Submitted by Nancy Suska DVM Gerry Beekman DVM Paula Monroe DVM Carlye Rose DVM, DABVP (Feline; Canine & Feline), CVA.  
7. Think Twice Before You Declaw  
13. And literature review: [https://www.avma.org/KB/Resources/LiteratureReviews/Pages/Welfare-Implications-of-Declawing-of-Domestic-Cats-Backgrounder.aspx](https://www.avma.org/KB/Resources/LiteratureReviews/Pages/Welfare-Implications-of-Declawing-of-Domestic-Cats-Backgrounder.aspx), 2016,  
Effects of a synthetic facial pheromone (Feliway) on behavior of cats

24 Sticky Paws TV commercial: https://www.youtube.com/watch?v=iNhjtH-Qug.
26 ScatMat from PetSafe https://store.petsafe.net/scatmat-pet-proofing-mats.
27 Feliscratch by Feliway (CEVA Animal Health) https://www.feliway.com/uk/Products/FELISCRATCH-by-FELIWAY.
INTRODUCTION

Otitis refers to inflammation of the ear tissue. Otitis externa (OE) refers to inflammation of the external canal distal to the tympanic membrane. Otitis media refers to disease involving the tympanic membrane and tympanic bulla. Otitis interna implies involvement of the hearing apparatus with neurological signs and deafness usually present.

CLINICALLY RELEVANT REVIEW OF EAR ANATOMY RELATIVE TO OTITIS MEDIA

Tympanic Membrane

The tympanic membrane slants downward an inward obliquely to form an angle. This increases the surface area of the TM. This is a semitransparent epithelial structure. It is divided into two parts. The smaller dorsal opaque portion is pink, loosely attached and contains blood vessels it called the pars flaccida. This part of the TM is important in healing of damaged TM. The larger gray semitransparent ventral part is called the pars tensa. In cats the pars flaccida is maybe difficult to visualize due its small size. The TM is composed of four layers. It functions as a barrier and this function is disrupted when it is inflamed. This allows for transmembrane migration of bacteria. When the TM is perforated it heals by proliferation of the fibrous layer. This appears as an opaque membrane in contrast to a normal translucent structure. The most depressed part of the TM is called the umbo. It is important not to damage this area when performing myringotomy because this area contains germinal epithelium where healing of TM begins.

Middle Ear (Tympanic Cavity TC) of the Cat

This is an air-filled chamber lined by pseudostratified ciliated columnar epithelium containing goblet cells. The mucous membrane is continuous with that of pharynx through the Eustachian tube. Mucous producing cells are found in the TC but not horizontal canal. If mucous is seen in the TC, it had to originate from the TC indicating damage to the tympanic membrane. The tympanic cavity is divided by a bony shelf.

CAUSES OF RECURRENT OTITIS

There are many classification schemes for determining the cause of otitis or recurrent otitis. For recurrent otitis the author uses the following simplified scheme. There are basically four major reasons for recurrent otitis.

- **Obstruction**: In cats, the most common obstructions of the ear canal include but are not limited to: neoplastic tumors, benign polyps, obstruction due to Otodectes debris, and caseation of exudate from the bulla, inflammatory obstruction and foreign bodies.
- **Undiagnosed Infection**: This includes primarily bacteria and yeast.
- **Undiagnosed or Inadequately Managed Underlying Skin Disease**: The most common cause in cats include but are not limited to hypersensitivity disorders (flea, parasites, environmental allergies, contact reactions, food), immune mediate disease (pemphigus), over-cleaning disease of owners.
- **Otitis media**: More than one cause can be involved in a particular patient.
COMMON CAUSES OF OTITIS MEDIA (OM)

Otitis media is generically defined as inflammation of the middle ear. It may or may not be infectious. In cats it can be a sequela to:
- Upper respiratory infection and/or chronic sinusitis
- Obstructive lesions, e.g. polyp
- Primary disease via retrograde ascension through the Eustachian tube into the TB
- Complication of ear mite infestation
- Anatomical abnormalities of the soft palate
- History of para-aural abscess

Otitis media in cats has been classified as being primary or secondary. Primary otitis media results from infectious agents ascending into the bulla from the Eustachian tube. It has been hypothesized that infection with feline herpes, feline calcivirus may reduce the ability of the Eustachian tube to protect the middle ear from infection. Secondary otitis media is associated with inflammatory polyps or otitis externa which results in damage to the tympanic membrane.

CLINICAL SIGNS OF OTITIS MEDIA

- OM can be unilateral or bilateral
- Many but not all cats with OM will have signs of concurrent OE.
- Suspect OM in any cat presenting with severe purulent OE.
- Cats with OM but no signs of OE may present for head shaking, pawing at the ear, pain when touched, pain when mouth is opened, and changes in eating habits or appetite, depression, suspect hearing loss.
- OI resulting from OM is a major differential diagnosis for any cat presenting with neurological signs: head tilt, circling, falling, generalized incoordination, difficulty rising or ambulating, nystagmus, facial nerve paralysis or Horner's syndrome.
- Nystagmus may be spontaneous, horizontal or rotary with the fast phase away from the affected side and head tilt.
- In cats with vestibular signs associated with concurrent OE/OM it is reasonable to assume the OI is the result of this infection.
- Cats presenting with OI in the absence of OE/OM are problematic as the causes of the neurological signs may or may not be due to OM. These cats require aggressive diagnostics to determine if the vestibular signs are peripheral or central. The three major differential diagnoses for cats with peripheral vestibular disease are OM/OI, obstructive mass, and idiopathic feline vestibular syndrome.
- Otoscopic examination typically reveals exudate and inflammation. Manipulation of the ear is painful and the tell-tale “squishing” sound indicative of fluid/exudate in the canal is common.
- If the TM is visualized it may be bulging, discolored, and/or a rupture may be seen.

POINT OF CARE DIAGNOSTICS-SPENDING MONEY WISELY

Cytology
- Cytology of the ear exudate is considered a core diagnostic test. It is not necessary to heat fix slides and, in fact, this is discouraged as it distorts cellular architecture.
- Malassezia are commonly found in cats and their significance depends upon the presence or absence of clinical signs. One study found that Malassezia were found in 23% of normal cats and in 64% of cats with OE.
- Bacterial overgrowth is uncommon; if bacteria are noted a culture is indicated.
- Presence of leukocytes indicates supportive otitis, is highly suspicious of OM and aggressive antimicrobial therapy is indicated based upon a culture and susceptibility.
**Microbial culture and susceptibility (C/S)**

- This should be done by reference laboratories that will speciate isolates and perform culture and susceptibility testing on multiple strains of a pathogen. This is particularly important in cases of chronic otitis.
- Spurious results will occur if the owner has treated or medicated the ear; if this has occurred wait 48 to 72 hours post last treatment to do C/S
- If the plan is to perform a C/S of the contents of the TB, do not waste money on a C/S from the vertical ear canal.

**Comments on diagnostic imaging**

For a detailed discussion of diagnostic imaging of the ear of the cat, see Forrest “Diagnostic Imaging of the Ear”. This is an outstanding review of the topic and is richly illustrated.

- **Radiographs**: useful screening technique for simple otitis externa/media. Use general anesthesia and include open mouth anteroposterior, lateral oblique and ventrodorsal views of tympanic bulla; radiographic changes involving the bulla are always associated with disease, but the absence does not rule it out
- **CT**: useful technique for examining bony structures, middle and inner ear, and treatment planning, no superimposition of structure as in radiographs
- **MRI**: useful technique for middle and inner ear and evaluation of soft tissue structures
- **Indications for Imaging of ears**:
  - Ear exudate, suspect otitis: radiology bulla series, CT if otitis not clearly identified on radiographs or cat is unresponsive to appropriate therapy
  - Respiratory strider, sneezing, nasal discharge in young cats suspect nasopharyngeal polyp: Radiography of bulla and lateral larynx radiograph, CT more sensitive for polyp evaluation also able to evaluate nasal cavity, MRI most sensitive with excellent contrast resolution
  - Head tilt: central vs vestibular disease: CT or MRI is the best choice, evaluate the brain and the bullae, can do radiography of bulla, can start with screening radiographs and if bullae are normal proceed with MRI to evaluate for brain lesion
- **Inflammatory disease will cause thickening of the tympanic bullae which is usually smoothly marginated; fluid accumulation in the bulla and inner ear can be seen alone or with bony changes**
  - Evaluate the bulla contour, it should be an air filled and thin walled, rounded bony structure
  - Evaluate the bulla for bony proliferation or osteolytic lesions
  - Evaluate the bulla for presence of fluid/soft tissue opacity; radiography cannot differentiate between soft tissue and fluid; with CT contrast enhancement or an air-filled interface can differentiate between soft tissue or fluid, respectively

**COMMENTS ON PERFORMING A MYRINGOTOMY AND MIDDLE EAR IRRIGATION**

- To date there is no absolute standard of care for performing a myringotomy and middle ear irrigation. Each case needs to be evaluated individually.
- Potential complications of myringotomy and middle ear irrigation include, but are not limited to: complications from anesthesia, development of neurological signs, head shaking, pain, and failure of the tympanic membrane to heal.
- In general, if clinical signs are severe enough to warrant diagnostic imaging and otitis media is suspected or anticipated, it is prudent to plan for this procedure.
- A myringotomy can be performed using a hand-held operating otoscope or via video-otoscopy. With the latter the TM can be visualized otherwise the procedure is performed blindly.
- The purpose of the myringotomy is to collect a culture specimen from the middle ear and provide drainage of the middle ear with or without lavage. The most common procedure is to use a Tom-cat catheter with the end cut at a 45° to pierce the TM. The incision is made in the ventral TM between 5 and 7 o’clock. Small volume of sterile fluid is flushed and then immediately aspirated from the bulla. Note: The volume of the TB of the cat is approximately 0.5 ml. If the disease is bilateral, culture both bullae. Be aware that if the TM is already ruptured the culture findings will need to be interpreted in light of the fact there may be contamination from the external ear canal.
- The bulla is flushed with warm sterile saline until the effluent runs clean.
• Otitis media should be treated with a topical and systemic antibiotic based upon culture and susceptibility and cytology (i.e. yeast).

COMMENTS ON SELECTED CONDITIONS

Clinically relevant information from retrospective imaging and necropsy studies

There is increasing evidence from retrospective imaging studies and necropsy studies that OM/OI is more common in cats than the current literature would indicate.

Gross and histological examination of 100 ears (50 heads) found gross lesions in 14/100 (14%) of ears. These findings included fluid, frank pus, hemorrhage and fibrous thickening of the auricular mucoperiosteum. In the same study 48/100 ears had histological evidence of on-going or previous middle ear disease. Of these 34 of 48 of these cats lacked gross evidence of disease. In 11 ears there was evidence of otitis interna. This study investigators did not have access to medical records.

In a second, medical records and necropsy findings of 59 cats with non-neoplastic disease were reviewed. Twenty-six had bilateral disease and 33 had unilateral disease. Fluid or caseous material identified in the tympanic bulla in all affected ears. Fifty three of 59 cats (90%) did not have any clinical signs of middle ear disease. Of the remaining six cats, five had signs of unilateral peripheral vestibular disease and one had Horner's syndrome. "Twenty cats had been euthanatized because of a poor prognosis related to a primary disease process unrelated to middle ear disease, 14 had been euthanatized for unknown reasons, 11 had been euthanatized because of a failure to respond to treatment for a primary disease process unrelated to middle ear disease, 6 had been euthanatized because of financial reasons, and 8 had died."

In a retrospective study of 77 cats with vestibular disease that underwent MRI scans, 37 cats were suspected of having peripheral vestibular disease (unilateral n=32, bilateral n=5). Disease was acute and progressive over a three-week period. In 15/37 cats had obtundated mental status and 12/15 had otitis media/interna. Visible lesions on MRI were noted in 21/37 cats with peripheral vestibular disease. Lesions were noted in the middle ear in 18 cats and 16/18 cats were suspected of having otitis media/interna. This confirmed by C/S in 10/16 cats. Cats with otitis media/interna were treated with oral antibiotics for a minimum of 8 weeks. Clinical signs resolved with medical treatment and no cats required surgery. Of note, five cats had subtle signs of intracranial extension of the inflammatory process into the caudal brainstem.

When findings from 310 cats that had CT imaging of the head were reviewed 101/310 (32.6%) had evidence of middle ear disease on CT. In this group, 41/101 cats did not have signs of external or middle ear disease but DID have nonspecific clinical signs, physical examination findings or CT findings that included fluid or soft tissue attenuating material in the external ear canal. Clinical signs included circling, falling to one side, ataxia, pain on opening of the mouth, head tilt, and discomfort when chewing. In this group bilateral disease was more common than unilateral disease. In 34/101 cats (34%) did not have a complaint of ear disease, clinical signs or physically findings of ear disease. This suggests the findings were subclinical. Of note, 27 of 34 cats (79%) of cats had concurrent nasal disease.

In a retrospective study that compared CT scans from cats with (n=46) and without (n =18) sinonasal disease reviewed, bulla effusion was present in 13/46 cats with nasal disease. Unilateral bulla effusion was found in only 1/18 cats in the control group. Clinical or historical evidence of chronic OM was not reported in these cats but interestingly, 8/46 cats with sinonasal disease had a clinical history of possible ear disease: ear pruritus, ataxia, deafness, head shaking, aural discharge, or third eyelid prolapse and 4/8 cats with clinical signs of bulla effusion. This suggests the bulla effusion was acute. The most common neoplasm associated with bulla effusion was nasopharyngeal lymphoma. Of note bulla effusion was not noted in cats with nasal carcinoma.

OTITIS MEDIA IN CATS WITH SOFT PALATE ABNORMALITIES

Five cats are described in the literature with either unilateral or bilateral otitis media/otitis interna that were diagnosed with soft palate abnormalities. These abnormalities are believed to be the cause of the otitis media/interna. Four of five cats were adults (>4 years of age when it occurred) and one was a 7-month-old kitten. In four of five cats, the soft palate abnormalities were not recognized during intubation prior to specialty examination. It was believed the abnormalities were congenital suggesting the development of otitis media was a predisposing factor and not a direct cause.
INFECTIOUS CAUSES OF OTITIS MEDIA

It is clear from a review of the literature that OM in cats can be caused by a wide range of pathogens. Infectious agents unique to cats may ascend the Eustachian tube that connects the pharynx to the middle ear. The bulla can become colonized with normal flora. Infections may develop because of decreased ciliated epithelium propulsion and/or Eustachian tube dysfunction. *Mycoplasma* spp have been isolated from the bulla of cats with OM. Mycoplasma was also found in a young cat with meningo-encephalomyelitis. Purulent exudate was found in the bulla of this cat. Increasingly there are anecdotal reports and case reports suggesting that *Streptococcus* spp are emerging pathogens of importance in cats with OM. Corynebacterium and *Aspergillus* can also be isolated from cats with OM.

SURGICAL ASPECTS OF TREATMENT

In an experimental study found that ventral bulla osteotomy did not significantly change the tympanic bulla conformation. In addition, complete regeneration of the bulla did not occur before 16 weeks post operatively.

REFERENCES

1. Kennis RA Feline Otitis: Diagnosis and Treatment Vet Clin Small Animal 2013; 51-56
3. Gotthelf LN. Diagnosis and treatment of otitis media in dogs and cats VCNA 2004; 34:469-487
7. Sula MM, Njaa BL, Payton ME Histological characterization of the cat middle ear: in sickness and in health Vet Path 2014; 51:951-967
17. Goodale EC, Outerbridge CA, White SD *Aspergillus* otitis in small animals-a retrospective study of 17 cases Vet Dermatol 2016; 27:3-62
Most Cats Would Agree, Bald Is Not Beautiful
Karen A Moriello DVM, Diplomate American College of Veterinary Dermatology

The Feline Hair Follicle
Cats have compound hair follicles. In general, there is a cluster of two to five primary hairs surrounded by groups of smaller secondary hairs. One primary hair is the largest (central) surrounded by groups of small primary hairs (lateral primary hairs). Each primary hair has a sebaceous gland, arrector pili muscle, sweat gland. Secondary hairs may only have a sebaceous gland. Five to 20 secondary hairs may accompany each primary hair. The two most common specialized hairs are sinus hairs (whiskers) and tylotrich hairs.

Skin Biopsy: When, Where, and How

When-Timing in the Diagnostic Approach: In general, the longer a disease process is present the more likely, that the classic findings associated with the underlying disease will be obscured by inflammatory changes. Systemic drugs can and do affect cellular infiltrates in skin biopsy specimens. No studies have been conducted on appropriate wash out periods; use the same washout periods for skin biopsy that are used for intradermal skin testing. If topical therapy is being used, allow for at least a one-week washout period.

Where-Lesion Selection: Skin biopsy specimens from cats with alopecia should include as many representative samples as possible, including a “normal” site. The latter will allow for comparison of the cellular filtrate and hair follicle stages between normal and abnormal. Avoid areas of with significant trauma; look for primary lesions. The skin should not be prepped in any way prior to the sampling.

How-Getting a Better Skin Biopsy: Skin biopsy specimens should be obtained under heavy sedation and concurrent local anaesthesia. The skin should not be prepped to avoid losing important surface finding and or prevent introduction of artefacts. Skin biopsy specimens shrink 50% when placed in formalin so take at least a 6 mm, or better 8 mm skin biopsy sample. The use of a new skin biopsy punch is recommended; this will avoid the introduction of shear. Harvest the biopsy with care to avoid introduction of crush artefact. The skin of cats is thin and should be placed between foam in a biopsy cartridge or on a wooden tongue depressor, (this can cause dehydration and shrinkage). It is very important to allow the specimen to fix for at least 24 hours before being sectioned by the pathologist. If it is necessary to submit all of the specimens in one containing making it difficult to label the specimens, biopsy cartridges are strongly recommended because important information can be written on the flattened front. If these are not available, a small part of the biopsy can be marked with a black sharpie and then a notation made for the pathologist. It is very important to take a large enough specimen so that many hair follicles are present and to leave the hairs long enough that the pathologist can see them grossly. If the hairs cannot be seen at the time of bisection, the specimen may not be oriented properly resulting in a "swiss cheese" cut versus a cut parallel to the hair follicles.

Causes of Alopecia in Cats

This seminar will focus on the work up of cats with inflammatory and non-inflammatory alopecia and discuss common and uncommon causes in each category.

The Otherwise Healthy Cat

The most important thing to determine when presented with a ‘bald’ cat is whether or not the cat is pruritic. If the cat is otherwise healthy, the first step in the work up of a “bald cat’ is to look for any evidence of self-trauma. As most veterinarians know, cats can be very secretive in their grooming habits and owners may often miss clinical itch behaviours.

Helpful First Step Diagnostic Tests:
Ear swabs for mites and for cytology
Skin cytology of affected area (clear acetate tape and/or glass slide)
Skin scraping with skin scraping spatula AND hair trichogram-examine for mites, hair shaft abnormalities and for any signs of dermatophytosis
Response to treatment trial with fluralaner to rule out ectoparasites

NOT helpful at first visit in otherwise healthy cat
Food trial-food allergy is an uncommon cause of pruritus in cats and is not indicated at first presentation
Routine laboratory work: serum chemistry, urinalysis, complete blood count (except blood smear looking for eosinophilia
Skin biopsy
Serum allergy testing or intradermal testing

Cat with Dramatic Clinical Signs and/or Signs of Systemic Illness
Ear swabs for mites and for cytology
Skin cytology of affected area (clear acetate tape and/or glass slide)
Skin scraping with skin scraping spatula AND hair trichogram-examine for mites, hair shaft abnormalities and for any signs of dermatophytosis
Skin biopsy to rule out neoplasia or other infectious diseases
Routine laboratory work: serum chemistry panel, urinalysis, complete blood count, T4

NOTES:
Taking the “Ahh” Out of the Diagnosis and Treatment of Dermatophytosis
Karen A. Moriello DVM, Diplomate American College of Veterinary Dermatology

Feline dermatophytosis is a superficial fungal skin disease of cats. The disease is not life threatening and is treatable and curable. The disease will also spontaneously resolve without any treatment in otherwise healthy animals. Severe disease is the result of severe physiological stress and the disease will self-cure if and when the underlying disease resolves. This disease has recently been reviewed in detail.¹

The primary pathogen of cats is Microsporum canis. This organism is not part of the normal fungal biome of cats and therefore isolation indicated true disease or fomite carriage. Less frequently Trichophyton sp and M. gypseum can cause disease in cats. Trichophyton sp infections in cats tend to occur in the fall and winter months and are most common in cats or kittens that are housed outdoors, are ‘hunters’, and/or are have exposure to large animals. Care must be taken when these two pathogens are isolated from a toothbrush culture as fomite carriage is common, especially with M. gypseum which is a soil based organisms.

Disease prevalence is hard to determine from published studies because of different methodologies. Many of the studies were published before awareness of the fact that M. canis is not part of the normal flora of cat hair and the issue of fomite carriage. Electronic medical record review found that feline dermatophytosis was not a common disease, occurrence 0-<4% of skin cases and was not even one of the top 10 skin diseases of cats. A review of risk factors found that the most at risk populations were kittens, stray cats, cats from hoarding situations, and outdoor cats. The use of immunosuppressive drugs was not a risk factor nor was positive retrovirus status alone in an otherwise healthy cat. Persian cats are at increased risk for subcutaneous nodular lesions.

One of the most important ‘new’ findings about M. canis dermatophytosis is that the primary mode of transmission is via direct contact with another infected animal. The successful establishment of an infection requires all of the following to occur: exposure to an adequate quantity of infective material, evasion of the host defense mechanisms, e.g. grooming and antimicrobial properties of the skin, skin micro-trauma, and moisture. It is RARE for people to contract M. canis infections from the environment (1 published case report) in the absence of direct animal contact. The major reason for cleaning is to remove spores from the environment that could otherwise cause fomite contamination and interfere with monitoring of response to treatment.

With regard to clinical signs, it is important to remember that the statement “It’s ringworm until proven otherwise is untrue”. The most common cause of skin lesions in cats are due to parasites. With respect to how dermatophyte lesions appear, individual lesion presentation reflects disease pathogenesis. Early infected hairs may appear normal unless examined by a Wood’s lamp tool. As the disease progresses hair loss, scaling, crusting, hyperpigmentation and pruritus may all develop. From a more practical perspective, given that the presentation of the disease reflects the global health of the cat infections are best considered as “simple”, “complicated” or “culture positive-lesion free”.

Simple infection: This group consisted of otherwise healthy cats or kittens with confirmed infections. Lesions were obvious but limited in extent. Cats responded well to a wide variety of treatment protocols and/or the disease rapidly cured without treatment.
Complicated infection: This group consisted of cats with wide spread lesions, inflammatory lesions, long-haired/matted hair coats, other illnesses (most notably upper respiratory infections), a history of prior treatment, surrender for “resistant dermatophytosis”, and/or are semi-feral or feral cats. These cats required more prolonged treatment and repeat courses of treatment to achieve mycological cure. These cats did not cure until their overall health was ‘normal’.
Lesions Free but Culture Positive Group: This group of cats consisted of cats mechanically carrying spores on their hair coat (i.e. “dust mops”) or cats with very early lesions that were not easily seen but mature enough to be shedding arthrospores. Fungal culture results coupled with a re-examination under both white light and a Wood’s lamp are helpful to differentiate fomite carriers from cats with early lesions; however, fomite carriers were most often identified by a rapid change in culture status from positive to negative with topical therapy alone provided they were in a clean environment.

There are only two tests that truly confirm active infection: direct examination of hairs and skin biopsy of a lesion. The Wood’s lamp and the dermoscope are tools, just like a microscope, that are used to find suspect hairs for direct examination or fungal culture. Skin scrapings and hair pluckings of lesions are diagnostic sampling techniques used to find hairs for microscopic evaluation. Fungal culture merely detects spores on the hair coat. PCR detects fungal DNA on the hair coat.

Skin biopsy a diagnostic TEST that is indicated in cats with nodular lesions or with dramatic skin lesions for which point of care tests do not find a diagnosis. It is not a routinely performed test but when done the sample size obtained should be 6 to 8 mm in size. There is approximately 50% shrinkage of samples in formalin and anything smaller is going to be non-diagnostic. Do not prepare the skin surface or alter it as key findings for dermatologic diseases are found in the skin surface. Submit several samples and be sure to inform the laboratory of the differential diagnosis list, particularly that a dermatophyte is suspect.

Direct examination is a simple diagnostic TEST where hair shafts are examined for the presence or absence of infected hairs. Suspect hairs can be found using a Wood’s lamp tool (see below), dermoscope (see below) or via skin scraping (See below). Normal hairs appear thread-like and abnormal hairs appear pale, wide and filamentous. Infected hairs are easily found at low power.

A recent study found that the best diagnostic sampling technique whether a lesions was Wood’s lamp positive or not was to use mineral oil and a combination of plucking of hairs from the margins of a lesion and scraping of the lesion with a skin scraping spatula. This test detected >87% of *M. canis, M. gypseum* and *Trichophyton spp* infections in dogs and cats. Clearing agents were not helpful. Mineral oil is preferred because it also allows for the dual examination of the specimen for mites and hair shafts for dermatophytosis.

Wood’s lamp examination is a point of care TOOL that is used to find fluorescing hairs of *M. canis* for direct examination and/or culture. The best used lamp is plug in with built-in magnification with a wave length of 320 to 400nm. A recent evidence based review of diagnosis and treatment of dermatophytosis found the Wood’s lamp is an good diagnostic tool for finding infected hairs. Studies reporting low fluorescence were retrospective reference laboratory studies looking at randomly submitted samples over decades. When studies on live animals were examined 100% of experimentally infected animals had positive fluorescing hairs and in cat with spontaneous disease 91% had positive fluorescence. As treatment progressed, fluorescence declined (as expected). It is important to hold the lamp close to the skin 2-4 cm, start at the head, move slowly, and remember to look under crusts for glowing hairs. Newly infected hairs are very short. Tips and tricks are detailed in an open access reference.²

Dermoscope is a point of care TOOL for finding hairs for direct examination and/or culture. It is a hand held lighted magnifying device that can be attached to cell phone. It is used to find abnormal hairs which appear as pale and broad.

Fungal culture merely detects the presence of spores on the hair coat of a cat and needs to be interpreted in light of clinical history and clinical findings. The toothbrush culture technique is recommended. ONLY the suspect lesion should be cultured and it should be aggressive enough to see hairs in the bristles. A recent study found that samples should be inoculated directly onto the surface of a flat fungal culture plate. Hairs should not be removed from the bristles as this resulted in contamination and false negatives. Poor sampling technique can result in false negative test results. Do not OVER inoculate the surface of the plate as this may result in lack of sporulation as fungal hyphae compete for nutrients.
PCR testing is relatively new. The test advantage is that results are available within days and not weeks. Samples should be taken only from suspect lesions. Hairs and crusts should be submitted. Inadequate sample submission is a major reason for false negative test results. If used for monitoring, bathe the cat prior to testing to remove any non-viable fungal DNA.

**2019 TREATMENT RECOMMENDATIONS AND OPTIONS**

**Confinement:** Contrary to popular Dr. Google cats the reason cats are confined are to keep them physically safe and to decrease the living area that needs to be cleaned. Infection risk from a contaminated environment is not efficient and is hard to document. Confinement to an easily cleaned room shortens overall treatment time. Review of the literature on animal welfare, quality of life and socialization of kittens and cats requires that veterinarians reassess this recommendation and give clients very specific instructions. Dermatophytosis is most common in kittens during the critical socialization period and proper socialization of a new cat into a home is necessary so both remain in a permanent and loving home. Most cases of dermatophytosis are in cats that are new additions to the home. Confinement can be limited to what the owner would do normally when adding a new cat to a home, i.e. kitten proof space while they are at work etc. Frequent use of topical therapy will disinfect the hair coat and minimize the amount of infective material on the coat. Owners can socialize with kittens and cats using safe precaution. If children are in the home, care should be taken to educate them on how to safely play with the kitten under supervision and minimize direct contact. With early disease detection, removal of shed hairs via combing, consistent and frequent topical therapy, and systemic therapy confinement can be limited to a short period of time. Topical therapy is protective against contact with the spores especially if lime sulphur is used.

**Cleaning:** Contrary to popular Dr. Google facts show that cleaning and disinfection is relatively easy, and spores do not live for years. A major myth in the literature is that the spores live for years in the environment. This statement was extrapolated from a laboratory study where 3 of 6 specimens stored for 18 to 24 months sporulated on fungal culture medium. The primary reason to clean is to minimize environmental contamination that can lead to positive fungal culture or PCR results from fomite carriage in cured cats. Review of the literature revealed that confirmed reports of transmission of the disease from a contaminated environment to a person in the absence of contact are rare. Points to emphasize with clients:

- Fungal spores do not ‘live’ in the environment and do not multiply. They do not invade and grow in any of the home surfaces. Spores can only live in keratin.
- Fungal spores are like dust, not like mildew.
- Fungal spores are EASILY removed by mechanical cleaning and washing with a detergent and water.
- Culturing of the environment is not needed unless there is concern about false positive fungal culture results.
- These spores do not represent a respiratory risk. This is caused by overgrowth of different fungi living in the home due to excessive moisture.
- It is very likely that they or someone they live with has or had human dermatophytosis, the most common being “toe nail fungus.” In addition, there is no need for alarm about exposure to spores as many studies have shown people are exposed to ‘ringworm spores’ in many environments including but not limited to: their homes, other homes, gym, pools, doctor’s offices, the beach, airports, etc.
- This is a zoonotic disease but compared to other zoonotic diseases it is not life threatening or life changing.

In practice, if it can be washed it can be disinfected. Exposed laundry need only be washed twice until no visible hair is present. Evidence based studies have shown that cold water without bleach is just as effective as hot water and bleach. With regard to ‘hard surfaces’ the key steps are to tell clients to clean as ‘if company is coming’ or as ‘if they are cleaning up vomit or feces’. Specifically, mechanical removal of
debris, washing of the surface with an over the counter detergent until visibly clean, rinse the surface to remove detergent residue which can inactivate a disinfectant, remove excess water which can dilute a disinfectant, and then careful use of a disinfectant. Regarding the type of disinfectant, studies have shown that any over the counter bathroom cleaner with label claim as efficacious against Trichophyton is effective. MOST homes need daily changes of bedding, mechanical removal of debris with a Swiffer and once or twice wet cleaning. Do not use bleach as this is an irritant and human health hazard and better ready to use products are available.

**Assessment** The first and most important aspect of monitoring is looking for and attaining a clinical cure. In otherwise health cats lesions will often start resolving within one or two weeks. Lack of a clinical response is an indicator of treatment failure for some reason. Given that this is a self-curing disease, if the client is administering treatment as directed, there may be some underlying disease or physiological stress, or possibly a secondary untreated disease. Wood’s lamp tools are very helpful for monitoring response to treatment. Prior to the wide availability of fungal cultures, the most common method to monitor response to treatment was via a Wood’s lamp examination. Detailed reports of the development and resolution of infections are consistent. During the early stages of disease, the proximal part of the hair shaft showed fluorescence. As the infection progress the entire shaft develops fluorescence and with eradication of the infection, it is lost in the proximal part of the hair shaft. As the cure progresses and the hair recovers, hair shaft fluorescence proceeds up the shaft until only the tips glow. These findings have been confirmed in experimental infection studies and field studies.

The next step in monitoring is to treat until mycological cure. There is no consensus on what constitutes mycological cure, i.e. how many cultures and at what intervals. The current literature recommends two negative cultures taken at two week intervals but this is not based upon any study or data and was made in 1959. In a recent unpublished review of the treatment of >300 shelter cats, the author found that in cats with simple infections the first negative culture was predictive of mycological cure. Given the sensitivity of PCR, a single negative PCR indicated mycological cure assuming adequate sampling. If the PCR is positive a toothbrush fungal culture should be obtained to determine if detected fungal DNA is viable or not.

**Topical Therapy:** Topical therapy is a non-optional part of the direct treatment of the infection. It is the part of therapy that disinfects the hair coat, directly protects people and other susceptible animals from disease transmission, minimizes the risk of satellite lesion development, and minimizes environmental decontamination. Two studies found that the use of topical therapy prevented environmental contamination within one week of starting treatment. Topical therapy recommendations include the following:

- Comb the hair coat to remove any easily shed hairs; use of disposable plastic comb is ideal.
- Apply topical therapy twice a week
  - Whole body rinses include lime sulfur (1:16) or enilconazole (1:100); do not rinse off. Whole body rinses are recommended in homes where there are children or where there is a high risk of contagion to other animals or people.
  - The best shampoo alternative is a 2% chlorhexidine/2% miconazole shampoo; shampoo therapy does not have any residual effect.
- Use concurrent focal topical therapy for hard to treat areas
  - Apply 2% vaginal miconazole to lesions on the face once daily along with twice weekly topical therapy.
  - Apply an ear otic solution containing miconazole/chlorhexidine or ketoconazole/chlorhexidine to disinfect hairs in and around the ears.
- For cats that cannot be wetted, climbazole/miconazole or chlorhexidine/miconazole leave on mousse can be used.
Systemic Therapy: The drug of choice for the treatment of feline dermatophytosis is NON COMPOUNDED itraconazole. It is available as a liquid formulation. This drug is safe and in a review of 11 studies, excluding the study conducted in the United States for licensing, there were no published reports of cats being treated for dermatophytosis that developed liver toxicity and died. The recommended treatment dose is 5 mg/kg orally on a week on/week off protocol for an initial six weeks or longer until mycological cure. Most cats will cure with six weeks of treatment. The reason the drug can be used on a pulse dose protocol is that it accumulates in the keratin in antifungal levels for prolonged periods of time.

“Itch” is one of the most commonly encountered dermatological problems in feline dermatology. The most helpful prospective study published was by “Clinical characteristics and causes of pruritus in cats; a multicentre study on feline hypersensitivity associated dermatoses” (Hobi, et al. 2011, Veterinary Dermatology 2011). In that study, 588 cats with chronic itch defined as itching for more than 2 months all underwent a systematic work up. The findings from this study provide the best evidence based information available about the causes of itch in cats.

The information in Hobi’s study is directly applicable to feline practice. The study found that the most common cause of chronic itch in cats was parasites. This included fleas, *Demodex cati*, *Demodex gatoi*, *Otodectes*, fur mites (many species), and ticks. Interestingly the study found that adverse reactions to food, ‘i.e. food allergy’ were the least common cause of chronic itching in cats. These findings are particularly important because adverse food reactions were confirmed by provocative challenge and not just the ‘owner impression’. This study also found that the food trial step was the most problematic response to treatment trial for owners and the part most likely to be subject to sabotage by cats. Another important finding was that about one out of four cats has a non-hypersensitivity disease causing the itch. This included but was not limited to bacterial and yeast skin infections, autoimmune skin diseases, metabolic skin diseases, in cats that went out doors dermatophytosis, etc. In addition, many cats had more than one cause of their chronic itch. For example, many cats had parasite control responsive itch complicated by secondary skin infections. Finally, about one out of five cats had a non-parasite responsive, non-food responsive itch or “feline allergic skin disease, ‘feline atopic dermatitis’.

The owners of “crazy itchy cats” are often frustrated and typically report having “done everything but nothing seems to work”. What is most helpful is to explain that many of these diagnostic tests and treatment trials have spanned a long period of time and a compact dedicated work up is needed.

Day 0
Step 1: Use a Visual Analog scale to assess the severity of the cat’s itch. This uses descriptors instead of subjective numbers. There are many on line just “google” Pruritus and visual analog scale for cats. Use the same scale to record that cat’s response to treatment at each visit.

Step 2: Perform all of the following core diagnostic tests at intake: ear swabs for routine cytology (do not heat fix glass slides or skip any of the dips), ear swabs in mineral oil for adults and kittens, hair plucking and skin scrapings in mineral oil using a skin scraping spatula (cost effective method to look for hair shaft abnormalities associated with dermatophytes and for mites), skin cytology using clear acetate tape and glass slides, nail bed cytology collecting specimens using a skin scraping spatula. Do not use a scalpel blade for collecting samples. When processing clear acetate tape samples, skip the fixative and stain routinely. Be sure to let the tape strip dry thoroughly before mounting it over a drop of immersion oil. DO NOT stick the tape to a glass slide and then stain it. This produces poor staining. Wood’s lamp tools and fungal culture are only indicated in cats that are considered high risk for dermatophytosis (outdoor cats, kittens). Contrary to popular belief, dermatophytosis is not common and the statement “it is ringworm until proven otherwise” is false.

Step 3: Perform aggressive parasite and infection control. Owners often are reluctant to “do flea control” because they do not see fleas. It is hugely helpful to use the term “parasite control” and inform them that parasite control includes not just fleas but mites that are often difficult to find or treat. The speaker prefers to use spot on fluralaner (Bravecto) because it is an isoxazoline which is a class of drugs with a wide spectrum of activity against parasites, including demodicosis. The three month duration of action ensures there is parasite treatment and control for the duration of the compact work up trial. Use of this product
also removes the necessity of doing lime sulphur rinses to rule out demodicosis. In almost all cases of ‘crazy itchy cats’ there is microbial overgrowth-bacterial and/or yeast. We now recognize that bacterial pyoderma and Malassezia overgrowth are common in cats. Cats can and will tolerate topical therapy and the drug combination recommended includes chlorhexidine/miconazole in a spray or mousse formation. Systemic antibiotics should be administered only based upon culture and susceptibility testing. If Malassezia is suspected, oral liquid itraconazole 5 mg/kg can be administered on a week on/week off basis. This treatment trial should be continued for at least 4 weeks.

Recheck 1: At or around Day 30

Step 4: If the cat is not itchy, continue with year round flea preventative and watch for a relapse. If the cat IS STILL ITCHY, the only causes left are: food allergy (rare), environmental allergies, or undiagnosed hypersensitivity dermatitis. The next step is to proceed with a food trial. Although adverse food reactions were rare in Hobi’s study (12% of 588 cats), in a private practice setting this would be the next step prior to referral for an allergen specific work up. Hobi did report in another study that although cats with adverse food reactions were indistinguishable from cats with environmental allergies, one finding was that cats with adverse food reactions had a trend toward more gastro-intestinal signs (soft stools, frequent stools, etc.). Be sure to use a complete and balanced commercial diet: hydrolysed protein diet or limited ingredient diet. Over the counter ‘hypoallergenic diets’ are not suitable as studies have found cross contamination in these diets.

Contrary to popular belief, the food trial does not need to be 12 weeks in length. Most cats with food allergies can be diagnosed in 4-6 weeks. It is important to ask if a food trial is possible in the home and determine if the cat will eat the diet. The details of how to perform and interpret a food trial in cats are discussed in detail in the seminar.

Recheck 2: At or around 4-6 weeks after starting the food trial

If the cat has responded to the food trial, relapsed with a provocative challenge then an adverse reaction to food has been documented. It goes without saying, but worth mentioning the target clinical sign is itch and this should have resolved with the diet trial and relapsed upon challenge. If this is the finding then the options are to pursue finding the cause or plan to feed the cat the complete and balanced diet used for the food trial.

If the cat has not responded to the food trial, then the diagnosis is allergic dermatitis. The following points are important to stress to clients:

1. Parasite control needs to continue year round as any infestation can cause a flare of secondary infections and the allergies.
2. Grooming and topical therapy to minimize pruritic secondary infections need to be continued.
3. Allergy testing is indicated only if the client wishes to pursue allergen specific immunotherapy. However some form of humane relief of pruritus is needed.
4. Humane relief of pruritus includes:
   a. Induction of itch remission using methylprednisolone 1.4 mg/kg OR triamcinolone 0.18 mg/kg orally once daily until itching is in remission, then dose taper by administering every 48 hours, if there is still a good response every 48 hours, further decrease the dose by 25% every week until the lowest possible dose is administered.
   b. Induction of itch remission using feline liquid cyclosporine 7 mg/kg once daily for 30 days and then dose taper to q 48 hours for another 30 days, afterwards attempt to administer twice a week
   c. Off label use of oclacitinib. This would not be a first choice drug but one for cats that cannot receive steroids or tolerate cyclosporine.
   d. Off label use of maropitant 2.2 mg/kg orally once daily. This was found to be helpful in a small group of cats during a 30 days study.
With increasing links to disease, new therapeutics, and rising interest from the public, the microbiome is becoming increasingly more important to the practice of medicine. This session will focus on the fundamentals, drug interactions, fecal microbiota transfer (FMT), diagnostic testing, prebiotics, and probiotics.

What is it? The microbiome consists of all the microscopic organisms present in a particular environment, which includes bacteria, archaea, fungi, and viruses/phages. Because the tools we have to assess the fungome and virome are still being developed, most people mean the bacteriome when referring to the microbiome. The different environmental niches that can contain a microbiome can include everything from soil and water to the skin and intestinal tract. The focus of this talk will be on body-site associated microbiomes, with an emphasis on the gut microbiome. The microbiome is an essential part of every individual from every species and is well known to be especially important for hindgut and foregut fermenters. Imbalances in normal flora microbiome is called dysbiosis. Furthermore, there is actually a lot of molecular cross-talk between microbes and with their host. The microbiome is known to stimulate the development of the immune system early in life, aid in the digestion of food, and can help prevent pathogen colonization. It can even affect the circadian rhythm through bile acid signaling pathways. More negatively, dysbiosis has been found to play a role in the development of diabetes mellitus, inflammatory bowel disease (IBD), neoplasia, hepatopathy, obesity, and many other chronic inflammatory conditions. The microbiome can also rapidly respond to changes in its environmental factors – this includes diet modifications, shampoos, antibiotics, and even changes in host physiology.

General Composition: Surprisingly, there is no single standard microbiome that all healthy individuals of the same species share. Most mammals generally have a microbiome dominated by two groups. Phylum Firmicutes are generally gram-positive cocci, many of which can produce endospores (examples: Bacillus, Clostridium). Firmicutes are more likely to be transmitted from mother to offspring and are generally considered “good” bacteria because of their association with the increased production of butyrate in the presence of fiber. Phylum Bacteroidetes are generally gram-negative rod-shaped bacteria that do not make endospores (examples: Bacteroides, Porphyromonas). Bacteroidetes are less likely to be transmitted from mother to offspring and are generally considered “bad” due to their increased presence on a high fat diet or in samples from obese individuals. This is a gross generalization and each phylum contains thousands of different bacterial species to which these statements may not be entirely accurate.

Drug Interactions: There are many different drugs that can affect the microbiome. The most obvious one is antibiotics, which can kill a pathogen but also obliterate a huge number of normal flora. The changes in the microbiome due to antibiotics are sometimes drastic and long-lasting, but it is not the only drug category that can modify and be modified by the microbiome. Ever wonder why some oral drugs works great in on patient but not in another? It may be due to that individual’s microbiome. It is known that the gut microbiome can alter absorption and metabolism of a variety of pharmaceutical compounds [1]. The gut microbiome is also very responsive to dietary changes. Meaning that prescription diets, probiotics, home-cooked diets, and treats really can have dramatic effects on digestion and metabolism.

Transplantation: FMT is where a fecal sample from a pre-screened healthy donor is collected and mixed into a saline solution, filtered to remove large debris, and placed in a patient via scope, jejunostomy tube, or enema. Administration orally or through an esophageal tube is generally not recommended since gastric fluids will kill most bacteria. FMT has seen a lot of recent successes treating human patients with chronic C. difficile infections. In veterinary medicine FMT has successfully been used in disease states associated with dysbiosis, including canine parvovirus [2] and inflammatory bowel disease [3]. However, while this technique can be used with great success in some cases, it still merits some caution. There have also been a few failures, such as when a human patient died after receiving a transplant that unknowingly contained a multi-drug resistant strain of E. coli.
Can you test for dysbiosis? While there is no singular “normal” or “healthy” microbiome, there are some changes that can indicate a problem. There is currently a fecal PCR assay available from Texas A&M University (developed by Dr. Jan Suchodolski) that assesses if the balance of bacteria in the canine intestinal tract is abnormal. It does this by measuring the abundance of eight bacterial groups and summarizes them into one number called the canine dysbiosis index [4]. If the submitted sample has sufficient changes in the eight bacterial groups, it is highly predictive of intestinal dysbiosis. While the test was developed to help identify patients with IBD, it is not explicitly diagnostic of IBD since there are many other conditions that can cause intestinal dysbiosis.

Prebiotics: Often used in conjunction with probiotics, prebiotics refers to dietary supplements (typically fiber) that aim to promote the growth of “good” bacteria in the gut. Commensal bacteria in the colon of non-fermenters break down the fiber into short-chain fatty acids such as butyrate (typically associated with Phylum Firmicutes) as well as acetate and propionate (typically associated with Phylum Bacteroidetes).

Probiotics: Probiotics are live microorganisms that confer a health benefit to the host. Naturally occurring bacterial strains cannot be patented and sold, but laboratory modified strains can. Most probiotic species have been identified from fermented human foods, such as yogurt. None of the currently known and commonly used strains are derived from canine or feline sources. Even the new anti-anxiety probiotic strain, *Bifidobacterium longum* (BL999), is originally a human gastrointestinal isolate. The purported mechanism is that BL999 leads to beneficial signaling in the gut-brain axis. The exact mechanism has so far remained elusive and is still under investigation. Some studies with similar probiotics have shown that there is no longer an effect on behavior when the vagus nerve is cut, which indicates some form of host-microbe interaction drives the effect. In addition, because BL999 is known for its ability to help human infants with the digestion of milk oligosaccharides, it may work synergistically with some of the anti-anxiety diets that have added milk proteins.

References:
The Human Microbiome Project as well as citizen science platforms, like the American Gut Project, are helping promote awareness of the microbiome and its influences on health and disease for humans. Because of this, just like with DNA ancestry reports, people are now interested in their pets’ microbiomes. This session will help you understand the limitations as well as the future promises of the emerging research into the microbiome. Furthermore, new methods to modify the microbiome are currently being developed besides drugs, diet, prebiotics, and standard probiotics discussed previously. However, microbiome studies are still in the research phase and not yet ready to be used in a clinical capacity.

The Job: In a typical Labrador retriever (30kg/66lbs), there are approximately 13 trillion canine cells and about 18 trillion bacterial cells. That is a lot of microscopic friends! Even a small (1g) stool sample from this patient will contain about 100 billion microbes, each of which has its own genome. If we estimate that each microbe has a genome size of 5 million bases, that is about 100,000 terabytes worth of data. Since an average new laptop has a 1 terabyte hard drive, that’s a truckload of information for just one small sample. Even with modern sequencing techniques and using a supercomputer, it would take a tremendous amount of time to process and analyze all of that data. Furthermore, we want to examine samples from several to many patients, not just one.

Who is there? In order to reduce the enormous job of sequencing microbial populations, a universal marker that is smaller and more manageable would be helpful. It turns out that all bacteria have fairly similar ribosomes for translating DNA into proteins. Since ribosomes are critical to cellular functions, the DNA sequence that encodes them is always present. Additionally, the 16S subunit of the ribosome has changed enough during evolution to be good for distinguishing species from each other. The V4 region (~255 base pairs) is the most commonly used for this purpose. Thus, this short DNA sequence has become a universal marker that can be linked to bacterial identification. This method is also independent of our ability to culture the bacteria in the laboratory, which means there has been new normal bacteria that have been discovered that otherwise die in the presence of oxygen. It is also important to note that just because this allows us to know who is there, it does not tell us what they are doing.

Diversity: Because the microbiome is a whole ecosystem of bacteria, we use ecological terms and calculations to discuss what is happening in sample populations. Diversity measurements are one of the key ways of assessing the microbiome. Alpha diversity refers to the number of different species of bacteria present within a single sample. For example, it has been found that dogs with IBD have lower alpha diversity (less species of bacteria) than normal healthy dogs [5]. Although, caution should be taken in just assuming more diversity is better. A desert has less diversity than a rain forest, but it is not less “healthy”. Beta diversity refers to the differences in bacteria present when samples are compared to each other. For example, skin samples from cats with skin allergies overall had the same alpha diversity (same number of bacterial species) as otherwise healthy cats, but when we look at the beta diversity the cats with allergic skin conditions have different types of bacteria from healthy controls [6]. There are many different methods of calculating alpha and beta diversity that take different factors into account. It is advisable to use the best one suited for your study data.

Graphs: The basic microbiome graph uses principal coordinates analysis (PCoA) to collapse the relationships between all the bacteria from an individual sample from a huge network to a single point on a 2D or 3D plot. Points (samples) on the plot that are close together are more similar and points that are far away from each other are more different. The clustering of points on the plot color coded for different factors can give insights into what is influencing the microbiome. An example will be shown and discussed.

Confounders: There an enormous number of confounders in a microbiome study and it is important to control for both biological variability and pipeline processing variations [7]. Biological variations that can influence samples are age, sex, diet, lifestyle, and even the time of day or the season. Things that influence results during processing samples into data are sample collection methods, shipping conditions, DNA extraction reagents, library construction, sequencing platform, and software processing.
Proper study design will account for these variables, but requires careful consideration of sample type and hypothesis. If the study is not well designed, analysis can take years and cost several hundred thousand dollars. It is always advisable to follow best practice recommendations [8]. It is also critical to keep and record all of the information related to these confounders in a excel file known as metadata (data about your data). Metadata is one of the most critical pieces of information that you create, especially if you are collaborating with someone else who is doing the 16S analysis for you.

What are they doing? One of the major limitations of microbiome analysis using the 16S marker gene is that while we can identify the bacteria present, we do not know what exact role they play in the ecosystem and how they influence the host. There are some additional analyses that can be done to attempt to asses function. Metabolomics (all of the molecules in a sample), transcriptomics (all the bacterial or host RNA), and proteomics (all of the proteins in a sample) are all methods to get more information about changing biochemical pathways. This information can help improve interpretation and conclusions about what is going on in the microbial ecosystem. All of these methods are all very expensive (multi-million-dollar machines and/or reagents) and time-consuming. Another way to get functional information is to do complete genome sequencing of all the bacteria present (shotgun metagenomics). While metagenomics is currently much more expensive and difficult than using the 16S marker, sequencing costs are continuing to decrease and methods to analyze everything is improving.

How might we use the microbiome in the future? While we cannot simply change our patient’s genome to one more beneficial for health, we can much more easily change the composition of the microbiome and the population of genes present there. For example, engineered probiotics are commensal strains that are genetically modified to produce a metabolite or enzyme that can actively treat a disease process. Phage therapy can also selectively alter the microbiome. It takes advantage of bacteriophage, viruses that very specifically target and kill bacteria. (Phage are to scalpel as antibiotics are to sledge hammer.) This has actually already been attempted in a few emergency cases in humans with great success. It was also found that phage can work synergistically with current antibiotics to fight multi-drug resistant bacterial strains [9]. There have even been a few recent studies suggesting that there is a predictable neoplasia microbiome. Small DNA fragments of these bacteria appear to be detectable in the blood, which means there really might be a blood test for various cancer types in the future. Furthermore, some day we may be able to use mutations in different bacterial species as biomarkers to non-invasively diagnose intestinal disease.

References:
Street Medicine: Providing for Pets of the Indigent

Jon M Geller, DVM, DABVP (Canine and Feline practice), CVPM, CHT-V

1. Homelessness in the US

In January 2015, 564,708 people were homeless on a given night. That number has not changed much through 2017.

*70% sheltered, 30% unsheltered*

Cars, couch-surfing, motel-sharing, uncounted?

2. Life on the Streets: A Hard Life Made Harder

*Man Charged With Trying to Kill Mannequin in Homeless Murder Sting*
March 7, 2017

- Service Animals for the homeless, especially women living on the street
- Service Animals vs Assistance Animals vs Emotional Support Animals

Homeless Dysfunction: Studies show up to 80% of chronic homeless have endured TBI

- US Homeless Veterans number 30,000
- Pets of the Homeless in the US:
  - Estimates: 5-10% of homeless individuals have pets in the US
  - 50,000-100,000 pets of the homeless, based on one million homeless.
- Homeless pets and pets of the homeless: what is the difference? Is a pet of a homeless person also homeless?

“Being homeless is illegal…” John Mark

- Oregon city's dog ban condemned as crackdown on homeless people
- Indianapolis moves homeless encampment out
- Santa Ana Riverbed shut down

“My Pet Always Eats First”, Irvine, L.: The homeless pet paradox

Economic Euthanasia:: An Inconvenient Truth- What is the incidence??

3. The Truly Indigent Client: What are the options?

- Friends or relatives?
- Free or greatly reduced services
- Relinquishment of pet
- Euthanasia


- At Animal Humane New Mexico, owners of pets seeking subsidized care at the nonprofit shelter's veterinary clinic must meet specified income limits and prove it by producing pay stubs, tax documents or comparable statements.
- Requirements at San Diego Humane Society are less exacting. .
- The Robin Hood Effect San Francisco SPCA

“Our hospitals are unique in that they are basically like a social enterprise,” said Dr. Jennifer Scarlett, a veterinarian and co-president of the organization “We’re not a low-cost hospital. Clients pay full price, and the revenue that is generated from that goes to support providing grants and basically, interest-free payment plan. No paperwork is needed in exchange for low-cost spay/neuter services, only the owner's word that he or she is strapped financially.

AVMA Statement on Means Testing

“Where applicable, means testing to determine eligibility should be conducted in compliance with each organization’s internal documents for clients accessing veterinary services.”
5. Non-profits making a difference

- Dove Lewis Emergency Animal Hospital
- The Velvet Assistance Fund
- The FACE Foundation provides financial assistance for San Diego animal owners who are unable to cover the full cost of their pets’ critical or emergency veterinary care. In-house 501(c)3, with many referrals from area clinics. Requires pre-qualification with documentation
- The Ladybug Fund
- Provides emergency funding for pet owners facing financial hardship “Trickle Funding”
- Dr. Steve Abrams Memorial Foundation – It’s a Jungle Out There
  
  Dr. Steve Abrams Memorial Foundation – Pet-Savers, Inc. *Dedicated to eliminating Economic Euthanasia by providing grants to pet parents who otherwise would be unable to provide critical Veterinary Care for their four-legged family members. Has close to 10 million dollars in pledged funding. No caps on case numbers or amounts.*

- Waggle.com
- Red Rover

6. Pets are not welcome in Homeless Shelters or on Public Transportation

- Public health considerations vs. personal needs
- Pet Policies of Public Transportation in Major US Cities and abroad

7. Expanding the scope of Shelter Medicine

- Noah’s Animal House, Las Vegas, NV- model Women’s Shelter project for pets of clients
- Animal Shelters adjoining Homeless Shelters: Family Promise for Pets, Scottsdale, AZ
- Animal shelters adjoining homeless shelters, Rehab centers, Hospitals, Prisons?
- PALS Domestic Violence Shelter, NYC
- Pet Friendly Homeless Shelter, Honolulu, HI

8. Taking Medicine to the Streets

- Mercer Clinic for the Pets of the Homeless
- WisCares medical services for pets of the homeless, started by UW vet students.
- Vet Touch, University of Minnesota
- The Street Dog Coalition: Provides free medical care to pets of the homeless
- Street Clinic at Las Vegas as part of WVC: Volunteerism as a wellness strategy
- Veterinary Street Outreach Services- Vet SOS: Pop-up Street Clinics in San Francisco
- Roxy’s Relief, Vancouver, Canada
- Pets of the Homeless, Reno, Nevada

9. One Health Clinics

- Knight’s Landing, California
- Venice Family Clinic (street outreach)
- Chicago Street Medicine team, Night Ministry

10. The art and science of street medicine

- Consider the risk factors for pets living outside 24/7
- History, history, history: it never repeats itself
- The Physical Exam: A lost art?
The color wheel
Diagnostics in the field

If you have a microscope:
- Blood smears, FNA cytology, UA strips, Ear swabs, Skin scrapings

Other field diagnostics
- Glucometer, Creatinine (JorVet), UA Strips, Azosticks
- Ophthalmoscope, SST, Flourescein dye strips
- EPOC/Istat- Blood gas/electrolytes
- ECG’s in the field: Alivecor
- Wireless Ultrasound probe

Getting creative with meds
- OTC
- Walmart $4.00 Rx
- Donated (damaged, short-dated) , not expired!

Home Remedies: Lotions and Potions
Street Medicine: wounds and trauma- inexpensive options
- Newspaper Splints
- Sugar and Honey
- Bubble wrap

Emergency spica, meta and back splints of newspaper: an economical way to manage fractures, luxations Jun 01, 2009 Dennis T. (Tim) Crowe, Jr.,

11. The future:
- The future of homelessness in the US: Are there better solutions?
- Free Supervised Campground with utilities for adults?
- Providing veterinary care to those that cannot afford it:
  Veterinary Medicine’s Biggest Challenge?
  Accessibility to Care, University of Tennessee
  Addressing the Needs of the Underserved Workshop, Family Medicine, Department of Family and Preventive Medicine, UCSD

Street Medicine: Doing More with Less: a new specialty?
Vet students are leading the way: The future looks bright
- Association for Student Run Free Clinics
- Street Medicine Institute
Contact information:
Jongeller6@gmail.com
970 219-1959
Thetstreetdogcoalition.org
Theladybugfund.org
HSVMA Animal Welfare Symposium
Controversies in wildlife research
Jon Geller, DVM, DABVP

Overview

Report of an IACUC Field Inspection and field mortality in Rocky Mountain National Park
Report of a research field mortality in Grand Canyon National Park
3 Cases of suspected rabies in captive bison leading to PEP
Report of a research field mortality in Grand Canyon National Park
Florida Panthers: Humane Lion-hunting strategies
Chronic Wasting Disease: A man-made disaster
Electro-fishing
Implications of field research on wildlife
What is the NPS IACUC?
The NPS IACUC: Established 2010
Assures compliance with the Animal Welfare Act in all wildlife research projects within US National Parks.
Projects involving harm, invasive procedures, and/or material alteration in behavior must be.

Implications of GPS Implant in Wildlife
Complications impacting animal health
Aesthetics and environmental impact
Are there alternatives to field darting?

2. Case Report: Rabies in Two Bison from Colorado
Rhyan,, Van Campen,, et al
Case Reports in Veterinary Medicine
June 2013

Bison dies of rabies at CSU research station.
3:10 PM, Jun 29, 2012

Larimer County officials said Friday that public health and veterinary experts believe the infected bison likely came into contact with a rabid skunk or possibly a fox or other wild animal with rabies.

A news release from the health department says two other bison have also died, but it wasn’t clear whether rabies was the cause. Health officials didn't immediately return a call.

The health department says CSU is taking steps to vaccinate the rest of the herd.

Were there actually 3 Bison Rabies Deaths?
‘We went to the landfill, dug up the bison and opened the cranial vault. The vault was entirely empty. The brain had liquefied and exited the cranial vault. I could not obtain even a remnant of brain tissue. Not even soup.’, Jack Ryhan, DVM, PhD, USDA-APHIS Supervisor

Who is supervising the supervisor?

RABIES Post-exposure prophylaxis (PEP)

Interviews with animal caretakers revealed that a skunk had been observed in the facility on May 25 wandering in a circuitous pattern and unresponsive to human presence. Over 8 persons had to receive PEP at significant cost.

Should preventive herd health be a responsibility of the research team, and should an IACUC require documentation of a herd health program?

Should there be a documented training program for animal caretakers?

Is there any accountability for adverse outcomes?

Death in Grand Canyon NP

NPS Biologists head into Grand Canyon NP in Sept 2012…

Should Wildlife Biologists be allowed to sedate large mammals without a veterinarian being present?

This resulted in the death of the Bighorn, despite reversal drugs.

Florida Panther Capture and Monitoring Project

Big Cypress National Preserve, Florida

Bull Elk GPS Project

How much are we changing the behavior of the animals we are trying to study?

Chronic wasting disease is a prion disease

Chronic wasting disease (CWD) infects deer, elk, and moose in North America. Although the origins of CWD are unknown, all research to date strongly suggests it is a non-native disease process that exerts unnatural effects on host populations in North America (Miller et al. 2000, Wild et al. 2011).

Dr. DeVivo and her co-authors found that the mule deer herd (in Southern Wyoming) could be extinct within 41 years, although selection for a known genetic resilience to CWD could preserve a population about one-tenth the size of the original—several hundred deer—through the next 100 years.

CWD- Ground Zero

Dr. Terry Spraker-

Late 1950’s to early 1960’s deer transported from western slope to DOW pens- potential contact with large herds of sheep.

There is a significant barrier to cross species transmission. Required serial intracerebral injection. Oral transmission in some squirrel monkeys, study repeated without transmission

Early 1970’s- Could some of the imported sheep have been infected with Scrapie?

Tame deer and elk were housed in pens on West Laporte Ave.and transported up to RMP for a Nutritional Bite Study- several animals were lost in the park.
1981- Elk collaring project by George Bear, noticed one of the collared elk was very thin, shot it, necropsy by Dr. Spraker, first case of CWD in the wild.

Other potential nose to nose contact between tame and wild cervids-

Ground zero for chronic wasting disease

“I have no doubt that Chronic Wasting Disease is a man-made disease.” Dr. Terry Spraker, DVM, DABVP

CWD Transmission

“We report that CWD can be transmitted to susceptible animals indirectly, from environments contaminated by excreta or decomposed carcasses.”


Prions are transmitted in urine

“…TSE infectivity is excreted in urine and may thereby play a role in the horizontal transmission of natural TSEs…”


CWD Prions in antler velvet

“Our studies indicate that antler velvet represents an additional source for human exposure to CWD prions. Widely used in traditional Asian medicine to treat a variety of ailments including impotence, arthritis, and high blood pressure, antler velvet can be readily purchased in caplet form and its usage has increased worldwide.”


CWD Transmission is mostly horizontal:

Detection of protease-resistant cervid prion protein in water from a CWD-endemic area

Direct and indirect transmission of CWD, cont

Under experimental conditions, mule deer (Odocoileus hemionus) became infected in two of three paddocks containing naturally infected deer, in two of three paddocks where infected deer carcasses had decomposed in situ =1.8 years earlier, and in one of three paddocks where infected deer had last resided 2.2 years earlier. Indirect transmission and environmental persistence of infectious prions will complicate efforts to control CWD …

Michael W. Miller, Colorado Division of Wildlife, Wildlife Research Center, 317 West Prospect Road, Fort Collins, CO 80526-2097, USA; fax: 970-472-4457

Increasing spread of CWD has raised concerns about the potential for increasing human exposure to the CWD agent. The foodborne transmission of bovine spongiform encephalopathy to humans indicates that the species barrier may not completely protect humans from animal prion diseases..


Oral Transmission of prions to nonhuman primates
After oral exposure, 2 squirrel monkeys had PrPres in brain, spleen, and lymph nodes at 69 months postinfection.


Cross species transmission:

Chronic wasting disease is a prion disease of cervids. Assessment of its zoonotic potential is critical. To evaluate primate susceptibility, we tested monkeys from 2 genera. We found that 100% of intracerebrally inoculated and 92% of orally inoculated squirrel monkeys were susceptible, but cynomolgus macaques were not, suggesting possible low risk for humans.


Is CWD Zoonotic?

"Terminal ill Utah hunter, age 30, could be the first victim of US 'mad deer' disease," read the headline of a press release issued last week from the Center for Food Safety in Washington, D.C. (1999)


CONCLUSION?

“The lack of evidence of a link between CWD transmission and unusual cases of CJD, despite several epidemiologic investigations, and the absence of an increase in CJD incidence in Colorado and Wyoming suggest that the risk, if any, of transmission of CWD to humans is low.”


Resulting long-term outcomes range from relatively low disease prevalence and limited host-population decline to host-population collapse and extinction. Our models suggest that disease prevalence and the severity of population decline is driven by the duration that prions remain infectious in the environment.

Electro-fishing for capture of research fish

Electro-fishing

Injury to Wild Brook Trout by Backpack Electrofishing
Bruce A. Hollender & Robert F. Carline
Pages 643-649 | Published online: 08 Jan 2011

More Questions than Answers

1. How can compliance be assured with wildlife research?
2. How should inspections occur? Where will funding come from?
3. Does wildlife research have scientific merit that justifies its impacts on wildlife?
4. Is it necessary? Does the need to know trump the risks?
5. How do we change the behavior of the animals we are trying to study?
Drug abuse and addiction in the veterinary workplace

Jon Geller, DVM, DABVP, CVPM, CHT-V
Veterinary Emergency and Rehabilitation Hospital,
Fort Collins, CO Jongeller6@gmail.com  Cell 970 219-1959

1. Drug Abuse and Addiction in the US
   Drug overdose deaths 46,000 in 2013, outpace deaths from motor vehicle accidents and firearms
   NOV 04 (WASHINGTON) - DEA Acting Administrator Chuck Rosenberg 4,100,000 in treatment =
   Loss of voluntary control, 21,400,000 addicted ,40,000,000 substance abuse/misuse, 260,000
   non-abusers

2. The Paradox

Despite the high level of environmental risk in the veterinary workplace, relatively little is done to reduce
the incidence of drug abuse, addiction and diversion.

Vulnerability + access

3. The Power of Addiction

BOULDER NURSE SENTENCED TO FEDERAL PRISON FOR TAMPERING WITH A CONSUMER
PRODUCT AND CREATING A COUNTERFEIT CON-TROLLED SUBSTANCE

EX-HOSPITAL WORKER GETS 39 YEARS FOR CAUSING HEPATITIS C OUTBREAK,

THE NURSE FIRED FROM A FRISCO HOSPITAL FOR FENTANYL THEFT WITHDREW ENORMOUS
QUANTITIES OF TWO NARCOTIC DRUGS FOR MORE THAN TWO MONTHS BEFORE SHE WAS
CAUGHT, a state investigation found.

JOHNSTOWN DOCTOR ARRESTED ON DRUG FRAUD CHARGES

4. Depression and Suicide

   CDC Notes from the Field: Prevalence of Risk Factors for Suicide Among Veterinarians — United
   States, 2014

Conclusion: …nearly one in 10 U.S. veterinarians might suffer from serious psychological distress and
more than one in six might have experienced suicidal ideation since graduation.

Prevalence of Risk Factors for Suicide Among Veterinarians — United States, 2014

• Depression and Suicide in the Veterinary Profession
• Stressors:
  • Demands of practice
  • Practice management responsibilities
  • Professional mistakes and client complaints
  • Compassion fatigue/Euthanasia
  • Long hours-challenging cases
  • Burnout
  • Access to psychoactive drugs
American Journal of Psychiatry
Ketamine for Rapid Reduction of Suicidal Thoughts in Major Depression: A Midazolam-Controlled Randomized Clinical Trial
Drug testing in the US Workplace

Between 55% and 68% of small businesses in the US perform pre-employment drug and ongoing employee drug screening.

….only about one-quarter of temporary employees (27%) undergo drug and alcohol screening either pre- or post-hire. Source: Quest Diagnostics

- Why Drug Test?

1. Safety-sensitive workers should not use drugs

The U.S. Department of Justice estimates that 50 percent of all on-the-job accidents and up to 40 percent of employee theft is related to substance abuse.

Accident = Medical Error?

- Why Drug Test?

1. Drug use is increasing

- Positivity rate, US Workforce: 9.3%
- Marijuana positivity in US Workforce: 14.3%
- Marijuana positivity Colorado Workforce: 20%

Source: Quest Diagnostics

- Drug users try to avoid drug testing

  (US Govt. survey of current drug users)

- 83 % indicated that drug testing was the number one deterrent to drug use and 27 % said they would resume using drugs if the Navy discontinued its drug testing program.
- 40 % said they were less likely to work for a company that conducted random drug testing. 30% said they were less likely to work for a company that conducted pre-employment drug testing.
- Employers who drug test have seen their drug test positivity rates decline over time

2. Types of Drug Screening

1) Pre-employment
2) Random screening
3) Post-Accident
4) For reasonable suspicion
5) Return to work

- What’s in Your Hospital?
- Morphine, Hydromorphone, Fentanyl, Methadone
• Buprenorphine, Butorphanol
• Diazepam, Midazolam
• Phenobarbitol, Pentobarbitol
• *Dexmedetomidine, *Xylazine
• Ketamine
• Alfaxalone, *Propofol
• Tramadol, *Trazadone, *Gabapentin

*Not controlled

6. Surveys on Drug Testing in the Veterinary Workplace

7. THC: An Employer’s Dilemma
Marijuana Positivity Increases 6.2 Percent Nationally in Urine Drug Tests, but by Double Digits in Colorado and Washington

‘Colorado Supreme Court says companies can fire workers for using medical marijuana in their off-hours’ By Mark Berman June 15, 2015 The Washington Post

Are Veterinarians who use marijuana “Safe to Practice”? Physicians who legally use medical marijuana … are considered unsafe to practice medicine in the state of Colorado, according to a policy from the Colorado Physician Health Program, headed by Doris Gundersen, MD.

• Veterinarians ‘Unsafe to Practice’?

• There were decision-making errors 50% to 70% of the time in long-time marijuana users compared with 8% of the time in nonusers (Neurology. 2006;66:737-739).

• "Even at levels as low as 3 ng/mL there are some mind-altering effects…a concentration of at least 10 ng/mL is required to relieve symptoms such as chronic pain or nausea."

8. Drug Testing: Ethical Considerations
Drug Testing: Creating a safe workplace or an invasion of privacy?

• Employers have a right to inquire into anything that seriously interferes with an employee rendering a fair day's work. It's a well-known fact that drugs can significantly impair a person's work performance, lowering productivity. Employees who use drugs have double the rate of absenteeism, higher job turnover rates, and cost three times as much in terms of medical benefits as those who don't use drugs.

• Business’s responsibility to protect employees and employees’ rights goes far beyond protecting the rights of those who choose to engage in illicit drug use. Employers not only have a right to strive to maintain a workplace free from drug abuse, they have a duty to do so.

• Can drug testing improve the level of trust in a practice?

• Drug Testing: Some Final Questions
  • Is it ethical?
  • Is it needed?
  • Does it work?

News Flash: Drug testing is falling out of favor as employers struggle to fill open slots
9. Employee Assistance Services

- Colorado Veterinary Practice Act: Veterinarian Peer Health Assistance Program: free for veterinarians, fee charged to non-DVM staff. This is the case in many states.

“I am a recovering addict and alcoholic and abused my own "secure" setup by falsifying entries/records. I refused "help" until my addiction nearly killed me. I didn't intend to commit suicide but I was practicing "passive" suicide. For me, the stigma of being an addict prevented me from seeking help. I'll never know whether any program offered by our profession would have helped but it would have been better if it was at least available. Including myself, I know of 3 other addicted colleagues and that is astounding if you consider I practice in a very rural area of Southern Illinois.”

10. Drug Abuse by Pet Owners

- Opioids were involved in over 40,000 human deaths in 2016, with overdose deaths four times higher than the late '90s [CDC]. Over half of US states do not require veterinarians participate in prescription drug monitoring programs (PDMP) but a small number of states have required that veterinarians check PDMP databases before prescribing opioids to pets. However, some veterinarians have pushed back arguing that it is not their patient abusing opioids, but the owner, and since they are not the owner’s physician they should not have access to the owner’s

- Novel cases: malingering by animal proxy
  H W LeBourgeois, 3rd 1, Tonya A Foreman, John W Thompson, Jr
  We report five cases submitted by veterinarians in which clients (pet owners) are strongly suspected or confirmed to have been engaging in malingering to obtain controlled medications for their personal use. Cases bear a striking resemblance to malingering in the general medical setting for drugs to abuse. We propose that veterinarians, like their medical counterparts, are potential targets of malingering by their clients for drugs of abuse.

JAVMA News
Journal of the American Veterinary Medical Association

February 1, 2017, Vol. 250, No. 3, Pages 236-237

https://doi.org/10.2460/javma.250.3.236

A third of states and the District of Columbia require reports from veterinarians when they dispense controlled substances, reflecting the current debate on how to reduce drug abuse

Hurting Pets for Attention: Mental Disorder or attempt to get psychoactive drugs!

- Phyllis DeGioia, Veterinary Partner Editor
  Date Published: 09/10/2002 Date Reviewed/Revised: 03/21/2018

A mental disorder called Munchausen syndrome causes people to deliberately cause physical illness to others as a way of getting sympathy and attention. A group of British researchers published a letter in Archives of Disease in Childhood (2002;87:263) that indicates that people with this disorder sometimes also claim their pets are ill, or purposefully hurt their pets, so that they can get attention. The researchers refer to this activity as Munchausen syndrome by proxy.

The research indicates that two percent of non-accidental injuries of a pet may occur as a result of the owner deliberately causing the injury.
Solutions That Work: The Art and Science of Street Medicine

Taking Medicine to the Streets

- Mercer Clinic for the Pets of the Homeless
- WisCares medical services for pets of the homeless, started by UW vet students.
- Vet Touch, University of Minnesota
- The Street Dog Coalition: Provides free medical care to pets of the homeless
- Street Clinic at Las Vegas as part of WVC: Volunteerism as a wellness strategy
- Veterinary Street Outreach Services- Vet SOS: Pop-up Street Clinics in San Francisco
- Roxy’s Relief, Vancouver, Canada
- Pets of the Homeless, Reno, Nevada

One Health Clinics

- Knight’s Landing, California
- Venice Family Clinic (street outreach)
- Chicago Street Medicine team, Night Ministry

The art and science of street medicine

- Consider the risk factors for pets living outside 24/7
- History, history, history: it never repeats itself
- The Physical Exam: A lost art?
- The color wheel
- Diagnostics in the field

If you have a microscope:

- Blood smears, FNA cytology, UA strips, Ear swabs, Skin scrapings

Other field diagnostics

- Glucometer, Creatinine (JorVet), UA Strips, Azosticks
- Ophthalmoscope, SST, Flourescein dye strips
- EPOC/Istat- Blood gas/electrolytes
- ECG’s in the field: Alivecor
- Wireless Ultrasound probe

Getting creative with meds

- OTC
- Walmart $4.00 Rx
- Donated (damaged, short-dated), not expired!

Home Remedies: Lotions and Potions

Street Medicine: wounds and trauma- inexpensive options

- Newspaper Splints
- Sugar and Honey
- Bubble wrap
Emergency spica, meta and back splints of newspaper: an economical way to manage fractures, luxations Jun 01, 2009 Dennis T. (Tim) Crowe, Jr.

The future:

- The future of homelessness in the US: Are there better solutions?
- Free Supervised Campground with utilities for adults: The Housing Laboratory
- Providing veterinary care to those that cannot afford it:
  - Veterinary Medicine’s Biggest Challenge?
  - Accessibility to Care, University of Tennessee
  - Addressing the Needs of the Underserved Workshop, Family Medicine, Department of Family and Preventive Medicine, UCSD
- Street Medicine: Doing More with Less: a new specialty?
- Vet students are leading the way: The future looks bright
  - Association for Student Run Free Clinics
  - Street Medicine Institute: International Conference
An Update on Neurological Emergencies
Michelle Carnes, DVM, MS, DACVIM
Specialists in Companion Animal Neurology
Naples, FL

I. Intracranial Emergencies
   a. Head Trauma
      i. Potential causes of mentation change

<table>
<thead>
<tr>
<th>Disease</th>
<th>Forebrain (FB) or Brainstem (BS)</th>
<th>Diffuse, Multi-focal or Focal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degenerative Disease</td>
<td>FB</td>
<td>Diffuse</td>
</tr>
<tr>
<td>Anomalous (i.e. hydrocephalus)</td>
<td>FB</td>
<td>Diffuse</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>FB and BS</td>
<td>Focal</td>
</tr>
<tr>
<td>Infectious/Inflammatory</td>
<td>FB and BS</td>
<td>Multi-focal or Focal</td>
</tr>
<tr>
<td>Trauma</td>
<td>FB and/or BS</td>
<td>Diffuse, multi-focal or focal</td>
</tr>
<tr>
<td>Vascular (CVA)</td>
<td>FB and BS</td>
<td>Focal or multi-focal</td>
</tr>
</tbody>
</table>

ii. Examination
A general physical examination is very important as the injured brain is very susceptible to changes in homeostatic functions of the body.

Cardiovascular Evaluation
Heart rate, heart rhythm, pulse quality, PCV/TS, mucus membrane color, and blood pressure should all be assessed on presentation.

Respiratory Evaluation
Airway patency, respiratory rate, respiratory character and thoracic auscultation should all be assessed on presentation.

Other
Hyperglycemia may indicate a more severe head injury; persistent hyperglycemia (>24h) has been associated with a higher mortality rate in people. Hyperglycemia has been demonstrated to worsen free radical production, excitatory amino acid release, cerebral edema, and vascular damage. Iatrogenic hyperglycemia should be avoided which is one reason why corticosteroid administration is contraindicated in head trauma patients. A minimum database should be obtained to assess renal and hepatic function prior to initiating treatment; jugular venipuncture should be avoided.
Neurological Examination
The aims of the neurological examination are to determine if the nervous system is affected, obtain a neuroanatomical diagnosis, and gain information about the prognosis using the modified Glasgow coma scale. Serial neurological examinations (q 30–60 minutes) are imperative to determine the stability of the patient and the best way to intervene.

iii. Pathophysiology of Intracranial Pressure (ICP)

The cranial vault is made up of 3 components - the brain (80%), blood (10%) and cerebrospinal fluid (10%). The Monroe-Kellie Doctrine states that in order to maintain cerebral homeostasis, an increase in any one component necessitates a decrease in another component. Without compensatory changes, ICP increases can lead to decreased oxygen and glucose delivery and neuronal damage. Acute elevations in ICP may trigger a "Cushing's reflex", which results in severe hypertension and reflex bradycardia. The presence of a Cushing's reflex indicates life-threatening increases in ICP and requires prompt treatment.

iv. Pharmacological Management of ICP

1. Mannitol
   a. 0.5 – 1.0 gm/kg IV over 20 minutes
   b. Effect lasts 2-5 hours
   c. May be repeated 2-3 times over 24 hours

2. Hypertonic saline (7%)
   a. 3 – 5 ml/kg IV over 5 – 10 minutes
   b. Effect lasts 1 hour
   c. Contraindications

3. Barbiturates

4. Aggressively treat/prevent seizures

b. Status Epilepticus

Anticonvulsant drugs that are used on an emergency basis commonly include diazepam or midazolam and phenobarbital. Diazepam (0.5–1.0 mg/kg) can be administered intravenously or rectally and midazolam (0.2-0.5 mg/kg) can be administered intravenously or intranasally. Doses can be repeated every few minutes; however, if the patient does not respond after the second dose it is unlikely to respond to further doses. If the patient presents in status epilepticus or clusters, after the seizure has stopped, loading of phenobarbital should be initiated. The loading dose is 15 mg/kg divided into 3 doses given intravenously as frequently as 30 minutes apart; however, the loading can be performed over 12-18 hours if seizures have become more controlled. Patients who continue to seize either during or after the phenobarbital load, should be placed on a constant rate infusion of midazolam (0.2–1.0 mg/kg/hr). Patients with significant hepatic disease may not tolerate benzodiazepines or phenobarbital. These patients could be alternatively managed with intravenous levetiracetam or potassium bromide per rectum.

II. Spinal Emergencies

The causes of acute spinal cord injury in dogs and cats are mainly compressive or concussive myelopathy due to intervertebral disc herniation (Hansen’s Type 1, acute non-compressive nucleus propulsus extrusion), vertebral fracture or subluxation/luxation. This mechanical compressive or concussive damage may lead directly to axon transection, demyelination, neural death and vascular disruption in the affected area of the spinal cord. Secondary biochemical injury to neural tissue follows this primary injury. Impaired blood flow and inflammation due to activated glial cells occurs in
the lesion (secondary spinal cord injury) result in ischemia and edema. In the most severe lesions, nociception is lost which may result in ascending-descending myelomalacia in around 10% of cases.

a. Assessment of spinal cord injuries

An initial brief neurologic exam should be performed to obtain a neuroanatomical diagnosis and determine if an unstable fracture is present. Non-ambulatory animals should be evaluated on presentation in the position in which they arrive. Until the presence of an unstable injury has been ruled out, all patients should be treated as if the spine is unstable. The initial neurologic exam should consist of a full cranial nerve exam and evaluation of mentation, segmental reflexes in all 4 limbs, superficial pain sensation in all 4 limbs, deep pain sensation in any limbs lacking superficial sensation, gentle spinal palpation to identify areas of instability, pain, crepitus, or malalignment, and panniculus reflex to provide additional localizing information in animals with thoracolumbar trauma. Schiff-Sherrington posture, due to lesions between T3–L3, is useful in the context of neurolocalization, but is not a prognostic indicator.

Management

Following neurological assessment, imaging studies should be performed to be able to decide if the patient is a candidate for medical or surgical treatment. Medical treatment can be successful for patients that present with pain only and stable fractures. Those cases presenting with neurological deficits and unstable fractures/luxations should be referred for surgery. Medical treatment consists of keeping the patient as immobile as possible; external coaptation can be used. Strict cage rest for 4-6 weeks is imperative. Good nursing care and pain management should be instituted.

b. Prognosis

The type and severity of the primary injury and the degree to which secondary injury has progressed will contribute to outcome. The most important indicator of prognosis is the presence or absence of deep pain perception. Patients with loss of deep pain perception at the time of presentation have a guarded to poor prognosis; if the loss of deep pain is secondary to fracture or luxation, the prognosis is grave.

References available upon request.
UPDATE ON SEIZURE MANAGEMENT
Michelle Carnes, DVM, MS, DACVIM
Specialists in Companion Animal Neurology
Naples, FL

I. INTRODUCTION
Seizure disorders in dogs occur frequently with an estimate of incidence over a lifetime varying from 0.5–5.7%. Seizures are the clinical manifestation of a paroxysmal cerebral dysrhythmia resulting from a transient disturbance in normal electrical pathways in the brain. Epilepsy is defined as recurrent seizures over time. Thus, a seizure is an event and epilepsy is the disease involving recurrent unprovoked seizures. Refractory epilepsy occurs when a patient has failed to become controlled with adequate trials of two seizure medications (called ACDs).

II. GOALS OF (CHRONIC) ACD THERAPY
The overall goal of ACDs is to eradicate all seizure activity however, this goal is rarely achieved. Most dogs benefit from ACDs by effecting a reduction in seizure frequency, severity or duration to a point that is acceptable to the owner without intolerable adverse effects of the drug(s) to the patient. The art of ACD therapy is finding the drugs that work the best in the individual patient and balancing the beneficial effects of the medication with the adverse effects.

Every patient is different, and must be considered individually, but it is recommended to start medication if:
- Any status epilepticus, clusters
- >3 seizures/6 months
- Severity of seizure or postictal change
- Owner’s wishes

III. REASONS FOR ACD THERAPY FAILURE
- Failure to obtain a diagnosis
  - Assumption of idiopathic epilepsy
  - Episodes are not seizures
- Advancement of disease
- Improper frequency of administration or dose
- Owner compliance
- Drug interactions
- Development of tolerance

IV. SELECTION OF APPROPRIATE ACDs
Anticonvulsant drugs modify the excitability of the neurons by altering membrane ion channels or the availability of neurotransmitters. The goal is to interfere with the rapid synchronous firing of neurons without impeding normal function.

Aspects that must be considered in the selection of an anticonvulsant include:
- Pharmacokinetics for that species
- Cost
- Dosing schedule
- Adverse effects
- Considerations of concurrent diseases that may affect drug metabolism or increase risks for adverse effects
VI. MANAGEMENT OF ACUTE SEIZURES (CLUSTERS OR STATUS EPILEPTICUS)

<table>
<thead>
<tr>
<th>Antiepileptic drug</th>
<th>T ½ (hrs)</th>
<th>TSS (days)</th>
<th>Suggested serum therapeutic range</th>
<th>Recommended dose</th>
<th>Possible side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenobarbital</td>
<td>32–89</td>
<td>10–18</td>
<td>20–35 mg/dl</td>
<td>2–3 mg/kg orally q12h</td>
<td>Sedation, ataxia, polyuria/polydipsia (PUPD), polyphagia, hyperexcitability, hepatotoxicity, induces P450 system, bone marrow dyscrasia, pancreatitis</td>
</tr>
<tr>
<td>Potassium bromide</td>
<td>21–24 days</td>
<td>2.5–3.0 months</td>
<td>1–3 mg/ml</td>
<td>20–40 mg/kg/day orally</td>
<td>Sedation, weakness, PUPD, polyphagia, pancreatitis, pruritus, behavioral changes</td>
</tr>
<tr>
<td>Topiramate</td>
<td>20–30</td>
<td>3–5</td>
<td>2–25 mg/l</td>
<td>2–10 mg/kg q12h orally</td>
<td>Vomiting, diarrhea, sedation</td>
</tr>
<tr>
<td>Clorazepate</td>
<td>5–6</td>
<td>1–2</td>
<td>20–70 μg/l (nordiazepam)</td>
<td>0.5–1.0 mg/kg q8–12h orally</td>
<td>Sedation</td>
</tr>
<tr>
<td>Zonisamide</td>
<td>15–20</td>
<td>3–4</td>
<td>10–40 μg/ml</td>
<td>2.5–10.0 mg/kg q12h orally</td>
<td>Sedation, loss of appetite, dry eye, ataxia</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>3–4</td>
<td>1</td>
<td>Not known</td>
<td>10–20 mg/kg q8h orally</td>
<td>Sedation, ataxia</td>
</tr>
<tr>
<td>Pregabalin</td>
<td>7</td>
<td>2–3</td>
<td>&gt; 2.8 μg/ml</td>
<td>3–4 mg/kg q8h–12h orally</td>
<td>Sedation, ataxia</td>
</tr>
<tr>
<td>Imepitoin</td>
<td>2–6</td>
<td>&lt;1</td>
<td>Not known</td>
<td>10–30 mg/kg BID</td>
<td>Polyphagia, TEL elevation</td>
</tr>
</tbody>
</table>

Acute, ongoing seizures present specific challenges in treating and controlling epilepsy. Multiple seizures are considered a cluster when >2 seizures occur within a 24 hour period. Status epilepticus is typically defined as continuous seizures or an individual seizure where the dog does not recover consciousness over a few minutes. Status epilepticus with generalized tonic-clonic seizure activity can be a life-threatening emergency and requires intensive nursing care and specific considerations with respect to treatment.

Rapid blood work should be done to rule out common extracranial causes for seizures: a complete CBC/Chem can be submitted when time permits.

- Blood glucose
- Body temperature
- Electrolytes
- Azostix
- PCV/TP

Whatever the underlying cause, it is imperative that the seizure is stopped. Provided there is not an obvious and readily correctable extracranial cause, benzodiazepines are the drugs of choice. Valium and midazolam are first-line anticonvulsant drugs in the acute care setting. More recently, intranasal midazolam has been shown to be effective and can be used if venous access is not readily available.

- Diazepam 0.5–1.0 mg/kg IV, 1–2 mg/kg per rectum
- Midazolam 0.2 – 0.5 mg/kg IV, 0.2 mg/kg intranasal
This will often break the cycle of status epilepticus and permit the patient to recover, but the seizures will likely recur if a long-acting ACD is not also administered. Phenobarbital is the most effective ACD, can be administered intravenously, and can be loaded to therapeutic blood levels quickly. Another drug that is effective is IV levetiracetam. If seizures continue despite or during loading of a long term ACD, starting a constant rate infusion (CRI) of benzodiazepines is best.

**Cannabinoids**

There is currently no information on the clinical use of cannabinoids for treating epilepsy in dogs and cats. Legal implications of cannabidiol (CBD) vary by state and they are still considered a Schedule 1 Drug under DEA. There are studies underway evaluating CBD and its efficacy in epileptic dogs. However, the recommendation of high potency CBD by veterinarians should also come with a discussion regarding potential toxicity. Dosing of 25–50 mg/kg/day of CBD has been recommended for humans with epilepsy; however, to this author's knowledge, there are no published dose recommendations for CBD use in dogs and cat. The FDA has approved a CBD drug for the indication of a specific drug resistant epilepsy syndrome in humans. Perhaps most importantly, there is evidence that CBD can change the metabolism of several drugs including phenobarbital and zonisamide; thus, significant caution should be exercised with polypharmacy and CBD.

References available upon request.
Common Spinal Diseases and Management
Michelle Carnes, DVM, MS, DACVIM
Specialists in Companion Animal Neurology
Naples, FL

Patients with spinal cord disorders are relatively common in small animal practice. Following a complete neurological examination, the practitioner should be able to localize the lesion and develop a list of differential diagnoses. In general, the spine is divided into 5 sections based on location of paresis and myotactic reflexes. Neuromuscular or lower motor neuron disease differs from spinal cord disease; these patients present with decreased to absent myotatic reflexes in all four limbs.

The Five Most Common Spinal Disease in Dogs:

1. Intervertebral Disc Herniation

Intervertebral disc herniation is the most common cause of myelopathy and spinal hyperesthesia in dogs. Chondrodystrophic breeds (Dachshund, French bulldogs, Beagle, Welsh Corgi, Basset Hound, Miniature Poodle, Shih Tzu, others) are at greater risk for disc disease and often present early in life (3–6 years of age). Clinical signs are based on where the disc herniation has occurred but typically include: spinal hyperesthesia, proprioceptive deficits and/or paralysis.

Initial diagnostics for a patient with presumptive disc disease can include a minimum database, spinal radiographs and thoracic radiographs, especially if the patient is geriatric. Spinal radiographs may help to rule out other differentials (e.g., aggressive bone lesion or discospondylitis).

Medical management is appropriate for dogs with pain and mild, ambulatory paresis. Pain management and cage rest are the hallmarks of medical therapy. The best approach to and success of medical therapy has been investigated in a limited number of studies. As many as 50% of nonambulatory dogs may regain ambulation with medical management but recovery is often longer (weeks to months until walking), incomplete (severe ataxia) and there is a greater chance for recurrence (30–50%). Strict rest (ideally in a crate) for 4 weeks should be emphasized as this allows the ruptured annulus to heal and prevent further extrusion of nucleus pulposus.

Surgical treatment is selected for dogs that are non-ambulatory paraparetic or -plegic or those which have a lack of clinical improvement with medical approaches. Surgery should be performed on an emergency basis for patients that are paraplegic/absent nociception, paraplegic with nociception or have minimal motor paraparesis, especially if clinical signs are rapidly progressive. Dogs with thoracolumbar disc herniation and intact deep nociception have a 86–96% of returning to voluntary ambulation, whereas dogs with absent nociception have roughly a 50–60% chance of recovery. Ascending/descending myelomalacia is seen in approximately 10% of dogs that have absent nociception secondary to intervertebral disc herniation. This syndrome is inevitably fatal and may develop even after surgery.

2. Fibrocartilagenous Embolism

Ischemic myelopathy due to fibrocartilagineous embolism (FCE) causes a peracute, often asymmetrical myelopathy that is non-painful and non-progressive over 6–12 hours. The clinical history often involves vigorous activity during which the dog screams and is then acutely unable to walk. Large and giant breeds are overrepresented for FCE, in addition to Miniature Schnauzers. Presumptive diagnosis is based on signalment, history and clinical signs. Antemortem diagnosis of FCE is made using MRI; in some cases it is a diagnosis of exclusion. Myelography may be normal in animals with FCE or may show focal intramedullary swelling. The prognosis for recovery of function following FCE is usually fair. In one study, > 70% of patients with presumptive FCE recovered ambulation. Most patients with severe clinical signs will have residual persistent deficits. Several prognostic factors have been established to assist in estimating recovery. These include size of patient, LMN vs. UMN injury, level of injury, symmetry of the lesion and presence of nociception.
3. Degenerative Myelopathy

Degenerative myelopathy (DM) is a slowly progressive degeneration of the white matter of the spinal cord. DM is a natural occurring model of human amyotrophic lateral sclerosis (ALS). The abnormal genes have been identified; most cases of DM are caused by a missense mutation in the superoxide dismutase 1 (SOD1) gene. In the Bernese Mountain dog the gene involved is SOD1. Historically, the disease was reported most often in the German shepherd dog (GSD). There are now many breeds affected with DM and the list continues to expand. Some larger breed dogs that may be affected include the Bernese Mountain dog, Boxer, Chesapeake Bay retriever, Golden retriever, Collie, Kerry blue terrier, Nova Scotia duck tolling retriever, Rhodesian ridgeback, Standard poodle, and Siberian husky. Affected small breed dogs include the miniature poodle, pug and Cardigan Welsh corgi. DM usually affects older dogs (i.e. 8-14 years) and presents as a chronic, progressive, non-painful thoracolumbar myelopathy. Definitive diagnosis is post-mortem histopathology. Antemortem diagnosis is based on ruling out other causes of chronic myelopathy. A genetic test for DM is available; the limitations of this test is that it does not prove clinical disease. One such test can be found at Error! Hyperlink reference not valid. www.caninegeneticdiseases.net/DM/sampleDM.htm. There is no treatment for DM however physical rehabilitation has been shown to significantly slow the effects of the disease. In one retrospective study of 50 dogs with a diagnosis of presumptive DM that received intensive physical rehabilitation had longer survival times (mean 255 days), compared with dogs that received moderate or no physical rehabilitation.

4. Infectious/inflammatory

Diskospondylitis is an infection of the intervertebral disk with extension to the adjacent vertebral endplates and soft tissues. Diskospondylitis causes severe spinal pain but may also cause myelopathy if inflammation results in compression of the spinal cord. The most common cause of diskospondylitis in dogs is usually bacterial; however, fungal diskospondylitis can occur, especially in German Shepherd dogs. Staphylococcus sp., Streptococcus sp., and E. coli are the most common bacteria implicated in diskospondylitis in dogs. Brucella canis is a rare cause of diskospondylitis in dogs but is important to rule out due to the potential for zoonosis. Spinal radiographs are often diagnostic for diskospondylitis in dogs; radiographs show lysis of the vertebral endplates adjacent to a disk with variable degrees to sclerosis and new bone formation. Treatment for diskospondylitis is ideally based on culture and susceptibility. Empirical antibiotic therapy with a cephalosporin such as cefpodoxime is appropriate. In a large, retrospective study, the average duration of treatment was 53.7 weeks. Radiographs should be obtained 4–6 weeks after diagnosis and then every 2–3 months to monitor for resolution. Oral antibiotics should be continued until there is radiographic resolution of lysis and ideally until radiographic changes are static.

5. Atlantoaxial luxation

Instability of the atlantoaxial joint that permits excessive flexion of the joint may result in compression of the spinal cord due to dorsal displacement of the cranial portion of the body of the axis into the vertebral canal. These conditions may result from congenital or developmental abnormalities, trauma, or a combination. Toy breed dogs are overrepresented, but atlantoaxial subluxation can happen in any breed of dog following trauma. Clinical signs associated with congenital atlantoaxial instability may have an acute onset, may be slowly progressive, or may be intermittent. Signs are indicative of a C1 and C5 myelopathy, and vary from mild cervical pain, to tetraparesis or tetraplegia and possibly death due to respiratory paralysis. Diagnosis of atlantoaxial instability is typically made from spinal radiographs; lateral, ventrodorsal and (carefully!) flexed lateral projections are made. In some cases, advanced imaging (MRI) may be necessary for definitive diagnosis. Medical management of AA subluxation involves exercise restriction with cage rest, external coaptation of the cervical spine and pain control. Nonsurgical management has been a plausible treatment option for dogs that show clinical signs of cervical spinal hyperesthesia with minimal neurologic deficits, or have owners with constraints that prevented surgical options or animals with immature bone that is not capable of withstanding implant placement. A retrospective study evaluated long term outcome in dogs with nonsurgical treatment for 1 year or longer
after removal of a cervical spinal splint that was in place for 12 weeks. A good outcome was reported in 10 of 16 dogs that had placement of a cervical spinal splint. Definitive treatment of AA luxation is surgical fusion of the joint. There are several variations of surgical technique, but the most commonly utilized approach is ventral fusion. Success rates vary from 61 to 90% in dogs treated with surgery. Positive prognostic indicators include: < 24 months of age, duration of condition <10 months and better preoperative neurological grade.

References available upon request.
The often acute, severe and progressive nature of central nervous disease can be challenging to manage in general practice. Due to the fact that the central nervous system is encased in bone, imaging the brain or spinal cord requires advanced imaging, and in most cases, MRI is preferred. Not all patients that present with signs of neurological disease require referral to a neurologist, nor will all pet owners be accepting of seeing a specialist. It is important to know what diagnostics can/should be done in general practice and at what point referral to a neurologist is indicated.

General Indications for Referral:

1. The patient is getting worse or not better with first line therapy.
   Most neurological diseases do not get better, and often get worse, if the appropriate course of treatment is not prescribed. If initial, first line therapy is not resulting in improvement or if improvement is short and transient, there is a high likelihood that either the initial diagnosis was incorrect, or the patient requires more aggressive/definitive therapy.

2. The disease is threatening life or limb.
   Severe disease presentations like paralysis or marked mentation abnormalities (stupor or coma) typically require aggressive targeted therapy. In most cases, these patients should be considered emergent and immediate referral to a neurologist should be facilitated.

3. The disease localization is uncertain.
   Our patients can display some very unusual paroxysmal behavior. In these cases, one may be uncertain if the disease process is affecting the CNS or some other body system. These patients can be particularly challenging for both generalists and specialists, alike, but it can be of benefit to work together to establish a rationale course of diagnostics or treatments.

4. The patient needs diagnostic and treatment modalities that are unavailable.
   In many neurological cases, it may be obvious where the problem is but in order to find out what the problem is, advanced diagnostics are often necessary. This may include MRI, spinal tap or electrodiagnostics. Similarly, if the patient requires neurosurgical intervention, referral to a neurologist is indicated.

5. The patient needs intensive care.
   Patients that have intracranial disease can have very severe disease presentations. Many seizuring or severe vestibular cases require continuous supportive care and attentive nursing in order to have a chance at a good outcome.

General Diagnostic Considerations:

The two most important initial diagnostics for patients with neurological disease are: a complete physical and neurological examination. Pet owners may make assumptions regarding what they are observing at home which can sometimes lead the clinician astray, especially if the basics are skipped. Following the complete examination, a list of differentials should be made, and a rationale diagnostic plan can be established. The following is a list of general diagnostic considerations for patients with neurological presentations.

1. Complete physical examination
   a. Otic
   b. Fundic
2. Complete neurological examination
3. Minimum data base: CBC, chemistry and urinalysis
4. Radiographs: Thoracic, spinal
5. Blood pressure
6. Infectious disease considerations
7. Thyroid testing
8. Bile acids
9. Anticonvulsant drug levels
The Practical Neurologic Examination
Michelle Carnes, DVM, MS, DACVIM
Specialists in Companion Animal Neurology
Naples, FL

IMPORTANCE OF A THOROUGH HISTORY AND PHYSICAL EXAMINATION
Before performing a neurological examination, it is important to first obtain a detailed clinical history and perform a complete general physical examination. In some cases, the patient may not exhibit the abnormality that the owner has witnessed at home. Information from the clinical history will then be the most important factor in selecting appropriate diagnostic and treatment options. Consider training your reception staff to encourage pet owners to record things they are seeing at home and bring the video(s) to their appointment. Neurological abnormalities can also be caused by systemic diseases, which can only be recognized after a thorough general physical examination.

OBJECTIVES WHEN PERFORMING A NEUROLOGICAL EXAMINATION

1. Determine if the problem is a neurological disease.
2. Localize which part of the nervous system is affected.
3. Devise a list of more common differential diagnoses based on neurolocalization.

BASIC COMPONENTS OF THE NEUROLOGICAL EXAMINATION

STEP 1: Observe the patient in the exam room.
It is helpful to have the owner place the patient on the floor while you are taking a more detailed history. This allows you to observe the patient without anyone directly interacting with it. Things to consider include: mentation, level of pain, appropriate or inappropriate behavior, circling, head-pressing, posture (head tilt/torticollis, kyphosis, low head carriage, tremors), general gait analysis (ataxia, hypermetria) falling/drifting, lameness vs proprioception deficits), visual acuity.

STEP 2: General physical examination.
A complete physical examination is necessary. Depending on the nature of the presenting complaint, certain aspects of the physical may be more relevant. For example, “difficulty walking” can be paresis, polyarthritis, bilateral cruciate ligament ruptures, etc. “Seizures” can be true seizures, syncope, reverse sneezing, etc. “Not able to get up” could be spinal or a hemoabdomen, weakness from systemic disease, pneumonia, etc.

STEP 3: Cranial nerve (CN) examination.
It is helpful to have an assistant restrain the patient so that you can perform a thorough neurological exam. Certain combinations of cranial nerve deficits can be indicative of disease processes; for example, CN VII and CN VIII often are indicative of otitis media/interna. In general, multiple cranial nerve abnormalities are consistent with brainstem localization. In forebrain disease, there may be a subtle asymmetry to the menace response or response to nasal stimulation. It is important to note that not all CN abnormalities localize to the brain and may be peripheral in nature.

STEP 4: Proprioception testing.
The most effective tests of proprioception in dogs and cats are paw placement (knuckling), hopping, extensor postural thrust and wheelbarrowing. Proprioceptive deficits are a strong and reliable indicator for the presence of neurological disease. Proprioceptive deficits can occur in animals with forebrain, brainstem, and spinal disease.

STEP 5: Spinal palpation.
While palpating the spine it is important to evaluate for subtle pain responses such as splinting of the abdomen, tensing of the epaxial muscles or cervical muscles. Palpation of the cervical spine and evaluation of cervical range of motion is performed. Some cases of cervical pain can result from intracranial disease.
STEP 6: Segmental reflex testing. Evaluation of segmental spinal reflexes, such as the withdrawal reflex and patella reflex, is necessary to localize a lesion in the spinal cord or neuromuscular system. It is important to recognize what a normal and complete withdrawal reflex looks like. This is a necessary aspect of the neurological exam when there is a gait abnormality and/or paresis.

STEP 7: Testing of nociception (“Deep Pain Perception”). This should only be tested in patients that have no voluntary motor function. In the paralyzed patient, this is one of the most important tests for evaluation of prognosis. Confirmation of a positive result requires the patient to provide some central/behavioral response, such as vocalization, pupil dilation, act of aggression or attempt to get away. A simple withdrawal of the limb is only a reflex and is not indicative of nociception.

LOCALIZE THE LESION

The combination of clinical signs and findings of the neurological examination is used to determine the neuroanatomical localization. This consists of one of the following: (1) forebrain, (2) cerebellum, (3) brainstem, (4) spinal cord, and (5) neuromuscular. The spinal cord is further divided into 4 functional spinal cord segments: C1-C5, C6-T2, T3-L3, and L4-S3 segments. Identifying the correct neuroanatomical localization is important to create a list of the most likely differential diagnoses.

1. Forebrain: Clinical signs may include seizures, decreased mentation, abnormal behavior, and central blindness. Additional abnormalities of the neurological examination include circling, asymmetric menace response, decreased response after stimulation of the nasal mucosa, and proprioceptive deficits. Proprioceptive deficits in an animal with a normal gait and behavior change is suggestive of a forebrain localization.

2. Cerebellum: Clinical signs include ataxia without paresis, hypermetria, central vestibular disease, and tremors. Cerebellar disorders are not associated with changes in mentation. In some cases, a decreased menace response may be identified.

3. Brainstem: Clinical signs include facial abnormalities, decreased mentation, generalized ataxia, hemiparesis, tetraparesis, and vestibular disease. Additional abnormalities of the neurological examination include proprioceptive deficits and cranial nerve deficits.

4. Spinal cord: Clinical signs can include a combination of ataxia and paresis, spinal hyperesthesia, and bladder dysfunction. Additional abnormalities of the neurological examination include proprioceptive deficits and alterations in spinal reflexes. Further classification into any of the 4 functional spinal cord segments is based on the number of affected limbs (tetra- or para-) and the presence of intact, increased, or decreased spinal reflexes.

5. Neuromuscular: The hallmark of neuromuscular disease is paresis without ataxia. Paresis in animals with neuromuscular disease can vary from exercise intolerance to flaccid tetraplegia. Other clinical signs include changes in voice and regurgitation. Additional abnormalities of the neurological examination can include decreased spinal reflexes and cranial nerve deficits.

References available upon request.
Vestibular Disease
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Naples, FL

Head tilt in dogs is a clinical presentation that most veterinarians encounter frequently in practice. The primary reason for a dog to exhibit a head tilt is dysfunction of the vestibular system. The vestibular apparatus is responsible for an animal’s maintenance of balance; the spatial orientation of the eyes, head, trunk and limbs relative to gravity. From a clinical perspective, it is divided into two components: peripheral, which involves the vestibulocochlear nerve (CN VIII) and its receptor in the inner ear; and central, which involves the brainstem and sometimes the cerebellum. Once a head tilt has been identified, the next step is to perform a complete neurologic examination of the patient to determine if the vestibular dysfunction is peripheral, central or paradoxical.

I. Clinical signs associated with localization

<table>
<thead>
<tr>
<th></th>
<th>Peripheral</th>
<th>Central</th>
<th>Paradoxical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of Consciousness</td>
<td>Normal</td>
<td>Normal or Impaired</td>
<td>Normal</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>Horizontal or rotary; &gt;60 bps</td>
<td>Horizontal, rotary or vertical; &lt;10 bps</td>
<td>Horizontal, rotary or vertical</td>
</tr>
<tr>
<td>Head Tilt</td>
<td>Ipsilateral</td>
<td>Ipsilateral</td>
<td>Contralateral</td>
</tr>
<tr>
<td>Leaning/Falling</td>
<td>Ipsilateral</td>
<td>Ipsilateral</td>
<td>Contralateral</td>
</tr>
<tr>
<td>Postural Reactions</td>
<td>Normal</td>
<td>Ipsilateral</td>
<td>Normal</td>
</tr>
<tr>
<td>Cranial Nerve Abnormalities</td>
<td>+/- CN VII, +/- Horner’s Syndrome</td>
<td>Any</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Gait</td>
<td>Normal or vestibular ataxia</td>
<td>Vestibular +/- proprioceptive ataxia</td>
<td>+/- Hypermetria Vestibular or Cerebellar Ataxia</td>
</tr>
</tbody>
</table>

II. Differential Diagnoses of Head Tilt in Dogs

Once the localization between peripheral and central vestibular disease has been identified, a list of differential diagnoses should be considered. In one study that evaluated 85 dogs that had MRI of the brain for the diagnosis of vestibular disease, 32% had peripheral vestibular disease, 44% had central vestibular disease and 25% had paradoxical vestibular disease.

<table>
<thead>
<tr>
<th></th>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degenerative</td>
<td>-</td>
<td>Storage disease</td>
</tr>
<tr>
<td>Anomalous</td>
<td>Congenital vestibular disease (rare)</td>
<td>Hydrocephalus, Chiari-like malformation</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Hypothroidism</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Vestibular nerve sheath tumor, otogenic neoplasia</td>
<td>Intracranial neoplasia – primary vs. metastatic</td>
</tr>
<tr>
<td>Nutritional</td>
<td>Thiamine deficiency</td>
<td></td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Geriatric (&quot;old dog&quot;) vestibular disease</td>
<td></td>
</tr>
</tbody>
</table>
III. General Diagnostics and Treatment Strategies

The diagnostic plan, treatment and prognosis can vary to a large extent based on whether the patient has peripheral or central vestibular disease. A careful history should be taken including: rate and duration of onset, whether the signs have progressed since onset, medications administered, diet, travel history, and existence of any other comorbidities. Physical exam should include a complete otoscopic exam and thorough neurological examination to determine if the patient localizes to peripheral or central disease. A minimum database, including CBC, serum chemistry, blood pressure and thoracic +/- bulla radiographs are recommended. If systemic infectious disease is suspected, serum titers or PCR for likely organisms can be performed. In patients where peripheral vestibular disease is suspected, additional diagnostic considerations may include: myringotomy with culture and sensitivity and a complete thyroid panel (T4, free T4 and TSH) based on clinical suspicion.

For patients that localize to central vestibular disease or those that are not improving with initial therapy, advanced imaging (ideally MRI) of the head and possible cerebospinal fluid analysis are highly recommended. Symptomatic treatment is often beneficial as many patients that have vestibular disease experience varied degrees of severity of clinical signs including nausea, anorexia, anxiety, recumbency, circling, falling and rolling. For dogs that are mildly affected, meclizine, an anti-emetic and anti-vertigo drug, is indicated. Other anti-emetics such as maropitant, ondansetron or chlorpromazine can also be considered. Patients more severely affected, will benefit from parenteral fluid therapy and intensive nursing care in an inpatient setting. During periods of severe vertigo (i.e. continuous rolling), these patients should be sedated to the point of relaxation; the author prefers intermittent dosing or CRI of dexametomidine at a low initial starting dose (62.5 micrograms/m² IV). In addition, these patients need to be situated in as much of a “normal” posture as possible while still rotating the patient every 4-6 hours. Following the acute phase of vertigo, physical rehabilitation appears to aid in the recovery of ambulation and is recommended.

References available upon request.
Diet choices in the first weeks and months of life can have long-lasting effects in growing puppies. Unfortunately, there are a number of prevalent myths about feeding puppies and as a result, both breeders and individual puppy owners often make poor diet choices for their puppies.

**Commercial Diets**
Growing puppies should always be fed high quality commercial diets designed for either growth or “all life stages” until they reach skeletal maturity (approximately 12 months for smaller breeds and up to 18 months for giant breeds). Ideally, the diet chosen should have undergone the Association of American Feed Control Official (AAFCO) feeding trials to make sure that it actually does support healthy growth of puppies. However, there are fewer and fewer companies that do feeding trials each year.

Whether feeding trials are utilized or not, it is wise to stick with well-established companies that have proven track records of experience with and research into growth and reproduction. Growing animals are much more sensitive to deviations from the nutritional ideal than are adult animals and it is important to ensure that the diet being fed has been tested before in puppies rather than just marketed for use in them. The growth period is not a good time to try out “trendy” diets with exotic or unusual ingredients.

There are pros and cons to feeding a diet marketed for “all life stages” versus one marketed only for growth. Some “all life stages” diets may be marketed as adult diets but may have been tested with feeding trials for puppy growth (e.g. Purina Dog Chow). Others only meet puppy requirements on paper and have never undergone feeding trials. Essentially, “all life stages” foods are puppy diets that are designed to also be fed to lactating bitches and adult dogs. This one-size-fits-all approach has advantages and disadvantages. For the multiple dog family, these diets may solve the problem of having to feed different diets to dogs of different ages. However, these diets often contain excessive calories for adult dogs at maintenance and potentially also too much calcium for growing large and giant breed puppies. For smaller breed puppies, it probably doesn’t matter if a “growth” or “all life stages” diet is fed.

For large breed puppies (above 50 pounds adult weight, although AAFCO uses 70lb as their cutoff), there are new recommended maximum limits for calcium and phosphorus that are currently being phased in. With these new guidelines for large breed puppies come new AAFCO statements for diets that are formulated to meet AAFCO nutrient profiles. While the new statements are still being phased in, they require that diets state whether or not they are appropriate for “growth of large breed dogs (adult weight greater than 70 pounds)”. The challenge with these new statements is that the wording is very similar (“including” vs “except for”) between the two options, making it easy to misread them.

**Home-prepared Diets**
In the author’s experience, the most severe cases of nutrition-related disease in growing puppies are nearly always the result of feeding home-prepared diets. Nearly all home-cooked diet recipes obtained online and in books are deficient in essential nutrients as has been shown in several studies and often the inadequate nutrients (e.g. calcium) are the ones most essential for growth. Growing animals are acutely susceptible to nutritional imbalance and the result of seemingly small errors in formulation can be life-long. It is therefore extremely important that growing puppies not be fed home-prepared diets. Clients should be strongly encouraged to wait until the puppy has reached at least a year of age (small breeds) or 18 months (giant breeds) before starting a home-cooked diet if this is their preference. Clients should be advised to obtain a recipe from a Board Certified Veterinary Nutritionist™ to ensure that an appropriate, nutritionally balanced, home-cooked diet is fed.

**Raw Diets**
Likewise, raw diets, whether home-prepared or commercial, should not be fed to growing puppies. Not only are many of these diets (even commercial products) deficient in essential nutrients, but the risk of serious food-borne illness secondary to contamination is more acute in young animals with developing immune systems.

**Developmental Orthopedic Disease (DOD)**
Developmental orthopedic diseases (DOD) are common in large and giant breed dogs and include hip dysplasia, osteochondrosis, panosteitis, hypertrophic osteodystrophy, and a variety of others (e.g., ununited anconeal
process, fragmented coronoid process). These diseases have multifactorial pathogenesis and are largely influenced by genetics, biomechanical forces, and nutrition. Nutrition is now known to have a major influence on modifying a dog’s genetic predisposition to developing DODs. Therefore, it is important to counsel the owners of large- and giant-breed dogs so that the incidence of these diseases is minimized.

There are two major known nutritional risk factors for DOD - rapid growth and excess calcium intake. Of these, rapid growth caused by excessive calories seems to be most prevalent. Puppies will put excess calories into growth before adding fat mass, so even a slightly fat puppy is likely growing at an unsafe rate. This problem is compounded by the fact that not only do these puppies carry more weight on developing bones, but there is evidence that rapid skeletal growth results in bones that are less able to handle the stress of increased body mass.

Multiple studies have shown that restriction of calories in growing large breed puppies reduces the risk for hip dysplasia, osteochondrosis, and osteoarthritis while provision of inappropriate quantities of high calorie diets can increase risk of these conditions. In one well-designed study, Labrador retriever puppies who were fed free choice versus sex-matched littermates that were fed 75% of their littermate’s intake, had over 2 times the rate of hip dysplasia as their calorie-restricted siblings. Likewise, radiographic prevalence of osteoarthritis in the hip and shoulder and severity of osteoarthritis in the elbow were significantly less in the calorie restricted dogs at 8 years of age. Caloric restriction and slowed growth was also associated with a significantly longer median lifespan (18-24 months) without additional morbidity. Caloric restriction does not alter the final size of the puppy, but alters the amount of time that it takes to reach adult size.

Excessive dietary calcium has also been linked to DOD. Unlike adult dogs, puppies have minimal regulation of intestinal calcium absorption for the first 6 months of life. Therefore excess dietary calcium is very readily absorbed even when the total body calcium exceeds physiologic needs. Hypercalcemia (even transient) leads to increased secretion of calcitonin which in turn may result in aberrant calcium deposition in rapidly growing bones, leading to abnormalities. The new AAFCO guidelines for large breed puppies recommend 3.0 g/1000 kcal as a minimum and 4.5 g/1000 kcal as a maximum. There are currently both puppy and adult diets on the market that contain way more calcium than is considered safe for large breed pups. On the other hand, there are some large breed puppy diets available that have been demonstrated to support normal puppy growth via feeding trials that contain as low as 2 g/1000 kcal calcium (The adult maintenance minimum is 1.25 g/1000 kcal). All diets that contain calcium below the AAFCO minimum of 3.0 g/1000 kcal require feeding trials to be sold as complete and balanced.

Although there are no specific regulatory guidelines for calorie content for advertised large breed growth diets, they are typically lower in energy density than “regular” puppy diets. These diets may also be supplemented with compounds such as glucosamine, omega-3 fatty acids, and chondroitin for potential orthopedic benefits. The common practice of switching large breed puppies to adult diets before one year of age should be avoided as this may result in inappropriate amounts of calcium, calories, and other essential nutrients being fed.

Many people still believe that dietary protein is a risk factor for DOD in dogs. However, studies have shown no effect of dietary protein when calcium and energy consumption are controlled. Vitamin C is also unrelated to DOD.

**General Feeding Recommendations**

Small and medium breed puppies – (< 50 lb adult weight, low risk of DOD) – Feed a standard puppy or “all life stage” diet to maintain a body condition score of 4-5/9 throughout growth. Feeding free choice or many small meals is likely appropriate during the first few weeks after weaning, but most small breeds should be able to be weaned onto 2 meals per day by around 6 months.

Large and giant breeds (> 50 lb adult weight) – Feed a large breed growth diet that has passed AAFCO feeding trials for growth until adult size is reached (12-24 months). Feed to maintain a body condition of 4/9 during growth and maintain a 4-5/9 throughout adult life. Puppies should always be meal-fed rather than allowed ad libitum consumption. Generally puppies over the age of 6 months can be fed twice daily. Large breed puppy diets with higher or lower caloric densities can be used as needed to balance feeding amounts, nutrient needs, and
individual metabolism. Puppies should be weighed and their food intake evaluated every 2 weeks during the growth phase. When in doubt, it’s better to have a “ribby” puppy than even a mildly overweight one.

For larger puppies of unknown parentage, or puppies of mixed breeds known to be prone to orthopedic disease (such as retriever and shepherd mixes), it is likely safest to treat them like known large breed puppies. Transition to an adult maintenance diet can begin at 1 year of age.

Preventing obesity
The time to prevent obesity is during the growth phase and early adulthood. It is important to discuss body condition scoring and weight management at all puppy appointments and reinforce this information every time the puppy comes in to the clinic for any reason. Every puppy owner should be taught how to body condition score their puppy and sent home with a copy of a body condition score chart (9 point scales are readily available from pet food manufacturers and other sources such as the AAHA Nutritional Assessment Guidelines and the WSAVA Nutrition Toolkit: www.wsava.org/nutrition-toolkit). When in doubt, it is likely safer to err on the side of slightly too thin.

At the time of spay or neuter, it is important to advise owners that energy requirements will decrease after surgery and many puppies will need to be fed less (up to 20% + less). This is a good time to reassess the diet that is being fed and determine whether switching to an appropriate puppy diet with a different caloric density would be useful.

Supplements
Growing puppies, especially large breeds, should not be supplemented with any vitamin or mineral supplements. These supplements are unnecessary when a balanced commercial diet is fed and can contribute to DOD. Some breeders insist that specific supplements are fed in order for the hip/health guarantees to be valid. This practice likely puts the puppies at greater risk of developmental problems but this information has clearly not been widely disseminated enough to prevent such restrictions. It is important to counsel owners about the risks of such supplements so that they can make an educated decision on whether to follow the breeders’ recommendations. Breeder clients should also be educated as to the known causes of DOD and ways of preventing the condition.

References
Common Pet Food Myths and Misconceptions
Cailin Heinze, VMD, MS, DACVN

There are many myths and misconceptions about pet nutrition and commercial pet foods that are commonly propagated online, in written marketing material for various pet food companies, and by third party "non-biased" sources such as pet lover magazines. Many clients will believe the propaganda and develop strong opinions of various diets based on incorrect or misleading information. It is important for all veterinarians to understand which commonly repeated mantras are accurate versus those that are only used to sell product and to be able to effectively communicate this information to their clients. Below, some common myths are discussed and debunked.

1. “By-products are bad”

   The American Association of Feed Control Officials (AAFCO) defines a meat by-product as “the non-rendered, clean parts, other than meat, derived from slaughtered mammals. It includes, but is not limited to, lungs, spleen, kidneys, brain, livers, blood, bone…and stomachs and intestines freed of their contents. It does not include hair, horns, teeth and hoofs…If it bears name descriptive of its kind, it must correspond thereto”.

   Chicken by-product is defined as “ground, rendered, clean parts of the carcass of slaughtered chicken, such as necks, feet, undeveloped eggs and intestines, exclusive of feathers, except in such amounts as might occur unavoidable in good processing practice.”

   While many Americans may feel uncomfortable with these types of foods in their own diets, it is important to realize that in many cultures, these parts are regularly consumed and often even considered to be delicacies! Some of these items may be very high in nutrients (e.g. organ meats, undeveloped eggs). Furthermore, when dogs and cats have access to whole prey, they often eat these parts first.

   It has become common for many manufacturers to advertise that their foods do not contain “by-products” and also to give definitions of by-products on their websites or written advertisements that may differ substantially from the legal AAFCO definition. For example, I’ve seen websites list by-products as including road kill, “euthanized dogs and cats including collars”, hooves, hair, and teeth. Other manufacturers include ingredients that could be considered by-products – such as liver – and list it simply as “liver”, rather than as a by-product, then advertise that they contain no by-products.

   By-products can definitely vary in quality and the consumer has to trust the manufacturer to use a high-quality product (many ingredients can vary greatly in quality, so this is usually the case, not just with by-products). Good manufacturers are very particular about their suppliers and inspect every shipment and perform analytical testing to ensure that it meets their nutrient specifications.

2. Bacteria in raw meat doesn’t make dogs or cats sick

   There is a plethora of misinformation regarding the safety of raw diets for pets. Many producers and promoters of raw diets either deny that their products potentially contain pathogenic bacteria such as Salmonella or E.coli or state in their marketing materials that animals do not get sick from these bacteria. It has been impossible not to notice that there have been numerous large scale recalls in the past few years of meat intended for human consumption due to contamination with pathogenic bacteria. There have also been dozens of recalls of raw pet foods for bacterial contamination in the past 5 years. It is safest to assume that all raw meat is contaminated, whether intended for human or animal consumption.

   While it is true that dogs and cats may be less sensitive to foodborne illness than people, there are numerous documented cases of severe and even fatal disease attributed to bacterial contamination of foods. Additionally, it is likely that many milder cases go undiagnosed as the clinical signs – vomiting, diarrhea, ± fever - are far from pathognomonic. Recent publications have suggested a link
between Campylobacter in raw chicken and cases of acute polyradiculoneuritis in Australia and cases of *Mycobacteria bovis* in cats transmitted from their raw beef diet.

Despite some manufacturers’ claims, there is no evidence that washing meat in hydrogen peroxide or grapefruit seed extract renders it sterile. Moreover, one study demonstrated that most methods that people use to sanitize pet dishes after feeding raw meat were inadequate to kill pathogenic bacteria. Although some diet manufacturers have started using high pressure processing (HPP) to kill bacteria in raw diets, this technique is rather new to the pet food industry and its efficacy is still unclear.

In addition to concerns with pet illness, direct contact with contaminated pet treats has been linked to human infections with pathologic bacteria. As in pets, it is likely that many other milder infections go undiagnosed and therefore unreported. There is even greater risk to people from contact with animals that consume diets contaminated with pathogenic bacteria. Raw food-fed pets have been shown to shed viable pathogenic bacteria, sometimes asymptotically, putting all people in the household at risk. Of particular concern are households with young children, the elderly, or people with weak or suppressed immune systems (such as the elderly and HIV or cancer patients).

It is important to keep in mind that while many clients (and even some veterinarians) may believe very strongly in the benefits of raw-feeding, there is no scientific evidence to substantiate any of the anecdotal benefits of raw feeding over feeding a cooked diet with similar ingredients. However, there is significant evidence to suggest that these diets and raw meat treats can cause harm to both pets and their human companions.

3. **Grains are bad for dogs and cats**

Another common myth is that dogs and cats lack the necessary enzymes to digest grains and that grains cause allergies, obesity and other health problems. Of the grains, corn is most often maligned. The truth is that properly cooked grains are generally well utilized by both dogs and cats. Each grain has a distinct nutritional profile and differing amounts of protein, fat, vitamins and minerals as well as amounts and types of fiber. It is overly simplistic to make blanket statements that apply to all grains as there are few strict commonalities that would lead an animal to react adversely to the entire group as a whole.

While allergies to grains have been occasionally documented, they likely reflect their widespread inclusion in commercial diets rather than enhanced allergenicity. In the author’s experience, most “grain allergic” dogs are not truly allergic. Diagnosis of food allergies is complicated by the lack of accurate testing modalities other than exclusion and re-challenge. In addition, many diets contain multiple common protein sources (e.g. corn, wheat, beef, dairy and chicken), meaning that any one of those proteins could be causing the reaction and it can be difficult to figure out which ingredient is the problem without a structured re-challenge which tends to be a hard sell to owners. There is also no evidence to support claims that corn (or any other grain) is responsible for health problems outside of the rare dog with a true allergy or familial gluten-sensitivity in some Bull Terriers and Irish Setters.

Many “grain free” dry diets simply substitute potato, tapioca, peas, lentils, or beans for the grains that would otherwise be in the diet. These diets may contain similar amounts of carbohydrates as grain-containing diets. Interestingly, while whole grains contain many vitamins, minerals and various types and amounts of fiber, potatoes and tapioca are relatively pure starches which contribute minimal amounts of most nutrients. Substituting potato or tapioca for whole grains may actually defeat one of the touted benefits of grain-free diets – fewer simple carbohydrates.

Recently there have been hundreds of cases of dilated cardiomyopathy reported to the FDA in dogs eating diets that contain exotic meats (e.g. kangaroo) and higher amounts of potatoes, peas, lentils, or beans. While some of the cases (especially in golden retrievers) are taurine-deficient, this does not seem to be the main mechanism for the majority of the cases that have been reported.
Until there is a clear mechanism for DCM discovered, it is recommended to avoid grain free diets and those with exotic meats.

4. Flax is a good source of omega-3 fatty acids
As research shows numerous health benefits of omega-3 polyunsaturated fatty acid (PUFA) supplementation in humans and animals, many pet food companies supplement their diets with omega-3 fatty acids and include this information in marketing materials.

There are two main groups of omega-3 fatty acids — short chain (α-linolenic acid, ALA, 18:3) and long chain (eicosapentaenoic acid, EPA, 20:5, and docosahexaenoic acid, DHA, 22:6). ALA is produced by terrestrial plants and can be found in flax, canola, walnuts, and other seeds and seed oils. EPA and DHA are found only in certain marine plants and in cold water wild-caught marine fish such as cod, salmon, and anchovies. While most mammals possess the enzyme (delta-6 desaturase) necessary to convert ALA to EPA and then DHA, this conversion process is uniformly poor in the species that have been studied — conversion rates range from 0-15%. Cats in particular have very low activity of the necessary enzyme and are unlikely to be able to produce clinically relevant amounts of the long chain omega-3s, regardless of the ALA concentration in the diet. (This low enzyme activity also is responsible for the essentiality of arachidonic acid (20:4 n-6) for cats, as it is produced from linoleic acid (18:2 n-6) using the same enzymatic pathway.)

The vast majority of the veterinary research demonstrating benefits of omega-3 fatty acid supplementation has investigated supplementing EPA and DHA directly rather than ALA. However, some of the research for osteoarthritis used a diet that contained EPA, DHA and a significant amount of ALA. To attempt to duplicate the tissue EPA and DHA concentrations using only ALA, 6 – 10 times the amount of EPA and DHA would have to be provided as ALA (for dogs). As fats contribute between 8.5-9 kcal per gram, this is a difference of as much as 81 kcal per dose based on a one gram daily dose (a small dog or cat may only consume 250 kcal per day). Additionally, omega-3 fatty acids of all types are prone to oxidation secondary to their multiple double bonds (polyunsaturation). High amounts of these fats of all varieties require careful attention to antioxidant provision and diet storage. It may be safer to use smaller amounts of concentrated long-chain omega-3s rather than large amounts of ALA.

It is important to examine the ingredient list of any foods that advertise omega-3 fatty acid supplementation to ensure that this supplementation is mainly in the form of EPA and DHA. Other ingredients that may indicate higher levels of EPA and DHA include fish oil, salmon oil, cod liver oil, fish meal, herring meal, and algal meal. The best way to determine the amount of omega-3 fatty acids in a diet is to contact the manufacturer and ask specifically about total omega-3, DHA and EPA concentrations. If there is a large difference between total and DHA + EPA concentrations, the remainder is likely ALA, even if typical sources of ALA (e.g. flax, canola oil) are not included in the ingredient list.

5. Natural diets are better
AAFCO defines natural as “a feed or ingredient derived solely from plant, animal, or mined sources, either in its unprocessed state or having been subject to physical processing, heat processing, rendering, purification, extraction, hydrolysis, enzymolysis or fermentation, but not having been produced by or subject to a chemically synthetic process...”. To label a pet food as “natural” requires that no synthetic compounds of any kind be included. However, phrasing such as “natural diet with added vitamins” is typically used to acknowledge the fact that most diets are supplemented with synthetic vitamins, amino acids or mineral complexes.

The current definition of natural gives little information about ingredient or product quality as rotten meat or leather (to make an extreme point) would be defined as natural whereas synthetic taurine would make the diet “unnatural”. Natural sources of many vitamins, minerals and amino acids used in commercial pet foods are not always practical and many synthetic sources are metabolically indistinguishable from natural forms.
Some commercial diets attempt to provide for all required nutrients using only whole foods. This approach, while emotionally appealing, presents several problems. First, these diets generally contain large numbers of ingredients sourced from many different vendors. Vitamin and mineral contents of ingredients such as fruits, vegetables and kelp (a commonly used iodine source) tend to vary, sometimes substantially, between sources and even by season. Therefore, a diet that meets all nutrient requirements in one batch may not meet them for the next batch, unless all the ingredients are analyzed with each batch and the appropriate safeguards built into the formulation. Ingredient analysis can be quite expensive, so it is common that individual shipments of ingredients may not be thoroughly screened, especially by smaller companies that purchase many different ingredients and lack the ability to do in-house analysis.

Whole food ingredients never provide only one nutrient, thus adding enough of a food to meet one nutrient requirement can result in an excess of another nutrient. Similarly, some ingredients such as taurine become less bioavailable with cooking (others become more available); it may be difficult or impossible to provide adequate amounts of these nutrients from whole food sources without altering the nutrient profile of the whole diet.

Additional ingredients also increase cost and can complicate diagnosis of food intolerance or allergy if the pet ceases to tolerate the diet. Synthetic vitamins and minerals, despite their chemical sounding names, should not be cause for concern. In fact, inclusion of these supplements increases the likelihood that the diet will provide all the essential nutrients required for a particular life stage.

Artificial preservatives are becoming uncommon in pet foods, mostly due to public opinion rather than any documented adverse effects. While there are many “natural” antioxidants such as vitamin C, vitamin E (mixed tocopherols), and rosemary extract that can be used in pet foods, these preservatives tend to be less effective than their synthetic counterparts and necessitate more attention be paid to setting appropriate expiration dates, proper storage and monitoring for spoilage.

References

Despite increased interest in nutrition from pet owners, multiple surveys have reported that client compliance with dietary recommendations, especially for veterinary-exclusive diets (we’ll call them therapeutic diets from here on out) is, to put it kindly, not always optimal. One of the causes of poor compliance with dietary recommendations may be inadequate communication between the veterinarian and the pet owner about the importance of diet modification to the diagnosis or treatment of the pet and the rationale for the recommendation of specific diets. Some veterinarians may reach for therapeutic diets without a good understanding of the theory or evidence behind these diets or may not feel comfortable discussing this information with clients while some pet owners may be inclined to dismiss all recommendations for therapeutic diets due to perceptions that the veterinarian is in cahoots with the pet food manufacturer or because of concern for the quality of foods produced by “Big Pet Food”.

For some medical conditions, therapeutic diets may not necessarily provide tangible benefit to the pet, while in other diseases they may be critical to diagnosis or disease management. Communicating this information effectively to pet owners may improve client trust as well as compliance.

Diseases/conditions where therapeutic diets should be considered to be the first choice and standard of care include IRIS stage 3 or 4 kidney disease, severe hyperlipidemia, hepatic encephalopathy, copper-associated hepatopathy, some types of urolithiasis, and for diagnosis of potential food allergies. Diseases that often do equally well on carefully chosen over-the-counter foods include diabetes, heart disease, many cases of liver disease and mild fat intolerance.

Addressing common resistance to the use of veterinary therapeutic diets

The internet and various lay publications for dog owners are often full of misinformation regarding the purpose, efficacy, quality control, and ingredients used in therapeutic diets. Many therapeutic diets utilize grains and by-products, which are both untrendy in today’s pet food market. A discussion of the current concerns regarding exotic and grain-free diets and dilated cardiomyopathy may help convince some clients to feed grains. Pointing out that by-products are typically things like organ meats and other perfectly healthy parts of the animal that people in the US don’t like to eat but that are commonly eaten in Europe and elsewhere can also help to allay fears. For certain diseases, these ingredients may be intentionally used due to their nutrient levels – for instance, chicken by-product meal is typically richer in nutrients per calorie than chicken meal, which may make it a better option for a low calorie diet such as for weight loss. White rice and brewer’s rice are low in protein, phosphorus, potassium, and sodium, making them great options for renal diets.

The author typically emphasizes to clients that she would never recommend a diet that she wouldn’t feed to her own pet and that wasn’t high quality. Clients can be advised that inclusion of these ingredients can be taken as a sign that the company is more interested in putting their money into the science and quality control rather than on fancy ingredients that appeal to the pet owner but have little or no benefit for the pet.

Many pet owners have concerns that therapeutic diets are more expensive than OTC diets, which may be true if they are feeding brands purchased at the grocery or big box store. For owners purchasing higher end brands from pet supply stores or pet “boutiques”, there may not be a dramatic difference in cost. It should be emphasized that an ideal diet for a pet with health concerns can save plenty of money in the long-run, such as if a cystotomy for uroliths is prevented or delayed. You can calculate the cost of feeding a therapeutic diet vs their current diet using this calculator: https://vetnutrition.tufts.edu/2019/06/pet-food-cost-calculator/

You may also consider scripting pet foods to online retailers, especially if you don’t carry a specific diet in stock. While this reduces [the often small] profit on selling diets in-house, it can improve compliance, reinforcing to the client that your goal is their pet’s health, not profit, and allows access to diets from manufacturers that your practice may not otherwise carry, which can be a huge benefit as each company has diets that are their “stars” and it’s not always possible to carry all of them.

Home-cooked diets

For medical conditions where a therapeutic diet is indicated, but existing diets are not acceptable to the client or the pet, a Board Certified Veterinary Nutritionist™ may be able to formulate a comparable home-cooked diet recipe. This strategy
can work well for gastrointestinal disease, kidney disease (especially in dogs), fat intolerance, copper-associated hepatopathy, and hepatic encephalopathy but is typically not ideal for struvite urolith dissolution or prevention or calcium oxalate urolith prevention or weight loss. Beware of diet recipes published online or in books – they rarely meet basic nutritional needs and may not appropriately address health concerns.

Notes on specific diseases

**Cardiac disease**

Evidence/Rationale to support use

- Reduced arrhythmias with diet supplemented with DHA and EPA in one dog study
- Many human studies addressing sodium

Important nutrients/properties

- Protein – want moderate to high to support lean body mass, avoid low protein unless comorbidity necessitates it
- DHA + EPA – 180 mg EPA + 120 mg DHA per 10lb body weight shown to have anti-inflammatory and anti-arrhythmic benefits in dogs
- Sodium – avoid high sodium – 0.5-1.0 g/1000 kcal = appropriate for most pets with heart disease

Notes

- Low sodium OTC diets and treats and fish oil supplements are reasonable suggestions for most patients

**Diabetes**

Evidence/Rationale to support use

- A few studies suggest benefit of specific therapeutic low carbohydrate diets versus other diet options in cats
- Dog studies suggest diet composition is less important
- Fiber may help blunt glucose spikes in both species, but potentially more useful in dogs
- Many diabetic cats are obese and may have very low energy requirements once controlled which can make it hard to meet their nutrient needs without diets formulated for weight loss

Important nutrients/properties

- Protein – enough to help maintain lean body mass, no metabolic downside for high protein in otherwise healthy diabetics
- Carbohydrates – complex better than simple, amount is likely more important in cats than dogs, but ideal carbohydrate amount and type is unknown for either species
- Fat – diabetic pets, especially dogs, are predisposed to hyperlipidemia so lower fat diets may be better
- Calories – should be high for underweight animals and low for overweight animals
- Fiber – may be helpful for glucose control, dogs > cats

Alternatives

- Lots of OTC options – therapeutic diets can be used, especially for particularly difficult to manage cases, but many animals can be well-managed using OTC diets
- Home-cooked diets are not ideal options due to less consistency than commercial diets; less ideal for weight loss

**Food allergies**

- Studies have shown benefits of limited antigen and hydrolyzed diets for dogs with skin disease and confirmed food allergies
- Hydrolyzed proteins are only available in therapeutic diets
- OTC “limited antigen” diets often have numerous potential antigens and have been shown in several studies to be frequently contaminated with other proteins

Important nutrients/properties
Hydrolyzed proteins – hydrolyzing proteins reduces allergenicity greatly and the smaller the peptides, potentially the lower the risk

Novel ingredients – if not hydrolyzed, both protein and carbohydrate diets should be novel to the pet

Should be manufactured on a “clean” line in factory to avoid contamination with other ingredients

Alternatives

- No appropriate OTC alternatives for a food trial – hydrolyzed are not available OTC and risk of contamination of novel limited protein OTC diets is quite high
- Home-cooked novel protein diets can be used
- May be able to transition to OTC diets once food allergies confirmed and allergens identified or if food allergies are ruled out

Note

- Salivary and blood allergy tests should not be used to choose diets as they are unreliable
- Consider stressing temporary nature of therapeutic diet – “we are doing this as a diagnostic test” to help get owners on board
- Stress quality control of therapeutic diets versus likelihood of contamination of OTC diet and poor likelihood that OTC can confirm or rule out an allergy

**Gastrointestinal issues (acute or chronic vomiting, diarrhea, flatulence)**

Evidence/Rationale to support use

- Studies show that hydrolyzed diets can be effective for dogs with GI signs from food allergies
- Digestibility and total dietary fiber data is rarely available for OTC diets
- Studies looking at effect of some of these diets on microbiome
- Better control of quality and ingredients than many OTC diets, especially for limited ingredient diets

Important nutrients/properties

- Fat – lower fat diets may reduce vomiting, address potential for pancreatitis while higher fat diets may allow for higher caloric intake in animals with GI compromise
- Digestibility/Fiber – high quality ingredients and studies to assess digestibility, high digestibility or “low residue” = more digested so less becomes stool, fiber types appropriate to slow transit time (diarrhea) and support microbiome, thorough quantification of types and amounts of fiber
- Ingredients – affects digestibility, can be important in the case of limited ingredient or hydrolyzed diets to diagnose food allergy or intolerance
- “Functional foods” – many contain ingredients like colostrum, prebiotic fibers, natural anti-inflammatory compounds, etc

Alternatives

- Home-cooked highly digestible, low fat, and/or limited antigen
- Trial and error with commercial OTC diets if food allergy is not suspected – likely will not be able to obtain digestibility or total dietary fiber data

Notes

- May only be temporary – once problem is under control, may be able to transition to OTC option

**Kidney disease**

Evidence/Rationale to support use

- Therapeutic renal diets shown to double lifespan in dogs and cats compared with maintenance diets
- Diets must contain at least 1.0 g/1000 kcal (dog) and 1.25 g/1000 kcal (cat) of phosphorus to be sold OTC (average ~3.0 g/1000 kcal). Therapeutic renal diets range from ~ 0.45-1.25 g/1000 kcal phosphorus (i.e. there are no OTC substitutes that aren’t too high in phosphorus for most animals with kidney disease)
• Most OTC dog diets have more protein (often as much as 2-3x) than is considered appropriate for stage 3-4 disease. Cat diets more variable as are recommendations for the amount of protein for cats with kidney disease

Important nutrients/properties
• Phosphorus – follow IRIS Treatment recommendations by stage, adjust diet to keep within phosphorus recommendations (extrapolate stage 2 recs to stage 1). Typically this means <2-2.5 g/1000 kcal for stage 1-2, and <1 g/1000 kcal for stage 2+ to 4
• Protein – avoid high protein in all stages, reduce protein below maintenance levels for stage 3&4, earlier if proteinuric
• Potassium – dogs often hyperkalemic, especially if treated with ACE-I, cats hypo – may be able to adjust with diet
• Sodium – avoid diets > 1 g/1000 kcal to reduce the risk of systemic or renal hypertension

Alternatives
• Home-cooked diets for dogs, but cats unlikely to eat them if not already accustomed to eating human foods and higher fat or carb diet
• For stages 1-2, there may be OTC options with lower phosphorus

Liver Disease

Evidence/Rationale to support use
• Therapeutic liver diets are lowest copper diets available – they contain about half the minimum amount of copper allowed to be in OTC diets
• Data show that therapeutic liver diet can be used to reduce copper stores in the liver alone, and in combination with penicillamine chelation therapy
• Data show that plant proteins and dairy are better options for hepatic encephalopathy and that plant proteins, dairy, and egg are lower in purines than meat or organ proteins.
• No specific rationale for using low copper or low protein diets in dogs or cats that do not have copper-associated liver disease or hepatic encephalopathy

Important nutrients/properties
• Protein type and amount – only relevant for pets at risk for hepatic encephalopathy or urate stones. For these pets, feed as much high quality egg, dairy, or plant-based protein as is tolerated. No indication for modification of protein amounts or types unless protein intolerance is documented or expected due to severity of liver dysfunction
• Copper – low copper diets are only of benefit if the liver copper levels are excessive
• Antioxidants – vitamin E and C are often recommended and seem like a good idea, but no data to support appropriate doses

Notes:
• Only available for dogs!
• Most dogs with copper-associated liver disease do not need low protein diets – consider home-cooked or low-copper protein supplementation on top of a liver diet
• Most dogs with hepatic encephalopathy do not need low copper diets – consider copper supplementation up to minimum recommendations
• Increases in liver enzymes without evidence of significant liver dysfunction or copper accumulation is not an indication for low protein or low copper diet

Obesity

Evidence/Rationale to support use
• Studies have demonstrated the potential for nutrient inadequacy with caloric restriction of maintenance diets
Maintenance diets are formulated to provide adequate essential nutrients only when fed at recommended feeding amounts, so feeding less than recommended amounts for ideal body weight can result in nutrient levels below recommendations.

Some data to support better satiety with therapeutic diets versus maintenance-type diets.

Data support healthy weight loss of 1-2% per week with therapeutic diets.

**Important nutrients/properties**
- High nutrient to calorie ratio – allows for calorie restriction without nutrient restriction
- Low calorie density – allows for larger volume with less calories
- Protein – need higher protein to compensate for lower calorie intake and to help preserve lean body mass
- Fiber – added fiber may improve satiety and dilute out calories

**Alternatives**
- Small number of OTC diets formulated for active weight loss – e.g. Science Diet Perfect Weight, Royal Canin Feline Ultra Light, Natural Balance Fat Dog
- Lower calorie OTC (< 300 kcal/cup dogs, < 350 kcal/cup cats) if overfeeding was more of a problem than low energy needs (i.e. if total calories being fed much higher than expected needs and so reduction is still within manufacturer’s recommendations), may also be an option if only slightly overweight as less caloric restriction may be needed.
  - Note protein – many low calorie OTC diets are quite low in protein
- Home-cooked diet not a good option due to logistics of frequent reformulation/amount adjustment during weight loss period

**Notes:**
- If ideal weight loss diet cannot be fed, reduce rate of weight loss to no more than 0.5% per week to minimize the degree of caloric restriction
- Can emphasize that goal is to get pet to ideal weight, may be able to use a maintenance diet at that point if energy needs aren’t too low – so could be only temporary to accomplish goal of weight loss.

**Pancreatitis/fat intolerance/hyperlipidemia**

**Evidence/Rationale to support use**
- Presence of fat in duodenum stimulates cholecystokinin, which in turn stimulates zymogen secretion. Theoretically, if zymogens are being activated within pancreas, this could worsen pancreatitis
- Dietary triglyceride is a big component of the total triglyceride in the blood of hyperlipidemic dogs
- Lowest fat therapeutic diets are about 20% lower in fat than lowest available OTC diets, 50% the fat of average OTC dry, and 15-30% the fat of OTC canned
- Omega-3 fatty acids shown to decrease blood lipids in humans and other animals, but not dogs (no data)

**Important nutrients/properties**
- Fat – for acute pancreatitis, author looks for < 2.0 g/1000 kcal, for other conditions, based on clinical signs, lab values (triglycerides) and previous dietary fat
- Fish oil – shown to decrease lipids in people, rodents, birds, ideal dose in dogs is unknown, may have anti-inflammatory effect for pancreatitis, but also increases overall dietary fat if added as a supplement
- Fiber – may have some benefits for hyperlipidemia

**Alternatives**
- Depending on severity of condition, low fat OTC diets (< 3.0 g/1000 kcal fat) – consider diets marketed as low fat or low calorie
- Home-cooked diet options
Uroliths

Evidence/Rationale to support use
- Studies show ability to dissolve struvite, RSS (relative super saturation) values appropriate for stone prevention for calcium oxalate
- Basically impossible to mimic all properties of these diets in OTC diet

Important nutrients/properties
- Struvite – moderate protein, phosphorus, magnesium, urine acidifying < 6.2, high moisture or designed to increase water consumption (high sodium), important for dissolution in dogs and cats, prevention in cats
- Calcium oxalate – moderate calcium, low oxalate, moderate phosphorus, +/- alkalinizing urine, high moisture or designed to increase water consumption (high sodium)
- Urate – low purine (low in meat proteins, substitute dairy, egg, soy) but not necessarily low protein, urine alkalinizing, high moisture or designed to increase water consumption (high sodium)
- Cystine – low but adequate methionine +cysteine, urine alkalinizing, high moisture or designed to increase water consumption (high sodium) – this is VERY hard to find for cats

Alternatives
- Struvite – acidifying canned OTC? Moisture and pH alone may be enough
- Calcium oxalate – not recommended, but high moisture, moderate calcium and oxalate OTC or home-cooked could be tried, especially for dogs
- Urate – home-cooked for dogs, vegetarian commercial diets
- Cystine – home-cooked for dogs, vegetarian commercial diets

Note:
- As struvite in dogs is nearly always associated with UTI, prevention diets are rarely needed. In cases of chronic UTI, they can be tried, but may not prevent recurrence
Renal disease is one of the medical conditions in veterinary medicine that is most rewarding to treat with nutritional modification. Research in both dogs and cats has demonstrated that specially formulated diets for renal disease help prevent uremic episodes, slow disease progression, and can double survival time.\textsuperscript{1,2} Dietary modifications for chronic kidney disease (CKD) are designed to help mitigate many of the metabolic changes that occur secondary to decreased renal function. Most “renal diets” are restricted in phosphorus, reduced in protein and sodium, +/- supplemented with potassium, alkalinizing, and enriched with B-vitamins and often omega-3 fatty acids. New diet options have made it easier to keep pets with renal disease eating well later into the disease process.

**Phosphorus**

The most critical nutritional modification in classical tubular-based renal disease is phosphorus restriction. Multiple well-designed studies have shown that controlling blood phosphorus concentration through dietary modification slows the progression of CKD. Reduced glomerular filtration rate (GFR) in CKD leads to decreased renal excretion of phosphorus. Hyperphosphatemia in turn leads to increased secretion of parathyroid hormone, leading to increased release of calcium and phosphorus from bone. This combination can cause mineralization of soft tissue, advances renal damage, and may lead to marked bone loss (“rubber jaw”).

The International Renal Interest Society (IRIS) has published guidelines on phosphorus management for renal patients at www.iris-kidney.com/guidelines/recommendations.html. The target serum phosphorous concentrations recommended by IRIS are all in the lower to middle range of most laboratory reference intervals and so pets with “high” phosphorus relative to the IRIS guidelines may still be within the lab reference interval. This seeming discrepancy is because increased concentrations of parathyroid hormone can occur when serum phosphorus is still within the reference intervals\textsuperscript{3}.

Commercial therapeutic renal diets range from about 50 – 120 and 80 -130 mg phosphorus per 100 kcal in dog and cat diets, respectively. The Association of American Feed Control Officials (AAFCO) minimums for dog and cat maintenance are 100 and 125 mg/100 kcal phosphorus, respectively, but it can be a challenge to find over-the-counter diets with less than 250 mg/100 kcal and some diets may be as high as 400-500 mg/100 kcal.

As renal disease advances, it often becomes impossible to keep phosphorus concentrations within the IRIS guidelines using diet alone. In these cases, phosphate binders should be used in addition to the lowest phosphorus diet that is appropriate for the patient.

**Protein**

Reduction of dietary protein is probably the best known and most controversial nutritional modification for patients with renal disease. As many publications point out, there is no evidence that high protein diets are harmful to the kidneys per se. However, many of the toxins that build up in the bloodstream when GFR is markedly compromised by advanced tuberulointerstitial disease (stage 3-4) are nitrogenous compounds. Reducing dietary protein can improve quality of life in these patients by reducing the effects of these toxins on other systems. Additionally, the limiting factor in phosphorus restriction in diets is often the animal protein content as most meats are high in phosphorus. This controversy has become particularly heated for cats and several higher protein but lower phosphorus “early renal” diets have recently been introduced to allow for higher protein without compromising phosphorus. Whether these diets will lead to better outcomes (such as improved muscle mass) in cats with kidney disease remains to be seen.

It is best to determine the appropriate degree of protein reduction necessary based on the individual patient’s physical exam findings, laboratory values and the need to balance protein intake with adequate...
phosphorus restriction and other nutritional modifications. Protein reduction, when appropriate, should be
done by maximizing the quality of the protein to ensure that the physiologic requirements for protein are
being met without adding excess amounts that will quickly be metabolized into uremic toxins.

In contrast to the situation for early CKD without proteinuria, for animals with evidence of a significant
protein losing nephropathy that are non-azotemic, protein reduction is also important for slowing disease
progression. It has been shown in many species that increasing dietary protein exacerbates glomerular
protein loss. Albumin is toxic to the tubules, leading to accelerated degradation of the entire kidney.
Therefore, although it may seem counterintuitive, many dog with PLN respond to lower dietary protein
with lower urine protein: creatinine ratios (UPC) and higher serum albumin.

Unfortunately, there is no known dose-response data to serve as a guideline for what amount of protein
should be used. For dogs, it is likely appropriate to switch to a commercial renal diet even if azotemia is
not present. Less is know about proteinuria in cats. For dogs or cats that were consuming particularly
high protein/phosphorus diets (such as many grain free and low carbohydrate diets, raw diets and/or lots
of meat-based treats) at diagnosis, reduction of protein and phosphorus down to around AAFCO
minimums [~18 and 23% protein calories (5.1 and 6.5 g/100 kcal) and 140 and 125 mg/100 kcal
phosphorus, respectively for dogs and cats] may be a good initial step. Further dietary modification can
then be based on laboratory and clinical response.

Sodium
The vast majority of commercial pet foods contain sodium well in excess of physiological requirements.
Due to mainly theoretical concerns regarding blood pressure and water balance, excess sodium is
generally avoided in diets for patients with renal disease. The sodium concentration in all commercial
renal diets for both dogs and cats is well above AAFCO minimums, but less than the sodium
concentrations of most OTC diets (usually < 120 mg/100 kcal.

Potassium
Serum potassium values can vary dramatically both among patients and between dogs and cats. Dogs
with CKD are more likely to have hyperkalemia, especially those on ACE-inhibitors for glomerular
disease, while cats with CKD are more likely to have hypokalemia. Commercial renal diets also vary in
their potassium concentrations, with feline diets generally being higher than canine diets. The wide range
of potassium concentrations in canine diets allows for selection of the diet most appropriate for the
individual patient.

Acid-Base
One of the many roles of the kidney is acid-base balance. The kidney regulates the excretion of hydrogen
ions and bicarbonate regeneration; consequently, animals with renal impairment often become acidic.
Commercial renal diets are designed to be relatively alkalinizing to help counteract these changes. In
cats, this is significantly different from OTC maintenance diets which are generally acidifying due to both
ingredient composition and specific intention to help prevent struvite-related urinary problems.

Omega-3 Fatty Acids
Research in dogs has shown potential reno-protective effects of supplementation with long chain omega-
3 fatty acids eicosapentaenoic acid; EPA; and docosahexaenoic acid; DHA. However, there is some
conflicting evidence and a clear dose response has not been determined for dogs. There are fewer data
for cats – one study showed improved outcome in cats that ate a renal diet that included omega-3 fatty
acids versus those that did not. Flax, which is a good source of the shorter chain omega-3 fatty acid
alpha-linoleic acid (ALA), has not been investigated to see whether it is beneficial for kidney disease in
dogs. As the endogenous conversion of ALA to DHA and EPA is poor in dogs and essentially non-
existent in cats, it should only be used as a last resort when supplementation with DHA and EPA is not
feasible (e.g. gastrointestinal upset).
Many commercial renal diets already contain supplemental fish oil, but the amounts vary. One recommended dose is 140 mg/kg\(^{0.75}\) DHA plus EPA. This amount can be supplemented on top of a commercial diet that is not already supplemented, or the total intake from the diet alone can be calculated and fish oil (or krill or algal oil) added to attain the final desired dose. For many patients, liquid fish oil may also be a palatability enhancer.

**When should a renal diet be started?**

In the absence of proteinuria, initial dietary modifications for early asymptomatic CKD (IRIS Stage 1 or when CKD is suspected but not confirmed) should be geared towards reducing phosphorus/keeping it on the lower end of the normal range. There are a handful of commercial diets (including some OTC) with phosphorus between 140 and 200 mg/100 kcal and moderate protein which can be considered. These diets will be higher in protein than renal diets and may not have the other modifications that have been previously discussed. There are also several “early renal” diets designed for cats, although none currently marketed that way for dogs. Once CKD progresses to stage 3 or if significant proteinuria or relative hyperphosphatemia (based on IRIS treatment guidelines) is evident, most patients should be fed only a renal diet.

**What about concurrent diseases?**

Common concurrent disease processes that can make it difficult to find an appropriate renal diet include food allergies and fat intolerance (e.g. hyperlipidemia, history of pancreatitis). For pets with fat intolerance, you should look for the lowest phosphorus low fat diets, or the lowest fat renal diets. For patients with early CKD and confirmed or suspected food allergies/intolerances, you may be able to be feed a commercial lower protein and phosphorus limited-antigen diet (you will need to compare the nutrient levels of the limited antigen diets that are appropriate for the pet to find the lowest phosphorus).

For suspected food allergies or intolerances in later stage CKD, the best option is generally to use the one hydrolyzed renal diet available (Royal Canin Veterinary Diet Multifunction Hydrolyzed + Renal) or trial a few of the renal diets to see if they are tolerated. For confirmed allergies to ingredients that cannot be avoided in the renal diets or for later stage kidney disease and significant fat intolerance, home-cooked diet formulations may be required.

**Juvenile Renal Disease**

Commercial renal diets are too low in phosphorus, protein, and sometimes calcium as well as potentially other nutrients to support optimal growth and development. Additionally, physiologically increased phosphorus, less muscle development, and lower hematocrits in young dogs and cats can make it harder to assess stage and progression than in adult animals. Commercial renal diets should especially be avoided in puppies or kittens less than six months of age. Each case should be approached on an individual basis and a referral to a Board Certified Veterinary Nutritionist is highly recommended. A renal panel with phosphorous should generally be monitored monthly with adjustments in diet until at least 12 months of age to attempt to provide adequate nutrients for growth while not encouraging renal disease progression. After 6 months of age in most breeds, an increase in phosphorus is likely a sign of disease progression as the growth-related portion should start to steadily decline as they reach skeletal maturity.

**What if the pet will not eat a renal diet?**

A slow transition from the previous diet is recommended whenever possible. Low protein palatability enhancers such as fish oil, homemade low sodium meat broths, honey, pancake syrup, applesauce and some human enteral products (e.g. Vanilla Ensure for dogs) can be added to the diet to increase interest. Meats and other foods high in protein, phosphorus, and sodium should be avoided as they have the potential to negate the benefits of the diet and actually make the patient feel worse in the short term.

It is common for pets with later stage disease to have cyclical appetites where they may not be interested in eating the same food every day. Rotating between several appropriate diets may help overcome this
issue. Appetite stimulant drugs can be used but may not result in consistent consumption of enough food to meet energy requirements.

Home-cooked diets may be more palatable to dogs with CKD than commercial diets. However, the vast majority of the recipes in books and online are unbalanced and may not be appropriate for renal patients despite assertions to the contrary. Custom recipes should be obtained from a Board Certified Veterinary Nutritionist (www.acvn.org). Some balanced (but not as customized) recipes for home-cooked renal diets can also be obtained by veterinarians for their clients from www.BalanceIT.com.

Unlike in dogs, appropriate home-cooked diets are rarely more appealing to cats than commercial diets. Palatability enhancers such as homemade, low sodium meat broths, fish oil and animal fats can be used to encourage intake.

**Assisted Feeding**

Assisted (tube) feeding should be considered for patients with CKD that are not able to maintain an appropriate body condition via voluntary intake of an “appropriate” diet for their stage of disease. Esophagostomy (E) tubes are the most commonly used and can make a huge difference in the quality of life for both owner and pet. An E-tube allows for an appropriate diet (i.e. a blenderized canned renal diet) to be fed in appropriate amounts to maintain body weight and often reduces the stress surrounding mealtimes for both pet and owner. Many medications as well as oral fluids can be administered via the tube, which can also enhance quality of life for the entire family.

Feeding tubes are best placed prior to the animal becoming significantly debilitated, rather than as a last-ditch attempt to prolong the life of an emaciated, anorexic, severely uremic animal. For this reason, consider discussing feeding tubes as an option with clients long before the pet requires it, even at the first or second visit after the renal disease is diagnosed.

Clients can be advised to consider that at some point a feeding tube may be appropriate and encouraged to decide as a family how they would handle the situation if it arose. It may be helpful to keep a list of good clients who have successfully used feeding tubes and are willing to talk to other clients about their experiences.

**References**

They want to feed what?! Alternative diets
Callin R. Heinze, MS, VMD, DACVN

Popular human diet trends such as gluten-free, ketogenic, paleo, vegetarian, vegan, and natural are being more commonly applied to pets and have been embraced by pet food manufacturers. Many pet owners also desire to feed their dogs or cats more like their wild relatives and feel strongly that raw, “evolutionary” diets offer superior nutrition. It is important for veterinarians to understand the pros and cons of various alternative feeding strategies so that they can appropriately advise their clients.

Vegetarian/Vegan
Vegetarian and vegan lifestyles are becoming more mainstream. These lifestyles are often adopted for ethical reasons, but many people also choose to eat this way for both perceived and demonstrated health benefits. Both of these motivations can drive pet owners to want to feed their pets in a similar manner. Diets based on dairy or egg protein, while uncommonly available commercially, can be nutritionally complete and meet the needs of both dogs and cats if properly supplemented, just like meat-based diets.

Vegan diets add an extra layer of complication and must be treated with additional care. Many nutritional supplements commonly used in pet foods are derived from animals (such as vitamin D3 and most omega-3 fatty acids). Therefore, while vegan diets generally require careful supplementation, the options available to do so are more limited and may not be available in ideal forms. Meat-free diets for both dogs and cats typically require supplementation with carnitine and taurine and possibly methionine as well as methionine is typically limiting in plant proteins and is used to make both taurine and carnitine. Deficiencies have been reported in dogs eating low protein or predominately plant-protein based diets and can be clinically important (e.g. dilated cardiomyopathy).

Dogs, as omnivores, may adapt better to vegan diets than cats and often appear to do well on carefully designed vegan diets. However, long-term studies of vegan (or vegetarian) dog diets have not been performed. There are an increasing number of vegan or vegetarian dog foods on the market, including some veterinary therapeutic diets used to treat certain health conditions (e.g. food allergies, cystine and urate uroliths, hepatic encephalopathy).

Cats, by virtue of their status as obligate carnivores, have high protein and amino acid needs relative to other domestic animals and also require a number of nutrients that are not readily obtained from plant sources, such as arachidonic acid, preformed vitamin A rather than beta-carotene, and taurine. Vitamin D3 from animals is more bioavailable than D2 from plants for cats. For these reasons, vegan diets, whether commercial or home-made, are not recommended for cats and serious nutritional concerns have been demonstrated for many commercial vegan cat diets and pretty much all home-prepared vegan cat diets.

Quality control of commercial vegan and vegetarian pet foods, especially over-the-counter options, has been questioned by two recent studies that found inadequate levels of protein and/or amino acids, labels that did not conform to Association of American Feed Control Officials (AAFCO) regulations, and presumed contamination with animal DNA in a substantial percentage of commercial vegan and vegetarian pet foods.1,2

Vegan homemade diets for dogs and cats are almost always deficient in protein and amino acids as well as all of the other essential nutrients that are commonly deficient in meat-containing home-prepared diets3,4 developed by pet owners, or found on the internet and in pet cookbooks.

Owners determined to feed their dogs a vegan diet should be steered towards veterinary therapeutic diets that are appropriate for long-term feeding of systemically healthy dogs as these diets typically undergo much more extensive testing than over-the-counter diets. Alternatively, these clients can be referred to a board-certified nutritionist for a balanced home-cooked diet recipe. These owners should be advised that long-term health effects are not known.

Cat owners should be counseled that this diet strategy is not in the cat’s best interest and is likely to lead to serious health concerns.

Raw Diets
Commercial raw diets are one of the fastest growing market sectors of the pet food industry. The ready availability of glowing testimonials from other pet owners online, as well as recommendations from pet store employees and sometimes veterinarians have increased the number of clients who may consider feeding these diets. More and more companies, many of which never previously sold raw foods, are now offering hybrid diets - mixtures of dehydrated raw and kibble or raw-coated kibble, which can be hard to distinguish from cooked kibble diets. Many pet owners also do not realize that freeze-dried raw diets are still considered to be raw with regards to pathogen contamination.

Nutritional adequacy is a concern with both commercial (mainly the all-raw ones rather than the kibble hybrids) and home-prepared raw diets (often referred to as ‘BARF’ – Bones And Raw Flesh or Biologically Appropriate Raw Food). For commercial products, it may not always be clear to the consumer whether the diet is actually designed to be complete and balanced and even some of those that are intended to be may have apparent nutritional gaps when subjected to increased scrutiny (be especially wary if no concentrated supplements are listed on the ingredient list and/or the AAFCO nutritional adequacy statement is not clear and properly worded).

Home-prepared raw diets have similar nutritional adequacy issues to those of home-cooked diets. Commercial raw diets have been associated with pancreatitis (they are usually very high in fat) and both home-made and commercial products have been associated with hyperthyroidism in dogs, as well as constipation, gastrointestinal obstruction and dental fractures.

An additional, significant concern with raw diets is biological contamination. Many producers and promoters of raw diets either deny that their products potentially contain pathogenic bacteria such as Salmonella or E.coli or state in their marketing materials that animals do not get sick from these bacteria. Contamination of meat intended for human consumption has been a serious concern for many years with large recalls every couple of years becoming commonplace.

Similarly, major raw pet food producers have recalled numerous raw pet diets due to contamination with Salmonella and other pathogens over the years (27 recalls for diets alone in 2018). It is currently both reasonable and safest to assume that all raw meat is contaminated, whether intended for human or animal consumption.

While dogs and cats may be less sensitive to food borne illness than people, there are numerous documented cases of severe and even fatal disease attributed to bacterial contamination of foods.\(^5\)\(^,\)\(^7\) Consumption of raw chicken has been linked to acute polyradiculoneuritis in a report out of Australia\(^8\) and a recent case series reported a number of pet cats who were infected with M.bovis from their raw diet.\(^7\)

Although some diet manufacturers have started using high pressure processing (HPP) or other non-traditional processes to kill bacteria in raw diets, these techniques are rather new to the pet food industry and efficacy is still uncertain (not much published). There have been recalls of raw diets treated with HPP although it is not always clear which diets are treated and which are not.

Raw food-fed pets have been shown to shed viable pathogenic bacteria\(^9\)-\(^11\), sometimes asymptptomatically, putting all people in the household at risk. Of particular concern are households with young children, the elderly, or people with weak or suppressed immune systems (such as HIV or cancer patients). The CDC and FDA both have put out strong statements discouraging the use of raw meat diets for pets due to concerns for human health. Several raw pet food recalls in the past few years have been instituted because of human illness rather than pet illness.

In summary, there is so far no scientific evidence to substantiate the majority of the anecdotal benefits of feeding raw versus cooked diets. However, there is clear evidence to suggest that these diets and raw meat treats can cause harm to both pets and their human companions. A comprehensive, although somewhat dated, review outlines what is known about raw diets – both the pros and the cons.\(^12\)

**Natural/Holistic diets**

AAFCO defines natural as “a feed or ingredient derived solely from plant, animal or mined sources, either in its unprocessed state or having been subject to physical processing, heat processing, rendering, purification, extraction, hydrolysis, enzymolysis or fermentation, but not having been produced by or subject to a chemically synthetic process...”. To label a pet food as “natural” requires that no synthetic compounds of any kind be
included. However, phrasing such as “natural diet with added vitamins” can be used to acknowledge the fact that many diets contain natural ingredients but are supplemented with synthetic vitamins, amino acids or mineral complexes.

The current definition of natural gives no information about ingredient or product quality. It is important to keep in mind that natural sources of many vitamins, minerals and amino acids used in commercial pet foods are not always practical and many synthetic sources are metabolically indistinguishable from natural forms.

Some commercial diets attempt to provide for all required nutrients using only whole foods. This approach, while emotionally appealing, presents several problems. First, these diets generally contain large numbers of ingredients sourced from many different vendors. Vitamin and mineral contents of ingredients such as fruits, vegetables and kelp (a commonly used iodine source) tend to vary, sometimes substantially, between sources and even by season. Ingredient analysis can be quite expensive, so individual shipments of ingredients may not be thoroughly screened, especially by smaller companies that purchase many different ingredients and lack the ability to do in-house analysis.

Whole food ingredients never provide only one nutrient, thus adding enough of an ingredient to meet one nutrient requirement can result in an excess of another nutrient. Similarly, some nutrients, such as taurine, become less bioavailable with cooking (others become more available); it may be difficult or impossible to provide adequate amounts of these nutrients from whole food sources.

Artificial preservatives are becoming uncommon in pet foods, mostly due to public opinion rather than any documented adverse effects. The most common “natural” preservatives used in pet foods are the antioxidants vitamin C, vitamin E (mixed tocopherols), and rosemary extract. These preservatives may be less effective than their synthetic counterparts and necessitate more attention be paid to setting appropriate expiration dates, proper storage and monitoring for spoilage.

The term ‘holistic” has no AAFCO definition and the term can be applied to any pet food product.

References
Too Many Cooks in the Kitchen: What Every Vet Needs to Know about Home-cooked Diets

Cailin Heinze, VMD, MS, DACVN

While still representing a small proportion of pet owners, home-cooking is becoming more popular, especially in the wake of some high profile pet food recalls in the past 15 years. Many pet owners opt to cook for their pets due to distrust of commercial pet food, a desire to have more control over ingredients, or because they feel that their pet prefers home-cooked meals. A smaller segment of pet owners end up cooking for their pets because medical conditions limit the available commercial options.

Nutritional Adequacy

While in theory a home-cooked diet can be a healthy alternative to commercial diets, there is currently no evidence that home-cooked diets have any nutritional benefits over commercial diets for healthy pets and the risks of nutrient deficiencies and other imbalances are much increased when a home-cooked diet is fed.

Many owners simply chose ingredients that their pet likes and mix them together with or without a vitamin or mineral supplement. These diets are invariably deficient in essential nutrients, even with added popular human or pet supplements, neither of which are designed to fill in the gaps in home-cooked pet diets. Other pet owners seek out published recipes for home-cooked diets from lay books, magazines, newsgroups, and on the internet. With few exceptions, the diets resulting from these recipes are deficient in multiple essential nutrients and contain vague preparation and supplementation instructions.1,2

Home-cooked diets frequently deficient in calcium, zinc, copper, vitamin E, vitamin D and choline. Vitamin D excess can also be seen in salmon-based diets and products that use human multivitamins. Meat-free home-cooked diets are usually deficient in total protein as well as amino acids like methionine.

Home-cooked diets are of particular concern for growing animals as these diets are commonly deficient in nutrients such as calcium that are essential for normal growth. As home-cooked diets become more popular, so does severe nutrient-related health problems in kittens and puppies. Many Board Certified Veterinary Nutritionist™ will only formulate home-cooked diets for puppies and kittens in situations of medical necessity when no commercial diet options exist.

Even with a well-designed recipe, ingredient variation and modifications (both inadvertent and intentional) to the recipe by pet owners can lead to imbalances or nutrient deficiency or toxicity. Two surveys of pet owners who purchased recipes from university nutrition services have reported that large proportions of pet owners modify recipes, often in ways that alter their nutritional profile substantially.3,4

Assessing Home-cooked Diet Recipes

When a client reports that they are feeding a home-prepared diet, it is important to have them provide a copy of the recipe or get a detailed accounting of what they are feeding. Amounts of various ingredients (ideally in weight, not volume), specific cuts of meat, how the ingredients are prepared (e.g. pan-browned 85% lean ground beef, boiled chicken breast), and any and all supplements should be easily accessible in the pet’s medical record.

To get a good idea of the levels of all essential nutrients in a homemade diet, a computer analysis or a chemical analysis of the recipe is required. As testing a diet for all AAFCO nutrients typically runs around $2-3k per sample, chemical testing is rarely done except for specific nutrients, often in a situation where a health issue has been recognized. Computer analysis is easier and considerably less expensive, but has limitations because owners may be feeding ingredients or preparing them in a way that is not available in standard food nutrient databases (e.g. USDA food nutrient database).

However, even without a computer program, you can do a quick analysis in your office that will usually identify some of the more common and serious nutritional adequacy concerns.
Quick checks:

- **Clear recipe** – no rotations of ingredients, no vague “2 parts meat to 1 part veggies” or anything else that would mean that two people using the same recipe would make two different diets. The recipe should provide specific measurements, preferably in weight for meats, and specific cooking directions. Rotation of ingredients rarely fixes nutritional inadequacies as the most commonly used ingredients have similar nutrient contents and limiting nutrients. There should also be guidelines on how much to feed/how many calories the recipe provides.

- **Animal protein source** – meat-free homemade diets are usually protein-deficient, but pet owners typically err on the high side for protein if they are using meat.

- **Essential fatty acid source** – common ingredients such as beef, pork, lamb, and fish will require a source of omega-6 fatty acids. This is typically going to need to be corn or canola oil. Olive oil, coconut oil, and flaxseed oil will not provide adequate amounts. Diets with dark meat poultry or significant calories coming from quinoa or oats may not need additional supplementation of omega-6s. Fish oil can be a good addition to a home-cooked diet, but is not strictly essential and will not replace a plant oil as a source of omega-6s.

- **Calcium source** – should be based on the calories fed, not on the pounds of meat. Can be inorganic (e.g. calcium carbonate) or organic (bone meal). Most recipes do not have enough calcium, even when a supplement is added. No supplement at all will definitely never be nutritionally adequate.

- **A vitamin/mineral supplement designed EXCLUSIVELY and ONLY for supplementing home-cooked pet foods.** Ex. Balance IT supplements (holds the current patent for an all-in-one supplement for home-cooked diets). A product marketed for use with commercial pet foods will not be adequate to balance a home-cooked diet. It is possible to balance a diet using a multitude (6-9 different products) of human supplements, but it is difficult and requires a good knowledge of available products as well as diet formulation software – it is not possible to by chance select an appropriate combination of supplements. Many human vitamin/mineral supplements contain excessive vitamin D for dogs, for example.

If the client is not using a recipe, you can assume that the diet is not nutritionally balanced. Even if the recipe at a glance passes the criteria above, it is worth having the diet analyzed unless it was recently obtained from a reputable source (such as a Board Certified Veterinary Nutritionist™) as the ingredients may not be in the proper proportions.

**Situations where you should consider recommending a home-cooked diet**

Home-cooked diets can be valuable for pets with certain health concerns such as:

- Kidney disease – homemade diets may be more palatable for some dogs who won’t eat commercial renal diets well
- For suspected adverse food reaction when a therapeutic hydrolyzed or limited protein diet elimination trial has failed
- For severe hyperlipidemia or lymphangiectasia that is not responding to the lowest fat therapeutic diet
- For chronic gastrointestinal disease that has not responded to a hydrolyzed or novel limited protein commercial diet or to a therapeutic gastrointestinal diet
- Multiple health concerns where commercial diets are not available or are not working – pancreatitis or hyperlipidemia plus renal disease or adverse food reaction, renal disease plus gastrointestinal disease, desire for low copper with high protein, etc

In some cases it may be appropriate to try an unbalanced home-cooked diet for a few weeks in an adult pet prior to adding in the supplements, but it should be made clear to the pet owner that long-term harm will come if the diet is not properly balanced but continues to be fed.

For medical cases, simple balanced recipes can be obtained from BalanceIT.com with a vet account, or you can refer your clients to a Board Certified Veterinary Nutritionist™ (www.acvn.org) if the case is more complicated or you or they desire more comprehensive nutritional management.
References

Struvite and calcium oxalate are by far the most common types of uroliths in cats and dogs. Less commonly seen are urate and cysteine, although the prevalence within specific breeds may be high.

The overreaching goals of nutritional management of urolithiasis, regardless of stone type, are to:

- Decrease urine specific gravity,
- Decrease dietary stone precursors,
- Produce appropriate urine pH,
- Increase the concentration of stone inhibitors.

These strategies decrease the risk of stone formation and may allow for dissolution, depending on stone type. When in doubt, urine dilution is safe and useful for all stone types.

**Therapeutic Urinary Diets**

Unlike over the counter diets that make urinary claims, therapeutic “stone diets” are tested using technology called “Relative Supersaturation (RSS)” or “Activity Product” or similar. Animals in controlled situations are fed the diet to be tested and all their urine is collected and assayed for the normal things we check in a urinalysis as well as a number of other compounds and then this information is fed into a software program that predicts the likelihood of stone formation. The diets are tested as 100% of calories, so adding in treats and other foods could reduce their efficacy and these diets will not prevent stones in all pets, especially if urine specific gravity is high.

Several therapeutic diet manufacturers are now putting diets for other health conditions through this testing so they can be used in patients that have both stones and other health conditions. Typically, this is indicated by a symbol in the product guides or on the diet labels themselves.

**Struvite – Magnesium Ammonium Phosphate**

**Dissolution:**

Pure struvite stones can usually be dissolved using an acidifying diet (urine pH < 6.2) and by decreasing the urine specific gravity (<1.020 for dogs, 1.025 for cats). USG is critical to encourage dissolution and can be accomplished by increasing moisture (canned diets or adding large amounts of water to dry diets). Increased dietary sodium or severe protein restriction (to cause medullary wash out) are also utilized in some commercial stone dissolution diets. High sodium diets should be avoided in animals with hypertension or liver, heart or renal disease.

All dogs and cats suspected of having infection-induced stones should be placed on appropriate antimicrobial therapy based on culture results during dietary dissolution therapy and should remain on antimicrobials for 2-4 weeks after the stone is no longer visible on radiographs. A therapeutic diet designed for struvite dissolution should be used and the diet should be fed as 90-100% of daily calories during treatment to ensure efficacy.

The risk of urethral obstruction theoretically increases as cystoliths become smaller and are able to leave the bladder and could be life-threatening, so this risk should be communicated to the client. Most struvite stone dissolution diets are also designed to reduce the risk of calcium oxalate crystals and stones and can be fed long-term if required; these diets are usually the most desirable options.

The urine should be cultured midway through the dissolution process and again 5-7 days after discontinuing the antimicrobial. Radiographs should be performed at least monthly. Stones that have not disappeared or gotten smaller after 1 month are either not 100% struvite, or indicative of owner non-compliance.
Contraindications for Dietary Dissolution

Some animals have concurrent health problems that may be contraindications for dietary dissolution such as marked hyperlipidemia and/or a history of recurrent pancreatitis, hypertension and/or heart disease (for the higher sodium diets), or confirmed food allergies to ingredients common to the dissolution diets. Most commercial dissolution diets are moderate to high in fat and/or high in sodium and contain common ingredients.

These diets are also contraindicated in pregnant or growing animals and may not be ideal for clients known for poor compliance or with limited funds as several weeks to months of rechecks, radiographs, antibiotics, and a prescription diet for a large dog may cost more than surgery in the long run at some practices, especially if the urolith ends up requiring later surgical removal after dissolution failure.

Prevention Strategies for Struvite Urolithiasis (sterile or recurrent)

No specific dietary prevention is necessary in dogs with infection-induced struvite – measures to prevent/detect future UTIs in a timely manner should be instituted. For cats, the very rare canine sterile stone former, or when infection cannot be completely eradicated, prevention strategies are similar to those of dissolution although it is critical to select a diet that is designed to be fed long-term.

There are multiple therapeutic diets for both dogs and cats formulated to help prevent struvite urolithiasis; however, canned acidifying OTC diets (some labeled as “urinary” are available for cats and may be helpful, especially if owner finances are tight.

Calcium Oxalate

Calcium oxalate uroliths can be associated with health conditions such as vitamin D toxicity or hypercalcemia and are thought to be caused by hereditary modifications in calcium and oxalate metabolism in many small breed dogs.

Prevention

Calcium oxalate uroliths will not dissolve, unlike struvite, so mechanical removal is necessary (urohydropulsion, cystotomy, lithotripsy). Recurrence is common (up to 50% within 3 years) and dietary therapy is generally one of the main methods of prevention if an underlying medical condition (i.e. hypercalcemia) cannot be identified and addressed.

Nutritional Prevention Strategies for Calcium Oxalate

- Reduce urine specific gravity
- Reduce dietary oxalate – Oxalate can be obtained from the diet or manufactured endogenously through the metabolism of ascorbic acid (vitamin C), glycine, glyoxylate and tryptophan. Oxalate is commonly found in plant ingredients. Common ingredients high in oxalate include soy, spinach, beets, whole grains, and potatoes.
- Ensure adequate calcium intake – High dietary calcium levels can result in increased calcium excretion and could encourage stone formation. Low dietary calcium can actually increase oxalate absorption in the small intestine; calcium will bind oxalate in the intestine and produce an insoluble product that is then excreted in the feces.
- Avoid acidifying diets – Oxalate stones can form at a wide range of pH and will not dissolve regardless of pH although crystals are most soluble in alkaline urine. Acidifying diets may increase the risk of calcium oxalate by inducing metabolic acidosis and subsequent mobilization of calcium and phosphorus from bone. Acidification also contributes to a decrease in citrate excretion, which inhibits crystallization of calcium oxalate in the urine by favoring the formation of calcium citrate. Overall, however, urine pH is much less important than it is for struvite, which is why many dual-purpose struvite and oxalate preventative diets do produce acidic urine pH.
- Avoid vitamin C supplementation – Ascorbic acid is metabolized to oxalic acid before excretion.
• Avoid supplements containing calcium and vitamin D unless needed to balance a home-prepared diet or treat a known deficiency.

• Avoid very high protein diets, treats, and rawhides — Very high protein diets may cause increased oxalate excretion due to the metabolism of excess glycine and tryptophan. High protein diets also tend to be acidifying. Rawhides and “animal part” treats contribute little nutritionally, but are a source of extra glycine and hydroxyproline that is then metabolized to oxalate.

There are a number of veterinary therapeutic diets that are appropriate for long-term feeding for calcium oxalate prevention in dogs. There are also a number of options for cats. Almost all of these diets are formulated for prevention of both struvite and calcium oxalate.

Despite dietary management, recurrence of calcium oxalate stones is quite common. For animals with recurrent stones, or those that cannot be fed a stone prevention diet, supplementation with potassium citrate may reduce risk as citrate can complete with the oxalate for binding to calcium but is much more soluble than oxalate. Potassium citrate may also increase urinary pH somewhat.

Urate

The two main causes for urate urolithiasis are reduced activity of uricase, the enzyme responsible for converting uric acid to allantoin for excretion (i.e. Dalmatians, Bulldogs, Jack Russell Terriers), and liver disease (e.g. portosystemic shunt) resulting in hyperammonemia and hyperuricemia and stone formation. In the case of the latter, managing the underlying disease process is the best way to prevent stone recurrence. Urate stones in cats are usually related to liver disease. For dogs with decreased uricase activity, diet is very important to prevent recurrence.

Uric acid and allantoin are the end products of the degradation of purines. Two common purines are adenosine and guanine, two nucleotides found in DNA and RNA. The cornerstone of management of urate stones are reducing dietary purines and decreasing urine specific gravity. Alkalinization of urine (pH > 7) may also be beneficial.

Because nucleotides are part of DNA and RNA, the goal is to avoid metabolically active tissues that are likely to have more nuclei and thus more DNA. Dairy, egg and plant proteins tend to be lower in purines than meat proteins (especially organ meats) and are ideal sources of proteins for animals with a history of urate crystals or urolithiasis. Diets lower in protein tend to have less purines, but this is not a firm rule and protein source is likely more important than gross amount. Diets with higher amounts of plant ingredients have the added benefit of causing urine alkalinization.

Diets to consider for dogs include egg- and soy-based therapeutic diets made for allergies and liver disease and therapeutic vegetarian diets. While there are a few soy-based diets therapeutic diets designed for cats with allergies, low protein diets made for kidney disease are frequently used for urate stone prevention in cats despite typically containing organ meats. However, renal diets also have other modifications (such as low phosphorus) and pets should be monitored closely if used for urate stones. Pure urate stones can sometimes be dissolved utilizing a combination of diet and allopurinol in dogs but use of allopurinol increases the risk of xanthine stone formation, which must be surgically removed.

Cystine urolithiasis

Cystine uroliths can occur in dogs and occasionally cats that have a defect in the proximal renal tubules that prevent reabsorption of cystine and usually ornithine, lysine and arginine as well. As cystine is the least soluble, it precipitates out forming uroliths. These tubular defects are usually inherited and are seen most commonly in English Bulldogs, Mastiffs, Dachshunds and Newfoundlands, but have been described in other breeds. Some types of cystinuria are androgen-dependent and are resolved by neutering affected male dogs.
Prevention
Nutritional strategies for prevention of cystine uroliths include decreasing urine concentration, minimizing dietary methionine and cysteine, and alkalinizing urine. As methionine is an essential amino acid, care must be taken to make sure that deficiencies are not created. As plant-based proteins tend to be limiting in sulfur-containing amino acids, vegetarian or mostly vegetarian diets may be good options. However, the quality of commercially available vegetarian diets (particularly OTC diets) is extremely variable and often questionable. Reduced protein renal diets are sometimes recommended for cystine urolithiasis in dogs; however, these diets have additional modifications (i.e. mineral alterations) in addition to much decreased protein, making them considerably less appealing as life-long diets.

For cats, a hydrolyzed soy diet can be used but otherwise the options are limited to diets formulated for renal disease with all the caveats listed for dogs.

As dogs utilize methionine and cysteine to make taurine, taurine supplementation (or regular measurement of levels) is recommended for dogs with cystinuria. Cats are dependent on dietary taurine already, so this is less of a concern. Some dogs may also lose carnitine in their urine or not produce as much endogenous carnitine due to decreased dietary methionine; carnitine supplementation can also be considered (carnitine status cannot be reliably determined from blood concentrations).

For dogs that cannot be managed on diet alone, drug therapy (2-MPG and occasionally D-penicillamine) can be used to dissolve or prevent further formation of cystine uroliths.

### Dietary treatment of urinary stones in one table

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<thead>
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<th>Struvite</th>
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<td>Potassium citrate</td>
<td>Allopurinol (watch for xanthine)</td>
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<td>Low oxalate, moderate Ca</td>
<td>Low purines, alkalinize urine</td>
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CANCER CLIFF NOTES FOR CATS: WHAT YOU NEED TO KNOW
Sue Ettinger, DVM, DACVIM (Oncology)
Dr Sue Cancer Vet PLLC, Tarrytown, NY, USA

FELINE HIGH-GRADE LYMPHOMA

What is it: Lymphoma (LSA) is one of the most common feline cancers, reported at 30% of all cancers. It is a systemic disease that requires chemotherapy in almost all cases. It is a collection of cancers arising from the malignant transformation of lymphocytes. In contrast to dogs, feline lymphoma most commonly affects the gastrointestinal (GI) tract. Lymphoma can occur in cats of any age, any sex, any breed. The median age is 11 years.

What I say: Gastrointestinal Lymphoma is a treatable cancer but outcomes for treated cats are less predictable than dogs, but cats tend to tolerate chemotherapy better than dogs. Treated cats live longer, and chemotherapy is generally well-tolerated.

Clinical appearance: Clinical signs include weight loss, vomiting, diarrhea, anorexia or hyporexia, and icterus. 33%. The onset is more rapid – days to weeks. A palpable mass is common. Rarely the cat will present with acute abdomen due to obstruction or perforation. Alimentary lymphoma typically involved the intestines alone or intestines, lymph nodes (LN), and liver. In the GI tract, it can be solitary vs diffuse.

Diagnostics: Early accurate diagnostics and careful staging are keys to proper clinical decision-making. The diagnosis is typically straightforward for high grade LSA, and the diagnosis is typically made with abdominal ultrasound and cytology/histology. Surgery is less commonly needed. The minimum tests required for treatment are cytological confirmation (lymph node or affected organ), CBC, chemistry panel and urinalysis. Additional tests to consider include thoracic radiographs, lymph node histology, urinalysis, bone marrow cytology and phenotyping. These tests are useful and informative, as they provide prognostic factors and a baseline for a patient’s response. Still, we must consider the owner’s financial issues. While it is ideal to perform all the tests, we can also consider each test on a case by case basis and help the owner make an educated decision. We can treat without but review pros and cons with the owner and let owner make educated decision and maybe choose more important tests for that cat.

Prognostic factors: The prognosis and response in cats with lymphoma are more variable than in canine lymphoma. Prognostic factors include anatomic location, achieving a CR, FeLV-status, substage, and a multi-agent protocol (CHOP vs COP?). Factors that are NOT prognostic in cats include stage and immunophenotype, age, weight, gender, and FIV.

Remember, prognostic factors cannot predict an individual’s response, and lymphoma is typically treatable and rewarding to treat for the patient, owner and the veterinarian.

Treatment: I typically recommend a CHOP multi-agent protocol such as the UW 25-week protocol. When using doxorubicin in cats, I use a lower dose (1 mg/kg). Cardiac toxicity is not clinical problem in cats in contrast to dogs, and renal function (BUN, Cr, USG) should be monitored in cats when giving doxorubicin. In dogs, data supports shorter maintenance-free protocol, but there is no data in cats, and some cats may need chronic chemotherapy. An alternative protocol is the COP protocol with reported complete remissions of 50-70%. This is commonly used in used in Europe with similar results to CHOP in 1 study. While the protocol requires less frequent visits, it is a longer 1-year protocol. Other studies support the addition of doxorubicin to COP for durable responses. For single agent options, Lomustine can be given at 50-60 mg/m2 every 4-6 weeks, which is given at a lower dose and less frequently than dogs. Single agent doxorubicin in cats is less successful with complete remission rates of <50%.

If chemotherapy is declined If chemotherapy is declined, another option is single agent steroids. Typical response rates are 50% with duration of 2 to 3 months. Without chemotherapy the prognosis for lymphoma is poor, with MST of 1 month.

Prognosis: Without Treatment: 1 month. With treatment: With prednisone only, typical response duration is 2 to 3 months. Multi-agent CHOP protocols are the most successful. For GI forms, the prognosis is overall extremely variable. For high grade GI lymphoma, response rates are 50-75%, median remission duration is 4-6 months, and expected survival is 6-8 months. 15-25% can live 1-2 years.

FELINE MAMMARY CANCERS (FMC)

What is it: Feline mammary tumors are the third most common tumor (after skin tumors and lymphoma), representing about 20% of tumors in the female cat. Mammary tumors in cats are generally malignant (80-90%), with adenocarcinomas most common, and metastasis is common (80%), typically to lungs, pleura and lymph nodes.
What I say: FMC is similar to breast cancer in women in that it needs to be treated locally with aggressive surgery and followed with adjunct chemotherapy due to the high metastatic rates. Like dogs, half of cases will present with multiple masses, and both chains can be affected.

Clinical appearance: Tumors can be discrete or infiltrative, soft or firm, ulcerated and may be fixed to underlying tissues. Ulceration is common in cats and suggestive of malignancy.

Diagnostics: Early detection and diagnostics are key. As with dogs, mammary masses are often incidental findings during routine wellness exams in older females, or owners find them. Owners should be encouraged to palpate their cats monthly for skin and subcutaneous masses—See Something, Do Something (SSDS). Why Wait? Aspirate.

The work up includes tumor measurement, lymph node evaluation, imaging (chest radiographs and abdominal ultrasound), and histology of the tumor. Incisional biopsy can be considered if a benign tumor is suspected, but in most cases, histology is performed on tissue removed at radical mastectomy. Size is the most reliable prognostic factor for cats.

Prognostic factors: Size matters! Size is the most reliable prognostic factor for cats. (And gender too.) MST based on tumor size with surgery alone:

<2 cm: > 3 years with mastectomy for females, 14 m for males
2-3 cm: 1-2 years for females, 5.2 months for males
> 3 cm: 4-6 months for females, 1.6 months for males

Surgery type: radical mastectomy decreases recurrence locally
Histology: complex carcinoma MST 33 months vs 15 months for other carcinomas
Grade: higher grade, shorter ST
Lymphatic invasion
Higher clinical stage at diagnosis is also associated with a worse prognosis
IMC has a similar poor prognosis in cats.

Treatment:
Surgery: Surgery is the first treatment of choice for mammary tumors when there is no evidence of distant metastasis based on staging tests. Unlike dogs, adjuvant chemotherapy is typically part of the recommended treatment protocol. The type of surgery in cats is different than dogs. The recommended surgery is radical mastectomy, and staged bilateral radical mastectomy is recommended if bilateral disease is present. Radical mastectomy reduces the risk of local recurrence. Local recurrence is >50% with incomplete resections. Underlying muscle and fascia should be removed en bloc. It is recommended to always remove the affected LN with the chain. In cats with unilateral disease, the benefit of bilateral surgery is not clear.
Chemotherapy: The goal of chemotherapy is to achieve is to delay the metastatic disease in high-risk patients, including cats with poor prognostic factors. Additional studies are needed to determine the best protocols and survival advantage. Various protocols often include doxorubicin, cyclophosphamide, and carboplatin.
Radiation: Unfortunately, there is little information about RT but can be considered as palliation for non-resectable tumors.

Prognosis:
1-year survival: 33-50% surgery alone, 59% with adjuvant chemotherapy
2-year survival: 15-20% surgery alone, 37% with adjuvant chemotherapy

ORAL SQUAMOUS CELL CARCINOMA
What is it: Oral cancers account for 3 to 10% of feline neoplastic diseases. Squamous cell carcinoma (SCC) is by far the most common feline oral malignancy. SCC makes up about 75% of all feline tumors and occurs in the gingival and sublingual area with equal frequency.

What I say: SCC is a tough cancer in cats with a generally poor prognosis. Oral SCC in cats is difficult to manage due to the invasiveness. Cats can have temporary responses to radiation and chemotherapy but side effects often require a feeding tube. I tend to opt for a palliative approach. The occasional cat with a small lesion found early may have extended survival with surgery, radiation and chemotherapy.

Clinical appearance: Older cats are affected, and cats with tumors in the rostral cavity may be presented for a visible mass, but unfortunately many tumors go undetected because they are difficult to see and many owners do not get to look in their cat's mouth. Often a suspicious mass is found at a dental or there are loose teeth with otherwise good dentition.
Common clinical signs include pytalism, halitosis, dysphagia, weight loss, hyporexia, reluctance to eat, local lymph node enlargement. Owners may notice pawning at the mouth or note blood stained water or food in the bowls, or on bedding. Other findings include facial asymmetry, exophthalmos, and nasal discharge. On exam the gingiva is often irregular and ulcerated. The gingiva may also be normal and there is a large swelling of the bone.
Oral SCC may be associated with canned cat food or canned tuna intake, flea collar use, and possibly environmental tobacco smoke

**Diagnostics:** CBC, chemistry panel and urinalysis is recommended prior to anesthesia. Hypercalcemia may be seen as a paraneoplastic syndrome. Regional lymph node metastasis is relatively uncommon. 3-view chest radiographs are recommended but metastasis to the lungs is rare at diagnosis. It may be possible to perform fine needle aspirate and cytology of the oral mass with the cat awake, but if sedation is needed, a biopsy is recommended. Severe inflammation may affect confirmation with cytology. Visual assessment typically greatly underestimates the extent of feline SCC. Dental radiographs or ideally CT scan of the affected site can help determine local extent, bone involvement, and aid in planning treatment with surgery or radiation.

**Treatment:**

**Surgery:** Without surgery, the response is poor, but cats may not tolerate extensive oral surgery and may have problems eating. For gingival masses, aggressive surgical resection is required including partial mandibulectomy or maxillectomy.

**Radiation:** Definitive radiation can be considered for residual disease for incomplete margins. Coarse fractionated radiation is not recommended due to poor efficacy and treatment side effects.

**Chemotherapy:** Chemotherapy alone offers little benefit for macroscopic SCC. Improved response rates may be achieved for non-resectable masses with combined radiation and mitoxantrone or gemcitabine chemotherapy.

**Medical management:** COX 2 expression was shown in canine oral SCC and cats with cutaneous SCC. The NSAID piroxicam has shown some benefit as a single agent and combined with chemotherapy. A dose of 0.3 mg/kg q 24 hours to q 48 hours is recommended if renal function is adequate. In addition, I stress the importance of pain management and nutritional support, including PEG tubes. I also recommend owners weigh their cats regularly.

**Prognosis:** Overall, the prognosis is poor for feline SCC and less than 10% survive 1 year. Local disease progression is typically the reason for death, rather than metastasis. Reported MST in cats treated with radiation and chemotherapy (without surgery) is typically poor and less than 6 months. If surgery is combined with radiation and a gastrotomy tube, MST is improved to 14 months. Euthanasia is typically due to local recurrence.

**SOFT TISSUE SARCOMAS (NATURALLY OCCURRING & INJECTION SITE)**

**What is it:** Soft tissue sarcomas (STS) are mesenchymal tumors characterized by local aggressive behavior and high recurrence rates. STS are tumors derived from non-epithelial extraskeletal tissue and includes tumors of muscle, fat, and connective tissue. In cats, we see naturally-occurring STS and injection site sarcomas (ISS).

**What I say:** STS are tumors of connective tissues. Whether naturally-occurring or injection site associated, they are locally aggressive. They have tentacle-like projections extending from the mass you can see and feel. I think of them like weeds with roots. If we just remove the mass, the weed, and leave the roots, the tumor grows back like a weed.

**Cause:** The most widely publicized cause is the association of vaccination and STS in cats. The most commonly implicated is rabies and feline leukemia vaccines, but other injections can cause this including antibiotics and steroids. The incidence is estimated to be 1:10,000. It is believed that inflammation at injection sites causes local cell proliferation which may lead to tumor development.

**Clinical appearance.** Cats are typically presented for the mass itself. They can vary in appearance. No one, not even a cancer specialist like me, can look at a mass or feel a mass and know what it is, benign or malignant, or tumor type. Cats with ISS are often younger (7-9 years) compared to cats with non-vaccine tumors. Typical locations for ISS are interscapular, dorsal lumbar, flank, or lateral thorax.

**Diagnostics:**

**Cytology:** Fine needle aspiration for cytology is an appropriate first step even though mesenchymal tumors often have low yield as they do not exfoliate well. Neoplastic fibroblasts can be difficult to differentiate from reactive ones. Still cytology can rule out epithelial and discrete cell tumors. Reports cite 93% accuracy with high specificity for diagnosis of malignant mesenchymal tumor.

“See Something Do Something. Why Wait? Aspirate.” (SSDS) provides guidelines for evaluating superficial masses in dogs and cats. These guidelines will increase client awareness and will promote early cancer detection, diagnosis, and early surgical intervention. In veterinary medicine, most skin and subcutaneous tumors can be cured with surgery alone if diagnosed early when tumors are small. **See Something:** When a skin mass is the size of a pea (1 cm) and has been present for at least 1 month, **Do Something:** Aspirate or biopsy, and treat appropriately!

Staging diagnostics may include incisional biopsy for grade, CBC, chemistry panel, urinalysis, chest radiographs, and abdominal ultrasound. Advanced imaging with CT or MRI can help determine local extent and aid in
treatment planning. Simple excisional biopsy is not recommended to confirm the diagnosis because this will be inadequate for local control.

The biopsy report: The report should be evaluated for grade, completeness of margins and mitotic index. Histologically, ISS are characterized with cystic areas, a transition zone with inflammation, aluminum-containing macrophages and myofibroblasts. Multi-nucleated cells may be present and correspond to high grade tumors. For cases in which histologic margins are not complete, further local therapy is recommended. This may be a scar revision in attempt to get wide margins, or adjuvant radiation is recommended.

Treatment:
All cats with STS require aggressive local therapy (surgery or surgery and radiation). For high grade STS and ISS, trimodality therapy is considered due to higher metastatic rates.

Surgery: Radical surgery is the treatment of choice when possible, and excision should involve gross tumor margins of at least 3 cm laterally and one fascial plane deep of normal tissue. Histologic margins of at least 5 mm and one fascial plane deep typically offers excellent local control. Tumor cells often invade beyond the pseudocapsule, which means that a sarcoma that shells out easily has most likely left microscopic disease behind and recurrence is likely. Tumors on the distal limbs and tail may be successfully controlled with amputation. In some locations, wide margins are often not possible, such as the distal limb, or a tumor will be too large to get wide and clean margins, and further therapy will likely be needed after surgery to improve local control. Post-operative options include adjuvant external beam radiation, a second surgery for a scar revision, and chemotherapy. This emphasizes the need for early detection and identification of what the mass is prior to resection. With adequate local control, MST exceed 3 years, and there are reports up to 5 years.

Radiation therapy (RT): Monotherapy has varying control rates, and a better approach is often surgery with adjuvant radiation. Adjuvant radiation is recommended when wide surgical excision not feasible. In cats with ISS, surgery and radiation are often the standard of care for local control as complete excision is often a challenge. In one report of cats with complete excision, 31% had recurrence. MST often exceed 2 years for cats treated with combined surgery and radiation.

Chemotherapy: Patients to be considered for chemotherapy include high-grade STS and ISS. However, the role of chemotherapy is much clearly defined, as the benefit of chemotherapy for high grade STS and ISS is still not clear. Doxorubicin is considered the most single effective chemotherapy may improve local control for ISS. Many oncologists including me recommend chemotherapy as studies are limited.

Prognosis: Wide excision for naturally occurring sarcomas can offer surgical cure, and the prognosis is good if complete excision can be achieved. This is most commonly tumors on tail, distal limbs, and small localized tumors. Another prognostic factor for ISS is the first excision done at a referral surgeon, as these surgeries are likely more aggressive. Unfortunately, complete excision can be challenging because of the locally aggressive nature and high recurrence rate with incomplete excision following surgery alone. For larger tumors treated with multimodality therapy (aggressive surgery, radiation and chemotherapy), cats can have an excellent long-term prognosis. However, a subset of cats with develop metastasis and/or local recurrence. Median survival with surgery followed by radiation is about two years. The metastatic rate for cats with the ISS is reported to vary from low to moderate. Although chemotherapy does not seem to improve outcomes following surgery and radiation, adjuvant doxorubicin may benefit cats for surgery alone. Cats with large non-resectable tumors or metastases at diagnosis have a poor prognosis.

Earlier detection and treatment may result is easier treatment and an improved prognosis.

Prevention:
Earlier detection may lead to more successful outcomes. Guidelines for monitoring cats with a mass after vaccination include:

- Mass increasing in size after 1-month post injection
- Mass > 2 cm
- Mass persists 3 months after injection

An incisional biopsy is then recommended. Vaccination is recommended in areas that allow for aggressive surgical removal, such as the limb. We should critically evaluate vaccination protocol for cats with low risk of contracting infectious disease. We must document vaccine location in the medical record. Finally, we must educate the clients about the risks and benefits of each vaccine and the importance of palpating cats monthly at home for early detection of these tumors.

REFERENCES:

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CANCER CLIFF NOTES FOR DOGS: WHAT YOU NEED TO KNOW
Sue Ettinger, DVM, DACVIM (Oncology)
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CANINE LYMPHOMA

What is it: Lymphoma is a common canine cancer and is a systemic disease that requires chemotherapy in almost all cases. It is a collection of cancers arising from the malignant transformation of lymphocytes. Lymphoma is one of the most common canine cancers, accounting for 7-24% of all canine tumors and 85% of hematopoietic tumors. Dogs of any age, gender, and breed can be affected with lymphoma. Affected dogs are typically middle aged to older dog. Multicentric (PLN) is the most common form, accounting for 80% of lymphomas.

What I say: Lymphoma is a cancer of one the white blood cells called the lymphocytes. These cells accumulate on the dog's lymph nodes, also called lymph glands – making the connection with Strept throat and swollen glands. Lymphoma is a treatable disease, and treated dogs live significantly longer, and treatment is very well tolerated.

Clinical appearance: The most common complaint is generalized lymphadenomegaly. Owners commonly report that lymph node size is rapidly increasing – over days to 1 to 3 weeks. Most dogs are typically asymptomatic and appear healthy in the early stages. Twenty to 40% are clinical (substage b) with anorexia, lethargy, fever, vomiting, diarrhea weight loss, melena.

Diagnostics: Early accurate diagnostics and careful staging are keys to proper clinical decision-making. The minimum tests required for treatment are cytological confirmation (lymph node or affected organ), CBC, chemistry panel and urinalysis. The next diagnostic I encourage owners to submit is phenotyping to determine B vs T-cell subtype. To stage or not to stage? Complete lymphoma staging includes lymph node cytological confirmation, CBC, chemistry panel, urinalysis, lymph node histology, urinalysis, thoracic radiographs, abdominal ultrasound, bone marrow cytology and phenotyping. These tests are useful and informative, as they provide prognostic factors and a baseline for a patient’s response. These tests can also help determine if there is a large tumor burden and risk for acute tumor lysis syndrome with induction chemotherapy. Still, we must consider the owner’s financial issues. While it is ideal to perform all the tests, we can also consider each test on a case by case basis and help the owner make an educated decision. We can treat without but review pros and cons with the owner and let owner make educated decision and maybe choose more important tests for that dog.

Prognostic factors: There are many prognostic factors, but the more significant predictors include:

- Phenotype: Phenotype is the best independent prognostic factor; prognosis is worse with T-cell than B-cell. 60-80% are B-cell and this is associated with higher rated of CR, longer remission rates, and increased survival time (ST). Most high grade LSA are B-cell.
- Substage: clinically healthy dogs tend to do better than sick dogs
- Histologic grade: high grade has better CR rate than low grade, but low grade often has comparable survival times with less intensive chemotherapy protocols.
- Administration of prednisone prior to chemotherapy is a negative predictor
- Higher stage (stage IV and V) tend to do worse than lower stage (I to III)
- Hypercalcemia: negative predictor due to association with T-cell phenotype
- Mediastinal mass: negative predictor due to association with T-cell phenotype

Remember, prognostic factors cannot predict an individual's response, and lymphoma is typically treatable and rewarding to treat for the patient, owner and the veterinarian.

Treatment: Dogs treated with chemotherapy live significantly longer than untreated dogs, and chemotherapy is generally well-tolerated in most dogs. Only a minority develops significant toxicity. Combination chemotherapy provides improved remission rates and duration in comparison to single agent protocols. Multi-agent CHOP protocols are the most successful, with complete remission rates of > 80% and remission durations of typically 6-11 months. Median survival times (MST) are 1 year when followed by rescue protocol, and 25% of dogs are long term survivors > 2 years. If multi-agent CHOP is declined, alternatives include single-agent Tanovea, single-agent doxorubicin, alternating Tanovea/ doxorubicin, and single-agent Lomustine.

If chemotherapy is declined: If chemotherapy is declined, another option is single agent steroids. Typical response rates are 50% with duration of 2 to 3 months. Prednisone should not be started prior to chemotherapy since it may decrease response rate to chemotherapy started after the steroids. Pre- chemotherapy steroids use is associated with shorter remission and survival times due to induction of multi-drug resistance. If staging tests are done after prednisone is started, higher stage patients may appear to be lower stage (down-stage). Without chemotherapy the prognosis for lymphoma is poor, with MST of 1 month.

Prognosis: Without Treatment: 1 month. With treatment: With prednisone only, typical response rates are 50% with duration of 2 to 3 months. Multi-agent CHOP protocols are the most successful, with complete remission
rates of > 80% and remission durations of typically 6-11 months. Median survival times (MST) are 1 year when followed by rescue protocol, and 25% of dogs are long term survivors > 2 years.

CANINE OSTEOSARCOMA
What is it: Osteosarcoma (OSA) is the most common primary canine bone cancer in dogs, accounting for 85% of all skeletal cancers and 5% of all neoplasia. OSA is a malignant mesenchymal tumor of primitive bone cells that produce ECM of osteoid. It is locally aggressive and highly metastatic. The majority of dogs with appendicular osteosarcoma have no evidence of metastasis at diagnosis, but most will likely succumb to metastasis. Dogs treated with local therapy and chemotherapy live significantly longer than dogs without treatment and with local therapy only, and chemotherapy is generally well-tolerated in most dogs. Only a minority develop significant toxicity.

What I say: Osteosarcoma (OSA) is the most common primary canine bone cancer in dogs, and it is locally aggressive and highly metastatic, but treatable.

Clinical appearance Most dogs appear in pain, and many presents with progressive lameness. Palpable swelling may or may not be present. Acute severe swelling is typical with pathologic fracture, but only 3% pathologic fractures due to OSA. If you are presented with a large- or giant-breed dog that is lame and has swelling at metaphyseal site, it is OSA until proven otherwise and do radiographs promptly. The dog SIZE IS MORE IMPORTANT than breed. The risk of OSA is 60 times higher in dogs weighing > 30 kg, and 8 times higher in dogs weighing 20-30 kg. Appendicular OSA accounts for 95% of all cases in dogs >40 kg but only 40-80% of all cases <15 kg. Axial OSA can occur in any breed and at any location. 75% of OSA is appendicular, and 25% is in axial bones. It typically occurs in the metaphyseal region of long bones, towards the knee and away from the elbow, front limbs are two times more affected than hind limbs. The most common sites are the distal radius and proximal humerus, while in the hind limbs, OSA lesions are evenly distributed among the distal femur, distal tibia, and proximal tibia. The proximal femur less common, and OSA distal to carpus and hock is rare.

Diagnostics: Early diagnostics are key. If you are presented with a large- or giant-breed dog that is lame and has swelling at metaphyseal site, it is osteosarcoma until proven otherwise, and do radiographs promptly. When taking radiographs, take good quality lateral and craniocaudal views. The abnormalities vary from mostly lysis to mostly osteoblastic. Common features include cortical lysis, soft tissue extension and swelling, new bone extension in sunburst pattern, Codman’s triangle deposition of new bone on cortex at periphery. While OSA does not cross joint, it can invade adjacent bones. Radiographic changes can be similar to fungal osteomyelitis.

While cytology is not definitive, it is supportive and can distinguish malignant vs non-malignant with an accuracy of 70-85%. In diagnostic samples, ALP staining can differentiate OSA from other sarcomas. Ultrasound-guidance can be helpful to aid sample collection. Pre-op biopsy is not required in cases with classic signalment, history, PE/location, and radiographic appearance, there is little possibility of fungal or bacterial osteomyelitis, and the owners are willing to treat aggressively. On the other hand, biopsy is recommended if there is non-diagnostic cytology, the owner wants confirmation, or it is not a classic case. Always submit larger specimen at surgery to confirm.

Staging includes local LN FNA, orthopedic exam for bone metastasis, 3-view chest radiographs or chest CT scan. Less than 5% of dogs have LN metastasis. Bone survey radiographs are not typically recommended unless there are suspicious or painful lesions. It involves taking a lateral of all bone and VD of the pelvis and can be considered to rule out bone metastasis and 6% of dogs have bone metastasis detected (vs 4% chest). Bone scans have conflicting reports of usefulness. Abdominal ultrasound is not recommended for OSA staging but can be considered if determining is the bone lesion is a metastatic lesion or there are abnormalities on the chemistry panel.

Prognostic factors: Well-established negative predictors include young age (<5 years old), large tumor size, humerus location, and high histologic grade. Other factors include small body size, larger tumors, extraskeletal tumors, percent bone necrosis, mitotic index, and over metastasis (lungs, LN). Stage III dogs with measurable metastasis have a worse prognosis with a MST of 2 months. Dogs with bone metastasis do better than lung (4 m vs 2 m), and LN metastasis is a negative prognostic factor (2 m vs 8 months) Dog with elevated ALP have shorter DFI and ST. Remember, prognostic factors cannot predict an individual’s response. Pathologic fracture is not negative prognostic factor.

Treatment: To determine the best treatment plan for a patient and owners, it is important to understand efficacy of the various protocols, the potential toxicities, and prognostic factors. Treatment for OSA is ideally both local and systemic. Since chemotherapy significantly improves the MST, it is considered part of the standard of care. The majority of dogs tolerates chemotherapy quite well and will maintain a good to excellent quality of life even during chemotherapy.
**Surgery** Surgical options include amputation or limb spare techniques to address the primary tumor. Amputation is the standard treatment for appendicular OSA. It is palliative and a very effective way to remove the source of pain, but amputation alone does not increase survival (other than preventing pain-related death) and most dogs succumb to metastasis. While we as veterinarians know that amputation is well-tolerated, many owners are shocked by the procedure and often reluctant to consider the radical procedure. It is important to screen the patient well and rule out concurrent musculoskeletal and neurologic abnormalities. Even middle-aged and older large-breed dogs with moderate arthritis are typically candidates. Owner satisfaction is typically excellent post-op, and most dogs compensate well. Alternatives to amputation includes limb spare techniques and stereotactic radiation.

**Chemotherapy** The goal of chemotherapy is to achieve is to delay the metastatic disease that develops quickly after amputation or limb-spare procedure. Most studies have evaluated doxorubicin, cisplatin, or carboplatin in varying combinations. Choice of protocol (single vs combination) does not result in significant differences in DFI or ST, the carboplatin protocol resulted in a lower proportion of dogs experiencing side effects, and helpful in maintaining high quality of life during treatment. Unfortunately, 95% of dogs will eventually succumb to metastasis.

**Other treatment options:** Other treatment options include bisphosphonates, immunotherapy, and COX-2 inhibitors. Pain control for the patients is a priority. Bisphosphonates are osteoclast inhibitors than inhibit bone resorption and are used in human patients with diffuse skeletal metastasis. Approximately 30% dogs have decreased pain. Direct cytotoxicity to has also been reported suggesting interaction with radiation therapy and/or chemotherapy. A canine OSA vaccine is currently being developed by Aratana. This is a recombinant HER2/neu expressing Listeria therapeutic vaccine being studied at UPenn. In a recent study by Dr. Mason, 18 dogs were treated with amputation, 4 doses of carboplatin, and the vaccine. The MST has not been reached but 11 of 18 dogs surpassed the MST of the control group (318 days). Adverse effects were mild to moderate and included fever, lethargy, nausea and vomiting. This is currently conditionally licensed and available at about 20 sites.

**Prognosis:** While OSA is highly metastatic, <10-15% have detectable metastasis at diagnosis, but 90% die within 1 year with amputation alone due to metastasis. The MST with surgery alone is 4 to 5 months, with 90 to 100% mortality rate in one year. With chemotherapy the 1-year survival rate is 40-50% and 20-25% of dogs are alive at 2 years.

**CANINE HEMANGIOSARCOMA**

**What is it:** Hemangiosarcoma (HSA) is an aggressive malignant cancer of transformed vascular endothelial cells. It causes local infiltration and rapid systemic metastasis. Dogs with splenomegaly and splenic masses generally follow the “double two-thirds rule”: 2/3 have splenic neoplasia, and 2/3 of those have hemangiosarcoma. (1/3 do not have cancer!) The likelihood of splenic tumor increases with anemia, nucleated RBC, abnormal RBC morphology, or splenic rupture.

**What I say:** Hemangiosarcoma (HSA) is malignant cancer of blood vessels that can be anywhere from the skin to internal, and the most common internal sites are spleen, heart and liver. Hemangiosarcoma (HSA) is the most common primary canine splenic cancer in dogs, and it is locally aggressive and highly metastatic. **Clinical appearance** Clinical signs are typically vague, non-specific and include enlarged abdomen, anorexia, lethargy, depression, vomiting, and diarrhea. Clinical signs also vary with how advanced disease is, so dogs may have acute and often dramatic acute signs including collapse and hypovolemic shock. In one study 80% of dogs with acute abdomen and no history of trauma had malignant cancer and 88% were HSA. Spleen is the most common primary site, but other common sites include right atrium, liver, skin and subcutis.

**Diagnostics:** CBC, chemistry panel and urinalysis: The likelihood of splenic tumor increases with anemia, nucleated RBC, abnormal RBC morphology, or splenic rupture. The anemia may be regenerative with splenic rupture depending on the duration. Neutrophilic leukocytosis may also be present. Other abnormalities include Howell-Jolly bodies, poikilocytosis, acanthocytosis, schistocytosis and/or thrombocytopenia. Thrombocytopenia is common in 75-97% cases and ranges from mild to severe. A coagulation panel should be run if HSA is suspected.

**Imaging** Three-view chest radiographs are mandatory to rule out pulmonary metastasis and pleural fluid. Three-views significantly decreases the false-negative rate. Abdominal ultrasound confirms the mass and allows detection of abdominal effusion, defines splenic architecture, and provides detailed evaluation of the abdominal organs and is less affected by abdominal effusion than radiographs. **FNA and cytology:** FNA is not recommended for mixed echogenicity masses suspicious of HSA as the masses are often extremely friable so there is an increased risk of hemorrhage in addition to the low diagnostic yield due to hemodilution. HSA effusions are serosanguinous or frank blood and usually do not clot. Unfortunately, cytology is typically non-diagnostic. But even with ultrasound-guidance, if non-representative tissues are sampled, you may get a false negative of benign or reactive. In one study, only 61% of cases did cytology match histologic diagnoses. **Cardiac evaluation:** Since
25 to 45% of dogs with splenic HSA have concurrent right atrial HSA, an echocardiogram is recommended. In my experience this is lower at presentation. Arrhythmias can occur with benign and malignant lesions. 

**Prognostic factors:** Prognosis for splenic masses cannot be determined without histology which usually requires surgery. A common clinical error is to assume HSA based on the presence of a splenic mass. Large masses are not necessarily malignant. Several splenic lesions have similar ultrasound and gross appearances. Dogs with HSA treated with local therapy and chemotherapy live longer than dogs without treatment and with local therapy only, but 1-year survival rates are still low (10%). Chemotherapy is generally well-tolerated in most dogs, and only a minority develops significant toxicity.

Adjunctive chemotherapy improves the MST. Stage I, non-ruptured tumors may have an improved prognosis when chemotherapy is administered after surgery. Low grade tumors may also have a better prognosis.

**Treatment: Surgery:** Except for lymphoma, splenectomy is the treatment of choice for splenic tumors when there is no evidence of metastasis based on staging tests. Even at surgery, it is often impossible to distinguish various diseases based on gross appearance of the spleen or liver – including hematoma, nodular hyperplasia, hemangioma and HSA. Ideally the entire spleen should be submitted fresh on cold packs or in formalin. Biopsy of normal liver is controversial and may not be useful. The abdomen should be thoroughly explored, and any suspicious lesions removed or biopsied. About 25% of dogs develop arrhythmias post-op. An ECG should be monitored during and after surgery, and they usually resolve within 24-48 hours.

**Chemotherapy:** The goal of chemotherapy is to achieve is to delay the metastatic disease that develops quickly after splenectomy. Since chemotherapy improves the MST, it is considered part of the standard of care. Single agent doxorubicin and combination protocols are most common. Recently low dose oral chemotherapy (metronomic) was comparable to conventional doxorubicin. This protocol included low dose cyclophosphamide, piroxicam and etoposide. Current studies are evaluating whether conventional chemotherapy followed by maintenance metronomic chemotherapy for VEGF-receptor kinase inhibitors such as tocerinib will improve outcome. EBAT is a new therapy being evaluated.

**Supplements:** such as Yunnan Baiyaoa and mushroom polysaccharides can also be considered

**Prognosis:** Overall, the prognosis with surgery alone is poor for hemangiosarcoma. Reported MST in dogs treated with surgery alone ranges from 1 to 3 months, and less than 10% survives 1 year. Adjunctive chemotherapy improves the MST to 4 to 6 months, and doxorubicin-based protocols are the mainstay. Stage I, non-ruptured tumors may have an improved prognosis when chemotherapy is administered after surgery. Low grade tumors may also have a better prognosis.

**MAST CELL TUMORS**

**What is it:** Mast cell tumors (MCT) are the most common cutaneous tumor in dogs, accounting for 16 to 21% of skin tumors. Early detection and aspirates before removal are important for successful outcomes.

**What I say:** One size does not fit all, and MCT are very treatable. While some low and intermediate grade MCT can be cured with a good surgery, some high grade MCT are more aggressive with survival times of less than 3 to 6 months without treatment BUT MCT even high grade ones with spread are treatable!

**Clinical appearance.** MCT are most commonly located on the trunk (50-60%) and then limbs (25%). They have varied appearance but are typically solitary. About 11-22% have multiple lesions. MCT are often called the great impersonator. They can be haired or hairless, slow growing or rapidly growing, present for many months or rapidly growing, ulcerated, and inflamed. No one, not even a cancer specialist like me, can look at a mass or feel a mass and know what it is.

**Diagnostics:** Cytology: “See Something Do Something, Why Wait? Aspirate.”® (SSDS) provides guidelines for evaluating superficial masses in dogs and cats. These guidelines will increase client awareness and will promote early cancer detection, diagnosis, and early surgical intervention. In veterinary medicine, most skin and subcutaneous tumors can be cured with surgery alone if diagnosed early when tumors are small. See Something: When a skin mass is the size of a pea (1 cm) and has been present for at least 1 month, Do Something: Aspirate or biopsy, and treat appropriately!

Staging diagnostics may include incisional biopsy for grade, LN FNA, CBC, chemistry panel, UA, abdominal ultrasound +/- liver/spleen aspirates, bone marrow cytology, and buffy coat.

**The biopsy report:** The report should be evaluated for grade, completeness of margins and mitotic index.

**Histologic Classification: 3-Tiered vs 2 tiered:** Histologic grade is prognostic for biologic behavior and clinical outcome, and an accurate predictor for metastatic behavior. Low grade: <10%. Intermediate grade: low to moderate, High grade: 55-96%. Metastasis is typically to local lymph nodes, liver, spleen, and/or bone marrow. Unfortunately, there is inter-observer variation among pathologists, and pathologists tend to opt for grade 2 when it is borderline between grade 1 and 2. If more pathologists are calling tumors grade 2, the prognostic value is weakened. Based on the original work by Patnaik, there is ~ 50/50 chance of 5-year survival for grade 2 tumors. A
2-tiered system had been developed and is based on the number of mitoses (< or > 7), presence of multinucleated cells or bizarre nuclei, and karyomegaly (increased nuclear size). High-grade tumors are significantly associated with shorter time to metastasis, mast cell tumor associated mortality, shorter overall survival time. MST for high-grade MCT < 4 months vs. > 2 years for low-grade MCT. Ideally you should be getting both grades in your histology reports.

For cases in which histologic margins are not complete, further local therapy is recommended. This may be a scar revision in attempt to get side margins or adjuvant radiation. Frustratingly, histologic assessment of margins may be unreliable. Not all incompletely resected MCT will recur. In some studies, only 20 to 36% with incomplete margins recur. Additionally, recurrence in dogs with clean margins has been reported to be 5 to 37%. Note that microscopic formalin-fixed parameters do not reflect margin size at surgery. Tissue shrinkage of up to 30% for cutaneous tissues occurs.

**Prognostic factors:** Majority of skin MCT are intermediate or low grade can be cured with surgery alone if they are diagnosed earlier when they are small. Prognostic factors include grade (based off surgical biopsy), stage (has is spread?), mitotic index, C-kit mutation, and MCT panel score. Others include size, location, recurrence, causing symptoms.

**Treatment:** Treatment of MCT can vary from simple and straightforward to complicated and controversial. Treatment decisions are often based in the clinical stage (presence of regional and/or distant metastasis) and the presence of prognostic factors. Surgical resection with clean and wide margins is recommended, but questions often arise in determining which dogs needs chemotherapy post-operatively. Should chemotherapy protocol be different for dogs with or without the c-kit mutation? And what to do with dogs with multiple MCT? Dogs with low grade/grade 1 MCT or that have low proliferation scores and are c-kit negative can typically be managed with local therapy (surgery and/or radiation).

**Surgery:** Surgery is the ideal treatment in areas amenable to wide resection. The recommendations for margins have historically been 3 cm, but 2 cm lateral margins may be adequate for most. For small and lower grade, extensive deep margins are just as crucial: 1 fascial plane deep, 4 mm deep margins. The majority of naïve dermal MCT are intermediate or low grade and will be cured with surgery alone, provided site is amenable. In some locations, wide margins are often not possible, such as the distal limb. In my opinion, amputation is probably too aggressive. But further therapy will likely be needed after surgery. Post-operative options include external beam RT, scar revision, and chemotherapy. This emphasizes the need for early detection and identification of what the mass is prior to resection.

**Radiation therapy (RT)** is recommended when wide surgical excision not feasible. Monotherapy has varying control rates with reported 1-year control rates of 50%. However, a better approach is often surgery with adjuvant radiation. First surgery is performed to achieve microscopic disease (clinical stage 0) followed by full course RT. A typical course of radiation is 15 treatments over 3 weeks and has high control rates of 85-95% 2-year control rates for grade 1 and 2 MCT. For macroscopic MCT, the combination of steroids with palliative radiation has been reported to have an improved overall response rate (ORR) of 75%. Palliative radiation is typically weekly radiation for 4 weeks.

**Intralesional therapy:** Intralesional therapies such as deionized water or corticosteroids may provide temporary shrinkage but unfortunately are rarely effective for long-term tumor control.

**Supportive medications:** Any dog that has grossly detectable tumor should have supportive medications, including H1 blocker (diphenhydramine) and a proton pump blocker (omeprazole) or H2 blocker (famotidine).

**Chemotherapy:** Patients to be considered for chemotherapy include high-grade MCT and intermediate grade tumors with regional or distant metastasis or high risk based on biopsy or special stains (including increased mitotic index and c-kit mutations positive). The goal of systemic adjuvant chemotherapy is to decrease the likelihood of metastasis and improve disease free intervals. Drugs used are vinblastine, Lomustine, Palladia, cyclophosphamide, hydroxyurea, and chlorambucil. Dogs with multiple MCT in a short time period may also be considered for chemotherapy, as well as for non resectable MCT in the neo-adjuvant setting (prior to surgery). Chemotherapy for non-resectable MCT is considered palliative. For dogs with measurable tumors, chemotherapy has variable response rates, and responses tend to be short-lived. Response rates of up to 64% have been reported, but studies have shown that combination therapies offer improved efficacy over single agent protocols. I prefer the combo of vinblastine and prednisone, which has reported efficacy for gross disease of 47%.

**Steroids:** Steroids can have an anti-cancer effect and also decreased peritumoral edema and inflammation. As a single agent in grade 2 and grade 3 MCT, the overall response rate (ORR) was 20% (5 of 25). But when steroids were given orally prior to radiation for non-resectable grade 1 to 3 MCT, the ORR was higher at 75% (18/24). When given prior to surgery (neo-adjuvant) for grade 1-3 MCT, the ORR was 70%.
REFERENCES:


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KEY POINTS
Lymphoma (LSA) is one of the most commonly occurring cancers in cats. Lymphoma is a systemic disease that requires chemotherapy in almost all cases. Treated cats live longer, and chemotherapy is generally well-tolerated. Outcomes for treated cats are less predictable than dogs, but cats tend to tolerate chemotherapy better than dogs. The most common form of lymphoma in cats is gastrointestinal lymphoma. Gastrointestinal lymphoma can be divided into large cell/high grade and low grade/small cell forms. For small cell lymphoma, a common diagnostic challenge for the clinician is to diagnose and distinguish it from inflammatory bowel disease based on presentation, examination, and diagnostics.

BIOLOGY OF LYMPHOMA
Lymphoma is collection of cancers arising from the malignant transformation of lymphocytes and is a diverse group of neoplasms with the common origin of the lymphorecticular cells. In contrast to dogs, feline lymphoma most commonly affects the gastrointestinal (GI) tract. Lymphoma is one of the most common feline cancers, reported at 30% of all cancers. In the FeLV era from the 1960-1980s, lymphoma accounted for 50-90% of hematopoietic tumors. However, there was a shift after the 1990s, also called the post FeLV-era. With the aid of FeLV diagnostic assays and elimination regimens in 1970s and 1980s, there was a dramatic decline in FeLV-associated LSA. Still lymphoma prevalence is increasing, especially the alimentary form.

FELINE CHRONIC SMALL BOWEL DISEASE (CSBD)
Feline chronic small bowel disease (CSBD) can a challenge for both the pet owner and the veterinarian. CSBD often is often considered normal by cat owners, and chronic vomiting is often considered normal by pet owners. Common causes of small bowel disease include gastrointestinal lymphoma and inflammatory bowel disease.

Idiopathic inflammatory bowel disease (IBD) is a common chronic enteropathy of cats that is immunologically mediated. Mucosal inflammation is most often found in the small intestine but can involve any part of the gastrointestinal (GI) tract. Some affected cats also have inflammatory disease in the liver and/or pancreas (‘triaditis’). IBD should be suspected in any cat with GI signs and weight loss.

LYMPHOMA ETIOLOGY
Viral: In the FeLV era of the 1960-1980s, two-thirds of lymphoma was associated with FeLV antigen. FeLV-positive cats had a 62-fold increased risk. This form was predominantly seen in younger cats, was the mediastinal form, T-cell, and the virus had a direct role in tumorigenesis. Being FIV-positive increased lymphoma incidence by 5-6x. In contrast to FeLV, FIV has an indirect role secondary to immunosuppressive effects and is associated with B-cell and the extranodal form. Cats that are both FeLV and FIV positive have an increased risk of 77-fold

Immunosuppression: FIV has an indirect role with lymphoma secondary to immunosuppressive effects. Ten percent of feline renal transplants develop lymphoma following transplant and associated immunosuppressive therapy.

Environmental: Environmental tobacco smoke (ETS) has been reported to increase the risk of LSA by 2.5 to 3.2-fold.

Genetic and molecular factors: The predisposition of oriental breeds suggests a heritable risk, but this is still being investigated

Chronic inflammation: While definitive proof is lacking, there is growing evidence of the link with chronic inflammation and lymphoma, in particular with and intestinal LSA. This has been as area of interest with IBD and GI LSA.

Diet and GI LSA: While definitive proof is lacking, the diet changes over last 20 years in response to diseases such as urinary tract and the increase in GI LSA has led to the suggestion of a link, but more studies are needed.
Signalment: Lymphoma can occur in cats of any age, any sex, any breed. The median age is 11 years, and a male predisposition is reported, and intact females are at decreased risk, suggesting a protective benefit of sex hormones. Overrepresented breeds include Siamese cats, Manx, and Burmese. Signalment varies with anatomic site and FeLV status.

LYMPHOMA PATHOLOGY AND BEHAVIOR
Alimentary lymphoma typically involved the intestines alone or intestines, lymph nodes (LN), and liver. In the GI tract, it can be solitary vs diffuse. 55% of GI tumors are lymphoma. Siamese are at increased risk. The GI form typically occurs in aged cats of 12 to 13 years old. The small intestines are four times more affected than the large intestines. Enteropathy-associated T-cell lymphoma (EATL) has 2 forms. EATL Type I is intermediate to large B-cell, high grade, lymphoblastic lymphoma. This form often has a palpable mass. EATL Type II is called small cell, low grade, lymphocytic lymphoma. This form is more diffuse throughout the GIT, and T-cell is more common.

LYMPHOMA CLINICAL APPEARANCE:
For low grade small cell lymphoma, clinical signs include weight loss (83-100%), vomiting/diarrhea (73-88%), anorexia (66%), and icterus (7%). 70% have abnormal palpation on exam, either thickened GI or a palpable mass 33%. The history is usually chronic over several months, with a median 6 months.

For high grade lymphoma, the clinical signs are similar, but icterus is more common, and the onset is more rapid – days to weeks. A palpable mass is common. Rarely the cat will present with acute abdomen due to GI obstruction or perforation.

DIAGNOSIS AND STAGING
Early accurate diagnostics and careful staging are keys to proper clinical decision-making. The diagnosis is typically straightforward for high grade lymphoma, and the diagnosis is typically made with abdominal ultrasound and cytology of a lymph node or organ. Surgery is less commonly needed. Cytology may be inconclusive and be reported as benign hyperplastic and reactive, and histology will be needed, but this is less common than cases of small cell/low grade lymphoma.

In addition, the minimum tests required for chemotherapy treatment are CBC, chemistry panel and urinalysis. For the GI forms, 23% have panhypoproteinemia and 76% are anemic. I also recommend testing for FeLV/FIV status since FeLV-positive is a negative prognostic factor. Serum cobalamin/folate levels should be evaluated; hypocobalaminemia is associated with high- and low-grade lymphoma but is also seen with inflammatory bowel disease (IBD).

Additional tests to consider include thoracic radiographs, lymph node histology, bone marrow cytology and phenotyping. Bone marrow cytology may be recommended especially for cases with anemia, leukopenia, or cellular atypia. Phenotype can be determined with PARR (about 80% sensitive) or flow cytometry. Currently, I only recommend PARR testing if histopathology was inconclusive as phenotype is not prognostic in cats (which is different from dogs).

While all these staging tests are useful and informative, as they provide prognostic factors and a baseline for a patient’s response, we must give the owner the ability to make an informed decision considering their financial situation. I consider each test on a case by case basis and help the owner make an educated decision. I recommend choosing the more important tests for that cat based on presentation, physical exam and diagnostic findings, and the owner’s budget. The diagnostic tests also help determine where we need to support the patient to provide the best overall treatment plan for the cat’s health. This goes back to the perception of quality of life and hope for the client.

Low grade lymphoma vs IBD
It can be challenging to distinguish low grade lymphoma vs IBD with abdominal ultrasound. With low grade GI LSA, 60-90% have an abnormal AUS with 50-70% diffuse small intestinal thickening, predominantly muscularis propria and submucosa layers. Mesenteric lymph nodes are abnormal in 45-80%. Focal GI masses are uncommon. For IBD, 10-50% have diffuse small intestinal thickening and mucosal thickening more common. The incidence of mesenteric lymph nodes is lower at 15-20%, and other abnormal organs are typically normal.
Cytology is rarely useful for distinguishing low-grade GI LSA vs IBD. The debate rages on regarding endoscopy vs full thickness biopsy (laparotomy vs laparoscopy). On histopathology, lymphoma typically has lymphoid infiltration beyond mucosal layer, epitheliotrophism, heterogeneity, and lymphocyte nuclear size consistent with malignant. If diagnosis is still equivocal, phenotype with PARR is recommended.

FELINE CHRONIC SMALL BOWEL DISEASE (CSBD)

The Norsworthy study highlights that CSBD often is often considered normal by cat owners. Excuses include: “He just eats fast”, “She is a nervous cat”, “He has a sensitive stomach”, “She gets hairballs”, “He’s always done this.” CSBD includes IBD and enteropathy-associated T-cell LSA (EATL) type 2. EATL type 2 most common infiltrative GI LSA in cats, and treatment is different than IBD.

In this study, the authors looked at the association of clinical signs and disease in 100 cats that had an AUS of small bowel >0.28 cm in ≥ 2 locations. These cats had ≥1: vomiting ≥2x /month for at least 3 months, several weeks of small bowel diarrhea, and weight loss >0.5 kg in last 6 months. Interestingly, 26 cats were getting wellness exam. 65 cats did not have surgery and were excluded. Clinical signs included weight loss 70%, vomiting ≥2x 61%, diarrhea 11%, and V/D 13%. 92% had at least 1 AUS measurement ≥0.3 cm, 8 cats 0.29-0.29 cm, and 76 cats 1 measurement <0.28 cm. 99 of 100 had cats had IBD or LSA. Only 1 cat had normal histology. 49% had IBD/chronic enteritis. 46% had LSA (n=44 EATL type 2). Cats <8 years old had enteritis, and cats > 8 years old enteritis or cancer. The 1 normal cat was 5 years old.

Cats with GI clinical signs are common and should undergo diagnostics. Do not let clients make excuses and get a good history. Chronic vomiting is often considered normal, but vomiting is not normal! Clinical signs should trigger abdominal ultrasound. One of the common excuses is vomiting hairballs is normal. Is vomiting hairballs is normal? Does chronic small bowel disease slow bowel movement and predispose to formation?

LYMPHOMA TREATMENT

Treatment: Dogs vs Cats

There are fewer feline data than for canine lymphoma. Papers often lump together small number of cases of multiple subtypes of various anatomic, phenotype and histologic grades. Outcomes are less predictable in cat and there is greater variation in histologic type and anatomic location in cats. But cats tolerate chemotherapy well and better than dogs. Febrile neutropenia is rare. Most owners happy they chose to treat, and the QOL improves.

Which protocol?

For intermediate and high grade/EATL I:

Improved remission rates and durations are achieved with combination chemotherapy protocols, and there are numerous protocols reported in the literature. There is an overall response of 50-80%, a median remission of 4 months, and median survival times (MST) of 5 to 9 months. Cats that achieve a complete remission have a longer median survival time of approximately 1 year. I typically recommend a CHOP multi-agent protocol such as the UW 25-week protocol. When using doxorubicin in cats, I use a lower dose (1 mg/kg IV). Cardiac toxicity is not clinical problem in cats in contrast to dogs, and renal function (BUN, Cr, USG) should be monitored in cats when giving doxorubicin. In dogs, data supports shorter maintenance-free protocol, but there is less data in cats, and some cats may need chronic maintenance chemotherapy. An alternative protocol is the COP protocol with reported complete remissions of 50-70%. This is commonly used in used in Europe with similar results to CHOP in 1 study. While the protocol requires less frequent visits, it is a longer 1-year protocol. Other studies support the addition of doxorubicin to COP for durable responses.

For single agent options, oral Lomustine can be given at 50-60 mg/m² every 4-6 weeks, which is given at a lower dose and less frequently than dogs. While complete response rates were low at 22%, some cats experienced a remission rate of 10 months. Single agent doxorubicin is cats is less successful with complete remission rates of about 26% and a median of 3 months, and I do not recommend this as a single agent (unlike dogs). I also recommend supplementing cobalamin as indicated.
If chemotherapy is declined: If chemotherapy is declined, another option is single agent steroids. I prefer prednisolone in cats and recommend treatment as long as clinical response is seen. Typical response rates are 50% with duration of 2 to 3 months. Without chemotherapy the prognosis for high grade lymphoma is poor, with MST of about 1 month.

For low Grade/ EATL type II, less aggressive chemotherapy protocols are typically used. Oral chlorambucil (Leukeran®) can be dose with pulse dosing (20mg/m² every 2 weeks or 15 mg/m² for 4 days every 3 weeks) or with chronic dose (>4 kg start @ 2 mg PO q 2 days, maintenance q 3 days; <4 kg start @2 mg PO q 3 days, maintenance q 4 days). For cats I prefer prednisolone, typically at 1 - 2 mg/kg orally daily and reduce to 0.5 to 1 mg/kg daily. In some cases, prednisolone may be discontinued. For relapsed cases, cyclophosphamide, Lomustine, and vinblastine are recommended. For severe or refractory cases, I will used CHOP or COP protocols.

Nutrition for EATL type II (low grade/small cell): With evidence of role of inflammation and many have concurrent IBD, there is thought to consider transition to a novel protein diet and to add probiotics. I also recommend running vitamin B12 levels and supplementing as indicated.

PROGNOSTIC FACTORS
Most cats with IBD respond well to treatment but some will fail due to compliance issues, severe disease, concurrent disease (such as hepatic or pancreatic disease, or hyperthyroidism), and misdiagnosis (especially cats that have been misdiagnosed). Low serum cobalamin has been correlated with poor clinical response in cats with chronic enteropathy.

The prognosis and response in cats with lymphoma are more variable than in canine lymphoma. Prognostic factors include anatomic location, achieving a CR, FeLV status, substage, and a multi-agent protocol (CHOP vs COP). Factors that are NOT prognostic in cats include stage and immunophenotype, age, weight, gender, and FIV. For the gastrointestinal forms of lymphoma, the prognosis is overall extremely variable. For EATL type I/high grade, response rates are 50-75%, median remission duration is 4-6 months, and expected survival is 6-8 months. 15-25% can live 1-2 years. For EATL type II/low grade, remission is generally defined as improvement or resolution of clinical signs, and 70%-85% will respond for a median survival time of longer than years.

Chemotherapy side effects and GI lymphoma in cats:
Chemotherapy is well tolerated in the majority of dogs and cats undergoing treatment. The overall toxicity rate is very low in veterinary chemotherapy patients. In my experience, only 15-20% experience side effects, and this is even less common in cats than dogs. The primary goal is to provide the best quality of life possible for as long as possible. As I say, live longer, live well. Most side effects are mild and medically manageable.

Gastrointestinal (GI) toxicity secondary to chemotherapy includes vomiting, diarrhea, decreased appetite, nausea. These are the same clinical signs that the GI lymphoma causes. Chemotherapy side effects typically occur 1 to 5 days after chemotherapy and are self-limiting – lasting on average 2 to 3 days. Even those side effects are less common in feline chemotherapy patients than in dogs, I recommend being very proactive with nausea/anti-emetic drugs in cats with feline GI lymphoma since they often present with clinical signs similar to the potential side effects from chemotherapy.

As in people, a common and dreaded side effect of chemotherapy treatment is Chemotherapy-Induced Nausea and Vomiting (CINV). As with other causes of inappetence it is important to be proactive, not reactive, especially in cats with GI cancer. Most of these cats have vomiting, diarrhea, weight loss, and some degree of inappetence PRIOR to chemotherapy. Concurrent medical conditions can also contribute and exacerbate CINV, such as pre-existing renal disease, renal disease, pain, and medications such as antibiotics. Additional preventative medications are recommended in these patients.
JUST IN CASE MEDICATIONS AND INFORMATION SHEET

My recently updated client information sheet that walks owners through managing side effects at home can be found at on my website in the Resources section. (https://drscuncanavet.com/pet-owner-resources/). Similarly, patients that present with inappetence or have diseases associated with inappetence and weight loss should also go home with medications and guidelines.

I recommend all patients go home on the first day of chemotherapy with Cerenia®, Mirataz® (mirtazapine transdermal ointment), metronidazole, a probiotic, and the client information sheet. Mirataz is indicated for the management of weight loss in cats.

TREATMENT OF IBD

Treatment for IBD in cats remains empirical as data is lacking or inadequate. Studies in cats with chronic lymphoplasmacytic enteropathy have found that most patients respond to treatment with diet, antibiotics, or immunosuppressive drugs. Sequential treatment helps determine which treatment will be most effective for the individual patient. Using this approach, a diet trial is performed first for at least 7 days and response is assessed. Most cats will benefit from long term dietary therapy even if other treatments are necessary.

The next step is a 14-day trial of metronidazole (15 mg/kg PO daily); if clinical signs improve, the dose is tapered by 25% every 2 weeks until it is discontinued. Cats that are non-responsive to diet and metronidazole are treated with oral prednisolone (2 mg/kg PO daily) alone or in combination with metronidazole. The prednisolone dose is tapered by 25% every 2 weeks to reach the lowest dose that controls clinical signs. Other immunosuppressive medication choices include chlorambucil (2 mg/cat PO every 72 hours) or cyclosporine (5 mg/kg PO daily). Some cats can be weaned from medications and maintain remission with dietary therapy alone. Cats with low serum cobalamin should be supplemented.

Not all owners are able to afford an optimal diagnostic investigation, including biopsies. Clinicians often must treat suspected IBD cases without diagnostic confirmation. These patients should be treated with the sequential therapy approach outlined above. Cats that have significant weight loss and watery small bowel diarrhea should be treated with cobalamin parenterally. Cats with signs of large bowel diarrhea may benefit from dietary fiber supplementation, such as ¼ teaspoon psyllium per meal. Patients that fail to respond to these treatments can be treated with prednisolone with the understanding that it may not be the best therapy should the patient have LSA or an infectious component.

LYMPHOMA PROGNOSIS:

Without Treatment: Approximately 1 month

With treatment: Improved remission rates and durations are achieved with combination chemotherapy protocols. Multi-agent CHOP protocols are typically the most successful. For high grade GI lymphoma, response rates are 50-75%, median remission duration is 4-6 months, and expected survival is 6 to 9 months. However, cats that achieve complete remission can be long term survivors, and 15-25% can live 1 to 2 years. With steroids only, typical response duration is 2 to 3 months.

REFERENCES

KEY POINTS

- **Osteosarcoma (OSA)** is the most common primary canine bone cancer in dogs, and it is locally aggressive and highly metastatic.
- The majority of dogs with appendicular osteosarcoma have no evidence of metastasis at diagnosis, but most will likely succumb to metastasis.
- Early diagnostics are key. If you are presented with a large- or giant-breed dog that is lame and has swelling at metaphyseal site, it is osteosarcoma until proven otherwise, and do radiographs promptly.
- To determine the best treatment plan for a patient and owners, it is important to understand efficacy of the various protocols, the potential toxicities, and prognostic factors.
- Dogs treated with local therapy and chemotherapy live significantly longer than dogs without treatment and with local therapy only, and chemotherapy is generally well-tolerated in most dogs. Only a minority develop significant toxicity.

**Who, What Where, Why**

**What** - Osteosarcoma is the most common primary bone cancer, accounting for 85% of all skeletal cancers and 5% of all neoplasia. OSA is a malignant mesenchymal tumor of primitive bone cells that produce ECM of osteoid. The biologic behavior is aggressive locally and highly metastatic. At the primary site, there may be bone lysis or bone production or both, soft tissue swelling, and pathologic fracture (not negative prognostic). While OSA is highly metastatic, <10-15% have detectable metastasis at diagnosis, but 90% die within 1 year with amputation alone due to metastasis

**Who** - OSA is estimated to occur in > 10,000 dogs per year, but this is likely an underestimate. It typically occurs in middle aged and older dogs, but there is a small peak at 1.5 to 2 years old. Rib OSA occurs in younger adults (5 years old). OSA is common in large and giant breeds with increasing weight and height. In the U.S., breeds most at risk are St Bernard, Great Dane, Irish Setter, GSD, Rottweilers, Dobermans, and Golden Retrievers. The dog SIZE IS MORE IMPORTANT than breed. The risk of OSA is 60 times higher in dogs weighing > 30 kg, and 8 times higher in dogs weighing 20-30 kg. Appendicular OSA accounts for 95% of all cases in dogs >40 kg but only 40-80% of all cases <15 kg. Axial OSA can occur in any breed and at any location.

**Why** - Large and giant-breed dogs are predisposed. Body size (increasing weight and more specifically height) appears to be most predictive factors for OSA, and it is more important than breed. Hereditary basis is suspected based on the large breed prevalence. The most thoroughly described mutation that contributes to formation and/or progression is p53. Additional genetic factors studied are RB and PTEN tumor suppressor genes. OSA is more prevalent in males than females, but in the St Bernard, Great Dane, and Rottweilers, females outnumber the males, and females more affected by axial (except rib and spine). Sex hormones also appear to have a protective role. In Rottweilers neutered before 1 years old, 1 in 4 developed OSA and more likely than intact dogs. OSA has also been associated with fractures, metallic implants, chronic osteomyelitis and ionizing radiation.

**Where** 75% of OSA is appendicular, and 25% is in axial bones. It typically occurs in the metaphyseal region of long bones, towards the knee and away from the elbow, front limbs are two times more affected than hind limbs. The most common sites are the distal radius bad proximal humerus, while in the hind limbs, OSA lesions are evenly distributed among the distal femur, distal tibia, and proximal tibia. The proximal femur less common and OSA distal to carpus and hock is rare.

In the axial location: 27% mandible, 22% maxilla, 15% spine, 14% cranium, 10% ribs, 9% sinonasal, and 6% pelvis. Multicentric is uncommon reported in <10% cases. Extraskeletal OSA is rare and some reported sites include mammary, SQ, spleen, GIT, eye, and kidney.
What do we see? Most dogs appear in pain, and many are presented with progressive lameness. Palpable swelling may or may not be present. Acute severe swelling is typical with pathologic fracture, but only 3% pathologic fractures due to OSA. If you are presented with a large- or giant-breed dog that is lame and has swelling at metaphyseal site, it is OSA until proven otherwise and do radiographs promptly.

DIAGNOSTIC WORK UP
A presumptive diagnosis is based on signalment, history, physical examination, and radiographs. Differentials include other primary bone (CSA, FSA, HAS, HS), metastatic bone cancer (usually diaphyseal), multiple myeloma or LSA of bone, systemic mycosis, bacterial osteomyelitis, bone cysts, and healing bone injury. When taking radiographs, take good quality lateral and cranio-caudal views. The abnormalities vary from mostly lysis to mostly osteoblastic. Common features include cortical lysis, soft tissue extension and swelling, new bone extension in sunburst pattern, Codman’s triangle deposition of new bone on cortex at periphery. While OSA does not cross joint, it can invade adjacent bones. Radiographic changed can be similar to fungal osteomyelitis.

Preliminary Diagnosis: Cytology
While cytology is not definitive, it is supportive and can distinguish malignant vs non-malignant with an accuracy of 70-85%. In diagnostic samples, ALP staining can differentiate OSA from other sarcomas. Ultrasound-guidance can be helpful to aid sample collection.

Pre-op biopsy
Pre-op biopsy is not required in cases with classic signalment, history, PE/location, and radiographic appearance, there is little possibility of fungal or bacterial osteomyelitis, and the owners are willing to treat aggressively. On the other hand, biopsy is recommended if there is non-diagnostic cytology, the owner wants confirmation, or it is not a classic case. Always submit larger specimen at surgery to confirm. When doing pre-op biopsy, plan carefully if limb spare is option so contaminated tissue is removed. There is also 10-20% false negatives rate. The open incisional approach collects a large sample, but post-surgical complications include hematoma, seeding, infection, fracture, and wound breakdown. The trephine technique is 94% diagnostic but increases the fracture risk. Closed needle biopsy can be done with a Jamshidi and is 92% accurate for tumor diagnosis and 83% accurate for tumor subtype, but accuracy is dependent on experience and comfort level. In some case, repeated attempts may yield “reactive bone”. Biopsy at CENTER of lesion, and incision and biopsy tract should be planned that will be removed a definitive surgery. Fluoroscopy or CT-guided biopsy can be useful. Samples collected from the peripheral bone lesion are more likely to be non-diagnostic and contain reactive bone.

Staging
Staging included local lymph node fine needle aspirate, orthopedic exam for bone metastasis, 3-view chest radiographs or chest CT scan. Treatment recommendations and prognosis are based on plain radiographs, not advanced imaging. Less than 5% of dogs have lymph node metastasis. Bone survey radiographs are not typically recommended unless there are suspicious or painful lesions. It involves taking a lateral of all bone and VD of the pelvis and can be considered to rule out bone metastasis and 6% of dogs have bone metastasis detected (vs 4% chest). Bone scans have conflicting reports of usefulness. Abdominal ultrasound is not recommended for OSA staging but can be considered if determining is the bone lesion is a metastatic lesion or there are abnormalities on the chemistry panel. CT is recommended for axial tumors.

PROGNOSTIC FACTORS
For appendicular OSA, the median survival time (MST) with surgery alone is 4 to 5 months. Well-established negative predictors include young age (<5 years old), large tumor size, humerus location, and high histologic grade. Other factors include small body size, larger tumors, extraskeletal tumors, percent bone necrosis, mitotic index, and over metastasis (lungs, LN).

For non-appendicular, the head locations (mandible, maxilla, skull) are locally aggressive but have a lower metastatic rate (37%). With skull surgery alone, the MST is longer than limb at 7 months. The 1-year survival with mandibulectomy is 71%, but the MST is 5 months for maxillectomy. Following rib resection, the MST is 3 month, and 8 months for surgery and chemo. Stage III dogs with measurable metastasis have a worse prognosis with a MST of 2 months. Dogs with bone metastasis do better than lung (4 months vs 2 months), and lymph node metastasis is a negative prognostic factor (2 m vs 8 months). Dogs with elevated ALP have shorter DFI and ST. Remember, prognostic factors cannot predict an individual’s response.
TREATMENT MODALITIES

Treatment pearls
Treatment for OSA is ideally both local and systemic. Since chemotherapy significantly improves the MST, it is considered part of the standard of care. The majority of dogs tolerate chemotherapy quite well and will maintain a good to excellent quality of life even during chemotherapy.

Treatment: Surgery
Surgical options include amputation or limb spare techniques to address the primary tumor. Amputation is the standard treatment for appendicular OSA. It is palliative and a very effective way to remove the source of pain, but amputation alone does not increase survival (other than preventing pain-related death) and most dogs succumb to metastasis. While we as veterinarians know that amputation is well-tolerated, many owners are shocked by the procedure and often reluctant to consider the radical procedure. It is important to screen the patient well and rule out concurrent musculoskeletal and neurologic abnormalities. Even middle-aged and older large-breed dogs with moderate arthritis are typically candidates. Owner satisfaction is typically excellent post-op, and most dogs compensate well.
Surgical limb-spare procedures allow the preservation of limb function and are an alternative when amputation is not physically an option or is declined by the owner. Limb-spare procedures do not increase survival times and systemic therapy is still recommended after the delay metastasis. There are various limb-spare procedures described but the techniques involve surgical resection of the affected bone and replacement with a bone implant, bone plating and arthrodesis to stabilize the joint. Since residual disease likely remains, the region is treated with radiation, IA cisplatin or chemotherapy impregnated beads. Distal radius and ulnar lesions are most amenable. These techniques have similar survival times but have much higher complication rates.

Treatment: Radiation
Like surgery, radiation is a local treatment option. Palliative radiation can be very effective for bone tumors, and is a good option if amputation is declined. Most dogs (75 to 90%) respond favorably and analgesia is improved. There is variation with duration of analgesia and most typically lasts 4 to 6 months, but it can be durable for greater than 1 year, and palliative radiation can be combined with adjuvant IV chemotherapy.
Stereotactic radiation therapy (SRT) is an alternative limb-spare technique for local control. SRT delivers extremely precise high dose radiation with multiple beams within submillimeter accuracy. Less normal tissue that surrounds the tumor is irradiated, so there are fewer radiation side effects, higher dose to tumor, and fewer treatments (typically 3).
For OSA, preliminary results are encouraging with MST of approximately 1 year, when combined with chemotherapy for systemic disease. One type of SRT is called CyberKnife Radiosurgery, which I did with our radiation oncologist for 7 years at my previous practice. OSA was the 3rd most common tumor we treated (after brain and nasal tumors). Not every dog with appendicular OSA is a candidate for SRS, especially if there is more bone destruction and increased risk of pathologic fracture. CT-based prognostic factors can help predict the likelihood of fracture.

Treatment: Chemotherapy
The goal of chemotherapy is to achieve is to delay the metastatic disease that develops quickly after amputation or limb-spare procedure. Since chemotherapy significantly improves the MST, it is considered part of the standard of care.
For appendicular OSA, the MST with surgery alone is 4 to 5 months, with 90 to 100% mortality rate in one year. With chemotherapy the 1-year survival rate is 40-50% and 20-25% of dogs are alive at 2 years. Most studies have evaluated doxorubicin, cisplatin, or carboplatin in varying combinations. Choice of protocol (single vs combination) does not result in significant differences in DFI or ST, the carboplatin protocol resulted in a lower proportion of dogs experiencing side effects, and helpful in maintaining high quality of life during treatment. Unfortunately, 95% of dogs will eventually succeed to metastasis.

Other treatment options
Other treatment options include bisphosphonates, immunotherapy, and COX-2 inhibitors. Pain control for the patients is a priority. Bisphosphonates are osteoclast inhibitors than inhibit bone resorption and are used in human patients with diffuse skeletal metastasis. Approximately 30% dogs have decreased pain.
Direct cytotoxicity to has also been reported suggesting interaction with radiation therapy and/or chemotherapy. I recommend zoledronate @ 0.1 mg/kg IV every 4 weeks. This must be diluted in 60 ml 0.9 % NaCl and administered over 15 minutes.

**Osteosarcoma Vaccine**

A canine OSA vaccine (COV) is currently being developed by Aratana. This is a recombinant HER2/neu expressing Listeria therapeutic vaccine being studied at UPenn by Dr Mason. Listeria monocytogenes is a gram-positive, intracellular bacteria that is capable of inducing potent innate and adaptive immune responses. Through its expression of listeriolysin (LLO), a pore-forming protein that enables it to escape from the phagosome prior to lysosome fusion, it rapidly gains access to the cytoplasm of the cell and is then involved in the MHC I-processing pathway. This enables the induction of CD8+ cytotoxic T-cell responses. The use of a highly-attenuated form of L. monocytogenes has been proven effective to introduce target antigens into antigen-presenting cells (monocytes, macrophages and dendritic cells). By genetically fusing the target antigen of interest to LLO, potent CD8+ T-cell responses can be generated against the target antigen. As one can imagine, this is of particular interest in cancer therapy whereby a specific tumor-associated antigen can be used to create a tumor specific immune response. ADXS31-164 is a highly attenuated strain of L. monocytogenes that has a chimeric human HER2/neu antigen fused to its LLO. In mouse models, this has been shown to break tolerance and induce potent CD4+ and CD8+ T-cell responses against HER2/neu. Using the same platform, a recombinant HER2/neu-expressing Listeria-based therapeutic vaccine (AT-014 or Canine Osteosarcoma Vaccine COV) has been licensed by Aratana and is currently in development. In the first small study, 18 dogs with appendicular osteosarcoma (OSA) treated first with amputation and chemotherapy (i.e., 4 doses of carboplatin) received one of four doses of ADXS31-164 intravenously every 3 weeks for 3 administrations. The investigators noted only low grade, transient toxicities. It was found that ADXS31-164 broke peripheral tolerance and induced antigen-specific IFN-γ responses against the intracellular domain of HER2/neu in 15 of 18 dogs within 6 months of treatment. Furthermore, there was a decrease in the incidence of metastatic disease and significantly increased survival time (1, 2 and 3-year survival rates vs. historical control group). The median survival time of 11 historical control dogs was 316 days vs. 8 of the treated dogs with grade I disease, that achieved a median survival time of 956 days (p<0.0001). 11 of the 18 treated dogs have surpassed the median survival time (MST) of the control group. Adverse events were mild to moderate and primarily consisted of fever, lethargy, and nausea/vomiting. Aratana has conditional licensure for this product in the USA and a larger safety study is currently underway.

**SUMMARY**

Osteosarcoma is the most common primary bone tumor in dogs. The biologic behavior is aggressive locally and highly metastatic, so its therapy requires both local and systemic treatments. For appendicular OSA, amputation addresses the local disease and is palliative. The MST with surgery alone is 4 to 5 months, with 90 to 100% mortality rate in one year. The ability to control the progression of OSA metastasis remains the challenge for our patients, and systemic chemotherapy is the backbone for therapy. With chemotherapy, the 1-year survival rate is 40-50% and 20-25% of dogs are alive at 2 years. Well established prognostic factors include adjuvant chemotherapy, low grade (rare for OSA), and normal total and bone ALP. Dogs treated with chemotherapy live significantly longer than dogs only treated with local therapy, and chemotherapy is generally well-tolerated in most dogs.

**REFERENCES**

Oncology is making continual strides with therapies and diagnostic in an effort to help not only diagnose cancers earlier but offer novel therapies to owners.

**CANCER VACCINES**

**Osteosarcoma Vaccine**

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**Oncept anti-CD20 LSA Vaccine**

The canine malignant melanoma (CMM) xenogeneic DNA vaccine has been shown to be safe, results in the development of antibodies and T-cells and is effective. Merial has gained conditional approval for its Oncept lymphoma vaccine, which uses the same concept but is designed to induce immunity to CD20. The concept is therapeutic immunization to be used on achieving remission with chemotherapy. In an abstract of 9 dogs the survival time of vaccinates after completion of 25-week CHOP protocol was >734 days, median not reached, which represents a significant improvement over previously reported historical survival times of 1 year.

Preliminary data suggest that the product needs to start early in the treatment protocol as the average time for the generation of the immune response is likely near one-month post completion of vaccine. Assuming a 6-month protocol + vaccine post completion (2 months), the average remission duration = 6-8 months. Thus, most patients are relapsing likely prior to the vaccine being able to generate an effective response. Data from a recently completed trial in which patients were administered the LSA vaccine during the second cycle of chemotherapy, noted an improvement in remission duration of 4 months.
CHEMOTHERAPY

Tanovea CA-1™ VetDC: Tanovea™ was discovered by Gilead Sciences, Inc., and licensed to VetDC for use in animal cancer, (previously known as VDC-1101). This agent was designed to preferentially target and attack cancer cells implicated in lymphoma. The data from studies totaling well over 330 patients have shown Tanovea™ to be highly effective against lymphoma (LSA) with a 60-80% overall response rate. Not surprisingly, responses are higher in naïve LSA vs relapse and in dogs with a B cell phenotype. Data suggests Tanovea™ is well-tolerated with a similar side effect profile as other commonly used agents. The drug is given via the intravenous route at 1mg/kg every 3 weeks. The FDA has recently given Conditional Approval and the drug is NOT restricted to only oncologists. While the 1st study looked at its use as a single agent, a recent study looked at the use in combination with doxorubicin. The overall response rate was improved to 80% and a progression free interval of 6 months. It is exciting that we have new drugs being developed, giving us more options for our patients.

Although the majority of side effects associated with this agent are similar to those of most chemotherapeutics, two unique side effects (and one that is not unusual) occur, that clinicians need to recognize and know how to treat.

- Pulmonary fibrosis: this was recorded in a small percentage of the patients treated and the mechanism is unknown. As this was fatal in some cases, screening with thoracic films and exclusion of patients with pre-existing pulmonary issues, or particular breeds at risk of pulmonary fibrosis, is warranted.
- Dermatopathy: occurred in a minority of patients and often appears along the pinna and chest. Per VetDC, resolution of the side effects occurs once discontinuing the protocol.
- Anorexia: Although the data provided by VetDC suggests that this is not dissimilar to other agents, the anorexia appears more prolonged than that commonly seen with other agents. We recommend being very proactive with anti-nausea medications and appetite stimulants, using them as preventatives over waiting for anorexia, nausea, and vomiting to occur. To prevent delay chemotherapy induced nausea and vomiting (CINV), I recommend being very proactive with oral nausea/anti-emetic drugs and appetite stimulants given at home in the days following chemotherapy. I always recommend oral Cerenia and Entyce for 7 days.

This represents the first lymphoma specific drug to add to our arsenal in a long time. It will likely be added to a multidrug protocol and the data will suggest where this agent best fits. One option that seems quite interesting based upon the presented data is the combination with doxorubicin, which is a less costly and time efficient protocol compared with standard CHOP.

CANCER SCREENING

The CADET™ BRAF Assay for Diagnosis & Monitoring of Canine TCC

Overall bladder cancer represents < 2% of all canine cancers, however, transitional cell carcinoma (TCC) is the most common bladder cancer. Generally, TCC is generally not diagnosed early but only after it has invaded into the bladder wall, at which point most treatment options are palliative at best. Diagnosis is based upon signalment, ultrasound, urine cytology (tumor cells may be identified in urine sediment in 1/3 of dogs with TCC, but reactive transitional cells may look like TCC cells), however definitive diagnosis generally requires histopathology (cystoscopy, traumatic catheterization, surgical exploratory).

Recent studies identified a mutation (V595E) in the canine BRAF (cBRAF) gene in a large proportion of canine urothelial carcinoma (UC) and prostatic carcinoma (PC). In assessing various cancers including epithelial, mesenchymal, and hematopoietic, the V595E mutation was identified in canine UC and PC with the highest penetrance rates of up to 87%. Knowing bladder and prostatic cancers shed tumor cells into urine, the presence of the V595E mutation in urine appeared to be an excellent molecular diagnostic marker. The molecular test, digital PCR, is a highly sensitive molecular technique enabling detection of a “rare” mutated sequence in clinical samples such as tumor DNA in plasma cell-free DNA, or, in the cancer of bladder/prostate cancer, urine. The digital droplet PCR assay identified the mutation in free catch urine samples from 83% of canine urothelial carcinoma and prostate carcinoma patients.

The assay has since been validated in hundreds of clinical cases, demonstrating the mutation is not present in the urine of healthy dogs, or from dogs that have benign bladder diseases (bladder polyps, inflammation or chronic
cystitis). In cases in which a biopsy of a mass was performed, there was concordance between BRAF mutation-positive in free-catch urine and pathology-based confirmation of a bladder/prostatic carcinoma. As such the presence of the mutation in canine urine is therefore a highly specific indicator of the presence of a TCC/UC.

In contrast, The Veterinary Bladder Tumor Antigen test (VBTA) is 85% sensitive for TCC but only 45% specific in the presence of other urinary tract disease (hematuria, pyuria). It can be used as a screening test and a negative result in 85% reliable but a positive result does NOT equal TCC, and further testing is warranted.

CADET® BRAF-PLUS has since been launched which was developed after testing thousands of dogs in Dr. Breen’s research lab at NC State. This assay detects a second signature in >2/3 of non-BRAF TCCs, i.e. >10% of the 15% BRAF wild type TCC. The new assay increases overall sensitivity to detect a TCC/UC from 85% to >95%. The PLUS assay launched commercially (July 2018) as an ADD-ON test for CADET®-BRAF wild type (mutation undetected) cases. I am currently using the BRAF testing for monitoring during chemo and the BRAF-PLUS as a diagnostic. As of the Spring 2019 this test is available through Antech Diagnostics.

Potential advantages of the assay:

• The free-catch urine system is fully non-invasive, allowing specimens to be collected while the patient is at the clinic, or by the owner at home.
• The assay permits detection for earliest detection of emerging TCC/UC – even before overt signs of the cancer become evident.
• Timely detection of TCC/UC allows owners to direct their resources toward effective treatment of the cancer itself, rather than the non-specific symptoms.
• Unlike prior bladder cancer tests, the CADETSM BRAF Mutation Detection Assay is not affected by the presence of blood, protein, sugars, bacteria etc., in the urine.
• Results are available in just 2-3 business days from receipt of the sample by the testing laboratory.

APPETITE STIMULATION:

Capromorelin
(Capromorelin/AT-002 (Aratana Therapeutics, Inc.) mimics the action of the hunger hormone ghrelin. Ghrelin is a 28-amino acid peptide produced primarily in the stomach and binds the ghrelin receptor. Ghrelin has a short half-life (~10 minutes) and accumulates in the bloodstream slowly between meals thus higher concentrations develop the longer the time frame between the meals. Specifically, ghrelin binds to receptors which increases signaling in the hypothalamus, resulting in hunger thereby increasing food intake. Secondary effects of ghrelin include stimulation of growth hormone (GH) secretion by activation of GHS-Rs in the hypothalamus and pituitary gland, which subsequently increases insulin-like growth factor-1 (IGF-1) production. This results in an increase in lean body mass.

Capromorelin is an orally active small molecule which has more sustained effects vs ghrelin. The mechanism of action involves binding to GHS-R, a G-protein-coupled receptor which in turn activates protein kinase C thereby stimulating GH releasing hormone (GHRH) release from the hypothalamic neurons and GH release from the pituitary gland. This ultimately results increased levels of circulating GH. The drug has been shown to be safe in both cats and dogs and more specifically has been shown to cause increased food intake and weight gain in both laboratory and client-owned dogs and increased food intake/body weight in cats.

Capromorelin oral solution has also demonstrated a wide margin of safety, being well tolerated at daily doses for 12 consecutive months. ENTYCE was granted FDA approval in May 2016, and Aratana has been commercially available for dogs since the fall of 2017. It is labeled for dogs but can be used at lower doses off-label in cats.

Mirtazapine
Mirtazapine is often considered for its potential appetite stimulant effects and is a commonly used appetite stimulant in cats. Used as an anti-depressant in humans, side effects include increased appetite and weight gain, but the specific method of appetite stimulation is not well understood. Mirtazapine is a 5-HT3 receptor antagonist with appetite stimulant properties. Recent pharmacodynamic studies have shown it is safe and can be excellent appetite stimulant. Higher doses, however, are commonly associated with side effects such as vocalization,
hyperexcitability, and tremors. Thus, the recommendation is the use of smaller, more frequent doses. Still a challenge is in the administration via a pill.

In a study of cats with CKD, the recommended dose is 1.88 mg PO q 48 hours (practically 1/4 of a 7.5 mg tablet). When used for 3 weeks, cats with CKD demonstrated a significant increase in appetite and activity and significant decrease in vomiting and a significant gain in bodyweight (median gain 0.18 kg) compared to placebo. The antiemetic effect noted in cats with CKD makes it an option for chemotherapy-induced nausea and vomiting in veterinary oncology. In dogs, mirtazapine may be less reliable and an unpredictable appetite stimulant than cats. Mirtazapine appears to be metabolized much more quickly in dogs and twice daily dosing may be more appropriate in this species.

Mirataz® (KindredBio) is mirtazapine transdermal ointment is a novel formulation for topical use in cats that are resistant to pilling. Approved in the summer of 2018, it is the first and only FDA-approved transdermal product for management of weight loss in cats. Data has shown the product is not only safe but result in weight gain normal cats. This represents another option for cats with cancer and inappetence in general.

Data has shown that mirtazapine transdermal ointment is safe and results in weight gain in normal cats and cats with underlying disease. It represents another option for cats with cancer and other diseases causing inappetence and weight loss. I find it helpful to use mirtazapine transdermal ointment with maropitant in cats that aren’t eating when it’s unclear whether nausea is contributing to the inappetence. This approach has been especially helpful for cats with gastrointestinal lymphoma.

REFERENCES:

- Thamm DH, et al. BMC Veterinary Research 2014;10:30-34.
- Johannes C. New Oncology Therapeutics on the Horizon VCS 2015
- Buhles W, Quimby JM, Labelle D, et al. DOI: 10.1111/jvp.12691
Diagnosis and Treatment of Canine Glaucoma
Mark Bobofchak, DVM
Eye Care for Animals
Akron, OH

Thanks to Tiffany Blocker, DVM, ACVO with ECFA in Tustin, CA for the majority of information in this presentation.

GLAUCOMAS

- A group of diseases characterized by progressive damage to the optic nerve and decreased retinal sensitivity which eventually leads to visual field defects and blindness
- Majority of the glaucomas are associated with increased IOP
  - D. Brooks

Secondary glaucoma

- Cataracts lens induced uveitis (LIU)
- Lens luxation (anterior, posterior, sub)
- Uveodermatologic syndrome VKH
- Other uveitides (infectious, idiopathic...)
- Intraocular tumor
- Retinal detachment
- Hyphema
- Traumatic

Primary glaucoma

- Intraocular pressure >25mmHg with outflow pathway changes
- IOP spike can permanently damage optic nerve within hours
- Any breed but remember the classic breeds!
- Classic signs BLIND, RED, PAINFUL, DILATED, DIFFUSE CORNEAL EDEMA
- When in doubt treat
Types of primary glaucoma

- Congenital glaucoma (uni or bilateral)
  - Great dane
  - Mastiff
- Primary angle closure glaucoma (PACG)
  - Basset, Beagle, Cocker, ChowChow, etc
- Primary open angle glaucoma (POAG)
  - Beagles
- Pigmentary glaucoma
  - Cairn terrier

Aqueous Production/Outflow

- Aqueous produced by ciliary body cells
- Most aqueous courses through space between iris and lens, through pupil, to exit eye through the iridocorneal angle
  - Conventional outflow
- Small percentage exits through ciliary and choroidal vasculature in posterior segment
  - Unconventional outflow
    - Dog = 15%
    - Cat = 3-6%
    - Horse > 20%

Glaucoma analogy

- “Sink” analogy works well
  - Ciliary body = Faucet
  - Iridocorneal angle = Drain
- Most canine glaucomas involve blockage of the outflow tract = Blocked drain
  - Anatomic
  - Inflammatory
  - Compressive
Diagnosing glaucoma

Signalment can be a huge clue!

Primary glaucoma breeds
*classic breeds seen at ECFA

- Arctic breeds *
- Bassett hound *
- Beagle
- Boston terrier
- Bouvier de flandres
- Bullmastiff
- Cairn terrier
- Cocker spaniel *
- Chow Chow *
- Dalmatian

- Flat coated retriever
- Golden retriever
- Great dane
- Norwegian Elkhound
- Poodle
- Fox/ Welsh terrier
- Springerspaniel
- Shar Pei
- Shiba inu
- Shih Tzu

Poster children for glaucoma
Presenting signs are second biggest clue!

**Glaucoma vs Exophthalmia**

- Acute history
- Normal size globe
- Normal lens
- Grey swollen or hyperemic optic nerve

**Chronic versus**

- History
- Buphthalmos
- Lens subluxation
- Atrophied or cupped optic nerve

**Chronic vs. acute**

- Chronic history
- Buphthalmos
- Lens subluxation
- Atrophied or cupped optic nerve

- Acute history
- Normal size globe
- Normal lens
- Grey swollen or hyperemic optic nerve
Examination and diagnostics

- Menace response
- Dazzle reflex
- Pupil size
- Direct/indirect pupillary light reflex
- Fundic evaluation
- Tonometry
- Gonioscopy (contralateral eye)
- Prompt treatment and referral
Using the Tonopen

- Make sure patient is relaxed with minimal restraint
- Neck, eyelids
- Apply topical anesthetic
- Aim for center of cornea
- Tip must be perpendicular to corneal surface
- MINIMAL PRESSURE!!
  - If you can see the cornea indent, you are pushing too hard → Falsely elevated reading
- Make sure final averaged reading has < 5% error

Interpreting the Tonopen

- Match reading with clinical signs
- 45 mmHg in a quiet, visual eye is likely false elevation
- Instrument has 2 mmHg built in error
- Have medications been given recently?
- Corneal surface
  - Severe keratitis may cause false elevation
  - Attempt to take pressure in clearest area of cornea
- It is much easier to obtain a falsely elevated reading than a falsely depressed reading
  - Lowest reading tends to be the more accurate

Scenario

- 5 year old MN Cocker Spaniel
  - Blind from acute glaucoma OD for past 4 months
  - Prophylactic tx OS and periodic tonometry rechecks OS
    - May: OS = 15mmHg
    - July: OS = 17mmHg
    - September = OS = 19mmHg
- Does this indicate disease progression?

Tonovet

- Rebound technology
- Probe bounces off cornea
- Pressure estimated by amount of reverberation of probe
- Accurate
- Minimal restraint
- No topical anesthesia
Goniolens

Normal vs. abnormal ICA

Medical therapy for glaucoma

Hyperosmotics

- Mannitol IV (20,25%) 2.2gm/kg *
  - 6 carbon sugar
  - Poor GI absorption
  - Excreted unchanged in urine
  - Onset 30-60 minutes; duration 6-10hrs
- Glycerin PO (Glycerin USP (1.25g/ml) *
  - Trihydric alcohol
  - Better GI absorption
  - Metabolized to carbohydrates, lipid, glucose
  - Onset and duration similar to Mannitol
### Drugs that increase aqueous humor outflow

- Miotics (direct, indirect acting parasympathomimetics)
  - Pilocarpine (0.5%,1%,2%,4%,8%) TID *
  - Pilocarpine (4% gel) once a day
- Demecarium bromide (0.25%) BID *
- Prostaglandin analogues
  - Latanoprost (Xalatan) once a day *
  - Bimatoprost
  - Travaprost

### Drugs that decrease aqueous humor production

- Carbonic anhydrase inhibitors
  - Methazolamide 2.2mg/kg BID-TID *
  - Acetazolamide
  - Dichlorphenamide (Daranide)
  - Dorzolamide (Trusopt) (2%) TID *
  - Brinzolamide (Azopt) (1%) BID
- Beta blockers
  - Timolol maleate (Timoptic) BID (.5%, 25%) *
  - Betaxolol (beta-1 specific)

### Combination drugs

- Timolol maleate + Dorzolamide = Cosopt
- Others

- Remember glaucoma drugs can have side effects even in the healthiest of patients!!

### ECFA emergency glaucoma treatment

- Mannitol 1g/kg IV over 20 min or Glycerin USP (1.25g/ml) 1.5ml/kg PO over 20 min
- Methazolamide 2.2mg/kg PO BID-TID
- Latanoprost (Xalatan) 1-3 X daily

- Demecarium bromide BID
- Timolol maleate 0.5% (Timoptic) BID
- Dorzolamide (Trusopt) TID
Glaucoma

A surgical disease or a medical disease?

Surgical treatment for glaucoma

• Diode laser cyclophotocoagulation
• Micropulse Laser Therapy
• Cyclocryotherapy
• Goniovalve procedure
• Intrascleral prosthesis *
• Enucleation *
• Ciliary body ablation *

*Permanently blind, painful eye

Transcleral laser cyclophotocoagulation

Endolaser Cyclophotocoagulation
Cyclocryothermy

Gonioimplant

Post op gonioimplant

Intrascleral prosthesis
Intrascleral prosthesis/enucleation

Primary glaucoma is a BILATERAL disease

- Permanently blind painful eyes are best treated surgically
- Prophylactic medical treatment of contralateral eye is beneficial
- Early intervention best chance of slowing progression of disease
- Long term prognosis is poor
- Client education is important

QUESTIONS?
Diagnosing the Acutely Blind Patient

Mark Bobofchak, DVM, DACVO
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Causes of Acute Vision Loss

- Pain
- Globe Retraction
- Elevated 3rd Eyelid
- Miosis
- Obstruction of Light Pathway
  - Corneal opacity
  - Cataract
- Hyphema/Vitreal hemorrhage
- Uveitis/Aqueous flare

Causes of Acute Vision Loss

- Retinal Dysfunction
- Detachment
- Degeneration
  - Detachment / PPA / SARD
- Failure of optic nerve transmission
  - Compressive lesion
  - Inflammatory sequelae
- Central Lesion involving Visual Cortex
  - Neoplasia
  - Encephalitis

Where do I start?

- When did the vision loss occur?
- Rapid or Gradual?
- Change in appearance to either eye?
- Breed?
  - Shih Tzus prone to retinal detachments
  - Terriers prone to lens luxation
  - Primary glaucoma breeds
- Other physical exam findings
  - Petechia
  - Lymphadenopathy
**Diagnosing the Acutely Blind Patient**

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Akron, OH

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**Is the Vision Loss Real?**

- True Vision Loss
  - SARD
  - Retinal Detachment
  - Acute Glaucoma
  - Optic Neuritis
  - Central Blindness

- Severe Vision Impairment
  - Pain causing 3rd eyelid elevation
  - Severe Corneal Edema
  - Severe Uveitis
  - Hyphema
  - Acute Cataract
  - Visual Hemorrhage

---

**Menace Response**

- Learned cortical response: *"Brain involvement required"*
- Not consistently present in puppies younger than 3 months
- Tap eyelids first to ensure eyelid paralysis is not present
- Avoid touching conjunctiva or creating air current
- Test different fields
- Inconsistent in cats and in stressed or stoic dogs

---

**Menace Response**

- Avoid air currents
- Move hand in front of eye from behind rather than moving hand toward the eye

- Intact with cataracts, corneal edema, early glaucoma, Uveitis

- Absent with retinal degeneration, retinal detachment, SARD, Optic Neuritis, Central lesion

---

**Dazzle Reflex**

- Bright light evokes a visible response
- Blinking
  - Moving away from light
  - Subcortical REFLEX
    - Does not require central vision processing
    - Present from birth
    - Useful when PLR cannot be assessed (Iris atrophy, Hyphema, etc.)
    - Use brightest light possible
      - Transillumination often not enough

- Positive indicates functioning retina, Optic nerve, and CN VII
Diagnosing the Acutely Blind Patient
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Pupillary Light Reflex
- Neur Pathway: Retina -> optic nerve -> midbrain -> CN III parasympathetic fibers.
- Direct vs. indirect
  - Useful when iris cannot be visualized
  - "Swinging flashlight test"
- Direct vs. indirect lesion
  - No response to direct light, constriction with light into contralateral eye

Cotton Ball Tracking
- Visual animals nearly always follow motion
- Well lit room
- Low stress
- Have patient look straight ahead first
- Test straight ahead as well as right and left peripheral vision
- Can be subtle
  - Eye deviation vs. full tracking of the falling object
  - BOTH are a positive response

Maze/Navigation Testing
- Useful to assess functional vision
- Elaborate maze or simple obstacles throughout the room
- Re-arrange obstacles and test in different light conditions
- Watch for avoidance of obstacles or extending head to sniff objects before reaching them as sign of vision
- PRA - Navigates well in normal room light, Appears impaired in dim light

Tonometry
- ALWAYS CHECK OCULAR PRESSURE WITH ANY ACUTE VISUAL LOSS
- Acute glaucoma eyes do not always show classic symptoms
  - Scleral injection
  - Hyphema
  - Corneal edema
  - Blepharospasm
Diagnosing the Acutely Blind Patient
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Electroretinogram
- Test to assess RETINAL function
- Tests retinal cell activity, NOT VISION
- Performed by Ophthalmologists
- Normal with Corneal opacities, cataracts, Vitreal Hemorrhage, Optic Neuritis, Central lesion
- Decreased/Absent w/ Retinal atrophy/Dysplasia, Retinal Detachment, SARD

Opaque Cornea
- Corneal Pigment
- KCS, Healed Ulcer, PUG
- Corneal Edema
- Endothelial Dysplasia, Corneal Ulcer
- Corneal Vascularization
- Corneal Ulcer, Immune Mediated Keratitis (Pannus)

Anterior Chamber
- Uveitis
  - Menace +
  - Dazzle +
  - PUL: Often difficult due to miosis/inability to visualize pupil
  - RubeoHyphopyon

Hyphema
- Findings depend on density of hyphema
- Menace ?, Dazzle ?
  - Trauma
  - Chronic cystoid
  - Chronic Uveitis
  - Acute retinal detachment
  - Neovascular
  - Systemic hypertension
  - Systemic clotting disorder
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Eye Care for Animals
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Anterior Chamber

- Hyphema
  - Work-up for any bleeding tendency
  - Petechia or ecchymosis?
  - Systemic Blood Pressure
  - CBC/Chemistry (Platelets)
  - Tick Panel (Ehrlichia, RMSF, Lyme)
  - Fungal Panel (Blastomycosis, Histoplasmosis, Coccidioidomycosis)

- Uveitis
  - Concurrent anterior uveitis and serum hyperlipidemia
  - Blood-aqueous barrier typically prevents large lipoproteins from entering eye
  - Rapid onset of "milky" appearance
  - Common Causes
    - Work-up: S lens extraction surgery
    - Dietary indiscretion (Too many hamburgers!!)
  - Typically responds to topical and systemic anti-inflammatories
    - Steroid or NSAID

Cataract

- Can be rapid onset
  - Diabetes Mellitus
  - Trauma
  - Heredity
  - Menace +, Dazzle +, Direct PLR +, Indirect PLR +

  Look close for signs of lens induced uveitis

- Start topical anti-inflammatories on all acute cataracts
  - NSAID or Steroid
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Vitreous
- Severe Astroid Hypnosis
  - Typically not acute
  - Can be severe enough to be blinding
- Vitreal Hemorrhage
  - Spontaneous
  - Systemic Hypertension
  - Retinal Detachment

Retina
- Retinal Detachment
  - Tear (Rheumatogenous)
    - Shih-Tzu
    - Post Intraocular Surgery
- Bullous (Non-Rheumatogenous)
  - Autoimmune
  - Systemic Hypertension
- Retinal Degeneration
  - PRA - slow onset, obvious retinal lesions
  - SARD - rapid onset, normal appearance

Giant Retinal Tear
Bullous Detachment

Optic Nerve
- Optic Neuritis
  - Isolated Optic Neuritis
  - Meningioma
  - Perineuritis
  - Neoplasia
  - Infection
  - Menace - Dazzle
- Optic Nerve Injury
  - Periphlebitis
  - Only 20% recover vision
Central Brain Lesion

- Normal Ophthalmic Exam
  - Menace -, Dazzle +, Direct PLR +, Indirect PLR +
- Fundus Normal
- Normal ERG
- Forebrain symptoms
  - Seizures, circling, Abnormal behavior/personality
- GME, Cerebral Hypoxia, Intracranial Neoplasia
- MRI usually needed to diagnose

Put in into practice…

- Patient presents for “acute blindness”
- Is “blindness” due to true defect in neurologic pathway or failure of light to reach the retina clearly?
  - Most pre-retinal causes of vision loss are treatable with fair-good prognosis
  - Retinal, optic nerve, and central causes have a guarded to poor prognosis

Conclusion

- Positive Menace, Dazzle or PLR = Vision Potential
- Some normally visual animals have negative test results
  - Test at multiple doses
  - Match test finding with clinical history
- Anti-inflammatories can improve vision in many cases
  - Topical for corneal or anterior chamber
  - Oral if visual cortical

Conclusion

- If positive Menace, Dazzle, or PLR but acting blind,
  - Painful and keeping eyes closed?
  - Ulcer, Corneal ulcers, Ulcers, Acute Glaucoma
  - Severe corneal or ocular discharge, exudate?
  - Pannus, Healing ulcer, Other corneal lesions
  - Opaque anterior chamber, lens, or vitreous?
  - Hyphema, Uveitis, Cataract, Visual Hemorrhage
  - Eye appears grossly normal?
  - Central Brain Lesion
Conclusion

- If negative menace, dazzle or PUP and acting blind,
  - Retina appears normal
  - SARD, Optic Neuritis, Acute Glaucoma
  - Retina Hypo reflective with thin blood vessels
  - PRA, Chronic Glaucoma

Questions?
Eyelid Disease and Surgery
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Basics
• Eyelid Functions
  • Protection
  • Tear film contribution
  • Tear film distribution
  • Prevent pathogen buildup
  • Clearing of foreign bodies

Anatomical Considerations
• Eyelids should sit without tension on the corneal surface and close easily and completely.
  • Typically, medial sclera should not be visible and small amount of lateral sclera visible when looking ahead.
  • Extensive vascular supply
    • Rapid healing
    • Intraoperative hemorrhage
• Eyelid skin typically thinner than other skin areas
  • Required for proper eyelid mobility
  • Important when considering partial thickness excision procedures

Congenital Anomalies
• Premature eyelid opening
  • Physiologic ankyloblepharon until 10-14 days
  • Severe ulceration possible
• Ophthalmia neonatorum
  • Bacterial infection beneath closed eyelids
  • Surgical opening of palpebral fissure and aggressive flushing
  • Topical antibiotics and lubrication
• Eyelid Agensis
  • Cats >> Dogs
  • Failure of a portion of the eyelid to form (Dorsal lateral most common)
  • Surgical correction if keratitis present
• Dermoid
  • Choristoma of ectopic skin in abnormal location
  • Eyelid, conjunctiva, cornea
  • Surgical removal (keratectomy) is curative

Eyelid Agenesis
Dermoid

Distichiasis

- Cilia arising from the meibomian gland openings
- Originate from the tarsal plate, 5-6 mm distal to the margin
  - Undifferentiated meibomian tissue
- English and American Cocker Spaniel, Miniature Longhaired Dachshund
- Often incidental finding

Distichiasis – Surgical Techniques

- Manual Epilation
- Distal Tarsal Plate Resection
- Cryoepilation
- Electrolysis
- Carbon dioxide laser ablation

Manual Epilation

- Performed with cilia forceps
- Easy (with magnification)
- High risk of re-growth
  - Presence of resistance when epilating indicates an active follicle – expect cilia re-growth
  - Useful to test if distichia are the problem

Cryoepilation

- Effective follicle destruction
  - -20º to -30º C
  - Fast freeze/slow thaw
- Minimal margin disruption
- Complications
  - Profuse swelling
  - Eyelid margin depigmentation
  - Rarely eyelid necrosis

Electrolysis

- Electrode inserted into distichia follicle
- Excessive current can cause scar tissue and eyelid distortion
- Hydrogen gas bubbles should be present when adequate current applied
- Indicated when few, single distichia present
**CO₂ Laser Ablation**

- Useful for few distichia
- Can cause excessive margin disruption if used for multiple cilia
- Destruction of meibomian gland
- Portable thermal cautery an alternative
- Protective eyewear and smoke evacuation required

**Ectopic Cilia**

- Variant of distichia – Cilia emerges through conjunctival surface
  - Middle of dorsal lid is most common location
- Typical signalment is young dog with intense blepharospasm
  - Often mistaken for indolent ulcer
- Surgical Technique
  - Incise conjunctiva around cilia
  - Often multiple hairs that must be followed back to their base
  - CO₂ laser ablation +/- Cryotherapy
  - Rapid, minimal hemorrhage

**Trichiasis**

- Normal eyelid hairs that deviate abnormally to contact the cornea
  - Primary or secondary
  - Often older dogs
- Stages Technique
  - Large ellipse of eyelid skin removed
  - Can close partially or leave completely open
  - Open wound granulates in and new tissue contains no hair follicles
Entropion

- Eyelid inverted
  - Normal cilia making contact with cornea
- Usually hereditary
  - Chow Chow
  - Shih Tzu
  - English Bulldog
  - Labrador Retriever
  - Rottweiler
  - Many others
- Surgery usually needed
  - Pugs & Pekingese
    - Medial entropion common
    - Surgery often not needed

Entropion

- Cicatricial
  - Excessive scar tissue from eyelid injury or disease
  - Relatively rare in animals
- Anatomic
  - Most common cause
  - Often due to macroblepharon or microblepharon
  - Usually identified in young dogs
    - Will occasionally grow out of the condition
- Spastic
  - Secondary to severe ocular pain

Temporary Tacking

- Useful for growing puppies
- Allows cornea to heal and minimize future fibrosis
- Allow puppy to mature before permanent surgery performed
  - Sutures
  - Staples
- Treatment for spastic entropion

Entropion - Concepts

- Evaluate amount of correction prior to anesthesia!!
- Topical proparacaine to remove spastic component
- Aim for perfection but if you must miss, err on slight undercorrection
  - Post-surgical tissue contraction
    - Easier to correct with second surgery than overcorrection
- With parallel skin resection techniques, important to remove skin and orbicularis oculi muscle

Parallel skin resection (Hotz-Celsius)

- Scissors or blade to make incision
- First incision – Parallel and 1-2 mm from eyelid margin
- Second incision depends on amount of correction required
- Important to remove deep muscle tissue and square off edges
- Single layer closure
Wedge Resection

• Important for entropion due to macroblepharon
• Full thickness wedge resection, usually from lower lid, taken near the lateral canthus
• Determine amount to remove while awake by using thumb to tighten the lid until it contacts the cornea correctly
  • The amount of eyelid that overlaps is the amount that must be removed
• Single layer closure

Figure 8 Eyelid margin suture

Ectropion

• Eversion of eyelid
  • Chronic epiphora, conjunctival hypertrophy, exposure keratitis
• Anatomic
  • Macroblepharon
  • “Diamond eye” breeds
• Cicatricial
  • Following trauma or overcorrected entropion

Ectropion

• Prone to recurrent conjunctivitis
  • Treat with topical steroids as needed
• Surgery often not necessary
  • Multiple described procedures
  • Combination of eyelid shortening and parallel skin resection works well

Wharton-Jones V to Y plasty

• Generally for cicatricial entropion or to correct overcorrected entropion surgery.

Macroblepharon

• Oversized palpebral fissure
  • “Normal” in many breeds
  • Most brachycephalic breeds
  • Mastiff
  • St. Bernard
  • Bloodhound
• Associated problems
  • Entropion/ectropion
  • Exposure keratitis from lagophthalmos
  • Pigmentation, vascularization, ulceration

Exposed medial sclera
Permanent Tarsorrhaphy

- Useful in brachycephalic breeds to prevent progression of pigmentary keratitis, correct medial entropion, and reduce chance of traumatic proptosis.
- Medial or lateral
  - Lateral is technically easier
  - Medial repair provides a more cosmetic appearance usually
- To spare or not to spare the puncta
  - Debatable
  - Few clinical problems with removing puncta
    - Added benefit of increasing tear contact time with cornea

Simple Reduction Canthoplasty

- Equal amount of eyelid margin removed from upper and lower lid
- Remove all caruncle tissue if medial
- Spare puncta if possible
- Angle incisions to allow for good apposition
- 2 layer closure
- Use temporary tarsorrhaphy to take tension off suture line

Blepharitis

- Immune mediated
- Bacterial
- Fungal
- Parasitic
- Nutritional
- Miscellaneous

Bacterial Blepharitis

- Staph Hypersensitivity
  - Acute hyperemia, swelling, and ulceration of eyelid skin
  - Often associated with meibomitis and chalazia
  - Systemic and topical antibiotics to treat as in pyoderma
  - Topical steroid to reduce swelling
- Blepharitis can be a part of Juvenile Pyoderma in puppies
  - Painful condition and self-trauma can exacerbate

Bacterial Blepharitis

- Meibomitis
  - Common bacterial infection of the meibomian glands
  - Most common bacterial isolate is Staphylococcus, but culture of meibomian secretions is ideal
  - Clinical signs
    - Mucoid Discharge
    - Swollen, white meibomian openings
  - Occasionally can cause vascular and ulcerative keratitis
  - Standard treatment here is Cephalexin for 3 weeks
Immune Mediated Blepharitis

- Allergic
  - Hyperemia and swelling following contact with allergen
  - Insect bite, environmental debris, drug-induced
  - Neomycin
  - Food allergy
- Pemphigus diseases
  - P. foliaceus, P. erythematosis, P. vulgaris
    - Inflammation and ulceration of m-c junctions
    - Autoantibody production against epidermal stromal matrix
  - Bullous Pemphigoid
    - Autoantibody production against epidermal basement membranes

Immune Mediated Blepharitis

- Uveodermatologic Syndrome (VKH-like syndrome)
  - Uveitis, poliosis, vitiligo, meningitis in people
  - Similar in dogs except for meningitis
  - Akita, Husky, Chow, Samoyed
  - Depigmentation, alopecia, and ulceration of eyelids, nasal planum, and lips
  - Atopic uveitis extensive panuveitis

  Tx: Systemic prednisone +/- azathioprine for life
  - Periodic CBC
  - Oral cyclosporine

Chalazion

- Granuloma due to leakage of meibomian secretions into surrounding tissues
- Usually yellow in appearance but can be pigmented
- CO2 or scalpel incision followed by curettage of contents
- In large numbers, may indicate qualitative KCS
  - Poor lipid secretion

Eyelid Masses

- Neoplasia
  - Canine
    - Vast majority are benign adenomas or papillomas
    - Adenocarcinoma, malignant melanoma, squamous cell carcinoma occasionally seen
  - Feline
    - Malignant forms more common
    - SCC, Fibrosarcoma, Lymphosarcoma, Mast cell tumor, Basal cell tumor, melanoma

Eyelid Neoplasia

- Surgical Options
  - Traditional excision (Wedge resection)
    - Recurrence rare
    - Proper apposition of eyelid margin important
    - If > 1/3 eyelid length, eyelid advancement flaps necessary
  - CO2 Laser ablation
    - Rapid, excelent cometic result, no sutures
    - Performed from conjunctival surface
    - Risk of recurrence if not aggressive enough
    - Not recommended for malignant tumors
  - Cryotherapy
The use of the carbon dioxide laser for the ablation of meibomian gland adenomas in dogs. JAAHA 2005; 41: 227-234

Questions???
The Canine Lens

Caution: This topic can get a little foggy

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Lens Development

• Surface Ectoderm
• Dependent on proximity to retina

Lens Development

• Anterior cells mitotically active
  • Form lens cortex
• Posterior cells elongate to form primary lens fibers = embryonic lens nucleus
  • Why this is important...
    • Cataracts confined to the embryonic nucleus are considered congenital and often will not progress to maturity

Lens Development

• Cortex formed from elongating anterior epithelial cells at the periphery
  • Form "Y-sutures" where they meet (dogs)
    • Anterior – Y
    • Posterior - Upside down Y
Adult lens anatomy

- Capsule
  - Thicker anteriorly
- Anterior cuboidal epithelium
- Mitotic cells for developing cortex
- Cortex
  - Grows throughout life
  - Epithelial cells elongate at periphery
- Nucleus
  - Adult
  - Fetal
  - Embryonic

Function of the Lens

- Final focus of light on the retina
  - Tear film, cornea, aqueous, and pupil contribute to focus
  - Tear film/cornea most important refractive structure (refractive index)
- Accommodate to change focus

Accommodation

- Humans/Primates/Birds
  - Alter lens curvature
- Cats/Dogs
  - Minimal ability to change lens curvature
  - Anterior and Posterior translocation
  - Reduced accommodative ability = decreased ability to change depth of focus
Cataracts -

- **Density**
  - Incipient
  - Immature
  - Mature
  - Hypermature
- **Location**
  - Capsular
  - Cortical (anterior/posterior)
  - Equatorial
  - Nasal
  - Nuclear
  - Suture
- **Age of Onset**
  - Juvenile
  - Senior
- **Cause**
  - Diabetes
  - Nutritional deficiency
  - Electrocution
  - Radiation
  - Metabolic (hypocalcemia)
- **Shape**
  - Punctate
  - Triangular
  - Intumescent
  - Linear

What is a cataract?

- Must first ask how a normal lens remains transparent
  - Lack of organelles and nuclei in lens fiber cells
  - Minimal refractive index fluctuation
  - Highly ordered lattice arrangement of fibers
  - Relative dehydrated state
  - High proportion of soluble proteins
- Any alteration of these conditions can result in opacification of the lens

What is a cataract?

- Most cataracts due to...
  - Disruption of normal fiber arrangement
    - Diabetes
    - Trauma
  - Increase in proportion of insoluble (albuminoid) proteins
  - Age-related cataracts
  - Hereditary cataracts
Cataract vs. Lenticular Sclerosis

• Retroilluminate
  • Cataracts cause shadowing effect
  • Lenticular sclerosis causes translucent “halo” effect
  • Fundus still visible with sclerosis
  • Vision changes?

Lenticular Sclerosis

Immature Cataract with Sclerosis

Breed Predispositions

• Many breeds susceptible
  • Boston Terrier
  • Pug
  • Toy Poodle
  • Bichon Frise
  • Miniature Schnauzer
  • Siberian Husky
  • Cocker Spaniel
  • Labrador Retriever
Management options

1. Do nothing
   - Incipient cataracts
   - No vision impairment
   - Not likely to progress

2. Medical management
   - Topical anti-inflammatory
   - Phacoytic soaks

Management Options

- N-acetyl carnosine
  - Ocuvet™, Bright Eyes™
  - Powerful antioxidant
  - Shown to disaggregate crystallins in vitro
  - 2006 preliminary study
  - Significant reduction of opacity for immature cataracts and nuclear sclerosis
  - 80% owners reported improvement in visual acuity

- Sounds great… except...
  - This was a preliminary study
  - My personal experience has shown little benefit with these drops

Cataract Surgery

- Phacoemulsification
  - Needle tip vibrates at ultrasonic frequency
  - Breaks up cataract
  - Vacuum to aspirate lens material
  - Irrigation of fluids to maintain eye pressure and cool the tip of the needle
  - Artificial lens
    - Rigid vs. Foldable
    - 415 Stippers – Dog
    - 53 Stippers – Cat

Cataract Surgery Facts

- Ideal stage is immature or early mature
- Early referral for evaluation
- 80-95% success rate for most dogs (1 year post)
- 70-75% success at 3 years post
- Post-op vision quality
  - Normal with IOL
  - Hyperopic (far sighted) if aphakic (no lens implanted)
- Cataract does not return in the dog or cat
  - Regrowth does occur in rabbits
  - Lens material can proliferate in periphery – not clinically significant
  - Fibrosis of the lens capsule can occur
Artificial Lens Implants

• Currently standard to implant lenses
• Rigid PMMA vs. Foldable Acrylic
• Seated in the lens capsule
  • Cannot implant if excessive zonule breakdown
  • Suturing lenses to sclera with capsule instability possible, but high risk of hemorrhage.
• 41.5 Diopters in dog, 53 Diopters in cats

Cataract Surgery Facts

• Diabetes Mellitus
  • Nearly 5% of operated cataracts
  • Very rapid onset
    • Increased blood sugar → Increased Sorbitol formation → Fluid accumulation within lens via osmosis
  • Lens intumescence (swelling)
  • Phacolytic uveitis
  • Spontaneous lens capsule or zonule rupture
  • Do very well with surgery if regulated
Complications

- Glaucoma
- Retinal Detachment
- Corneal ulceration
- Temporary ocular hypertension
- Endophthalmitis
- Capsule fibrosis
- Chronic uveitis
- Lens implant dislocation

Lens Luxation

- Primary
  - Hereditary zonule degeneration
    - Terriers, shar pei, border collie, chihuahua
- Secondary
  - Intraocular neoplasia
  - Chronic glaucoma
  - Severe trauma

Complications

- Glaucoma
- Retinal Detachment
- Corneal ulceration
- Temporary ocular hypertension
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- Capsule fibrosis
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- Secondary
  - Intraocular neoplasia
  - Chronic glaucoma
  - Severe trauma

Lens Luxation

- Subluxation
  - Partial zonule breakdown
  - Aphakic Crescent
  - Anterior chamber vitreous
  - Phacodensitis
    - "jiggling" of the lens when the eye moves
  - Treat with early surgery or miotics and anti-inflammatories
    - Pilocarpine
    - Demecarium Bromide
    - Latanoprost

Lens Luxation

- Posterior luxation
  - Dislocation of the lens into the posterior segment
  - Does not cause acute glaucoma spike
    - Glaucoma and/or retinal detachment common in the long run
  - Surgery can be more difficult
    - Must "fish" the lens out from the posterior segment
Lens Luxation

• Anterior Lens Luxation
  • Movement of the lens into the pupillary zone or completely into the anterior chamber
  • Clinical effects
    • Blepharospasm
    • Pupil dysconia
    • Lens equator visible
    • Focal corneal edema (endothelial touch)
    • Often pupillary block glaucoma
      • Axogenous cannot pass through the pupil to reach the iridocorneal angle
      • Severe, nonresponsive glaucoma

• Is this an emergency???
  • Yes…if glaucoma present and vision intact (positive menace, dazzle, and indirect PLR)
  • If chronic and no vision present, it should be referred soon, but not as an emergency
  • Enucleation recommended if vision not salvageable

• Treated with Intracapsular Lens Extraction
  • 180° corneal incision
  • Lens removed in one piece
  • Lens usually not replaced (aphakic)
  • Lens suturing techniques increase the complication risk
  • Vision is adequate for 90% canine activities
    • Blurry
    • Hyperopic (for right/left)

Lens Luxation

• Risks of ICLE
  • Persistent glaucoma
  • Patients remain on lifelong hypotensive therapy
  • Retinal detachment
  • Diode laser retinopexy often performed at the time of surgery to decrease risk
  • Progressive vision loss
    • Acute glaucoma spikes initiate cascade of retinal and optic nerve degeneration
    • Often continues even with normal intraocular pressures
To Grid or Not to Grid...
Corneal Ulcers and Other Confusing Corneal Conditions

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Topics

• Ulcers
  • Indolent
  • Stromal
  • Descemetoceles
  • Perforations
• Lacerations
• Autoimmune
• Dystrophy
• Degeneration

Indolent Ulcers

• "Boxer Ulcer", Non-Healing Ulcer, SCCE, REE
• Older Dogs
  • > 6 years old
• Typical history
  • Progressive "redness"
  • Cloudiness
  • Intermittent blepharospasm
  • Good days and bad days
  • Often no known trauma

Indolent Ulcers

• Failure of epithelial-stromal adhesion complex formation
  • NOT BM dystrophy
  • Other dogs
  • Normal BM in uninfected cornea
• Loss of BM
  • Normally cannot manually debride
  • Delays wound healing
• Stromal changes
  • Superficial acellular hyalinized zone
  • Barrier to adhesion complex

IHC for laminin
Success Rates

- 80-90% heal within 2 weeks with DBD or LGK
- < 50% heal rate for swab debridement
- Near 100% heal with keratectomy, but rarely necessary
- Continue topical antibiotics 2-3 X daily and oral pair medication (NSAID or Tramadol)
- E-Collar and Warm compresses

Red and Blue is good for you, Yellow or Green needs to be seen

Indolent

Medical Options

- Topical antibiotics
  - Epithelial cell toxicity?
  - Serum?
  - Ramend?
- EDTA
- Chondroitin Sulfate
- Cyanoacrylate
- Substance P
- PSGAGs
- MMP inhibitors?
  - Desoxynine

Surgical Options

- Debridement
  - Cotton Swab vs. Blade
- Linear Grid Keratotomy
- Diamond Burr Debridement
- Multifocal Superficial Punctate Keratotomy
- CO2 Laser Keratotomy
- Thermokeratoplasty
- Keratectomy

Stromal

Increased proteolytic enzymes
- MMP 2 and 9
- Inflammatory cells
- Epithelial cells
- Fibroblasts
- Bacteria
- Often rapidly progressive
- Varying degree of pain
- Breed

TREAT AGGRESSIVELY

Stromal Ulcers

Treatment Options

- Medical management
- Temporary Tarsorrhaphy
- Third Eyelid Flap
- Conjunctival Pedicle Graft
- Corneconjunctival Transposition
- Amniotic membrane / PRIS graft
- Frozen Cornea Graft
- Corneal Collagen Crosslinking with Riboflavin
Medical Management

• Antiprotease/Anticollagenase
  • Serum
  • EDTA
  • Doxycycline
  • N-acetyl-cysteine
  • Ilomostat (Galardin)

• Frequent dosing (q 1-2 hours initially then decrease)

• Systemic pain control
  • Tramadol
  • NSAID

Medical Management

• Culture/Sensitivity and Cytology
• Antibiotics
  • Ideally based on c/s – Not always practical
  • Gram stain
  • Drops preferable to ointments
  • Frequent dosing initially (q 1-2 hrs for several days)
• Mydriatic (Atropine)
  • Pain control by reducing ciliary spasm
  • Prevents synechia
  • Caution with KCS patients

Medical Management

• E-collar
  • IMPORTANT
• Exercise restriction
• Warm compresses
  • NOT for descemetocoles!!
• Frequent rechecks until cornea is stabilized and stromal thickness is improving

Medical

**Benefits**

• Cheap
• No anesthesia
• May have reduced scar

**Risks**

• May progress to perforation
• Frequent drops
• Dependent on owner compliance
• Variable time to healing
Surgical Options

- Temporary Tarsorrhaphy
- Third Eyelid Flap
  - Protect cornea during healing
  - Liken to bandaging an open wound
  - Not directly fixing the defect
  - Treat as for medical management

Conjunctival Pedicle Graft

- Effective technique
- Immediate tectonic support and blood supply
- Often leaves large scar
- Ideal for large, peripheral, or malacic ulcers

Surgical Options

- Corneoconjunctival Transposition
  - Clear cornea into defect
  - Thicker than CPG
  - Conjunctiva adhered to peripheral cornea
  - No immediate blood supply

Ideal for non-malacic, central corneal defects

Perforations

- Immediate surgery recommended
- Iris Prolapse into wound
  - Affects prognosis and difficulty of surgery
- If traumatic, evaluate for intraocular damage
  - Ruptured lens capsule
  - Retinal Detachment
- Surgical options
  - Direct suturing if laceration
  - Corneal or conjunctival graft
Autoimmune Disease

- Pannus (CSK)
  - German Shepherds
  - Grayhounds
  - Progressive vascularization and pigment
  - Ventrolateral limbus
  - Third eyelid
  - Mineral degeneration

- Cause unknown
- UV exacerbates
- Chronic topical steroids or CSA/Tacrolimus

Autoimmune Disease

- Pannus
  - Vascular ingrowth and CD4+ lymphocytes into anterior stroma
  - Epithelium unaffected
  - Cause unknown
  - UV exacerbates
  - Chronic topical steroids or CSA/Tacrolimus

Autoimmune Disease

- Superficial Punctate Keratitis
  - Dachshund, Shetland
  - Multiple punctate opacities
  - +/- ulcers
  - CSA/Tacrolimus
  - Corticosteroids

Autoimmune Disease

- Eosinophilic Keratitis (cats)
  - Progressive stromal vascularization
  - Multiple superficial white plaques
  - Fluorescein adheres to white plaques
  - Typically non-painful
  - Multiple eosinophils / plasma cells on cytology

Treatment
- Topical +/- systemic steroids
- Megestrol acetate as last resort
- FHV management ???

Autoimmune Disease
Corneal Dystrophy

- Hereditary central corneal mineral deposits
- Cholesterol
- Often bilateral and symmetrical
- Not painful
- No vision impairment unless advanced
- No response to topical tx
- CBC/Chemistry to r/o metabolic disease
- Keratectomy as last resort

Typical Breeds
- Beagles, Siberian Husky, Cavalier, Collie, Shetland Sheepdog, Airedale

Lipid Keratopathy

- Dense corneal crystallization due to systemic lipid disease
  - Hypothyroidism
  - Diabetes mellitus
  - Pancreatitis
  - Hyperlipoproteinemia
  - Postprandial plasma lipid elevation
- Unilateral or bilateral
- Avascular early
- Treatment
  - ID and treat underlying cause
  - Topical anti-inflammatories may worsen
  - Keratectomy as last resort

Corneal Degeneration

- Crystalline opacities due to corneal injury or disease
- Lipids, cholesterol, or calcium
- Corneal vascularization
  - May ulcerate
    - Stromal ulcers may develop as crystals slough in older dogs
- ID and treat underlying condition
  - Avoid topical steroids
  - 1% EDTA may help with calcium degeneration
  - Keratectomy if severe
**Endothelial**

- Dystrophy
  - Hereditary
  - Boston Terrier
  - Chihuahua
  - Dachshund
  - Starts temporal
  - Progresses to entire cornea
  - Non-painful and typically visual
- Degeneration
  - Progressive edema
  - Predisposing condition
    - Uveitis
    - Lens luxation
    - Anterior iris synechiae
  - Variable pattern

**Clinical signs**
- Corneal edema only
- Normal PLR
- Lack of scleral injection, blepharospasm, or vision deficits

**Endothelial Disease**

- Treatment
  - Treat underlying condition (degeneration only)
  - Topical 5% sodium chloride
    - Will not clear edema or stop progression
  - Decrease chance of recurrent ulceration due to bullae formation
  - Topical steroids
    - If underlying uveitis suspected
  - Thermkeratoplasty or Laser Keratoplasty

**Corneal Sequestrum**

- Unique to cats and horses
- Chronic corneal irritation
  - FHV
  - Entropion
  - Chronic ulcer
  - Iatrogenic (LGK)
  - Persian, Colorpoint
  - Melanin and degenerate corneal collagen
  - Variable vascularization and discomfort
Thank you for your time and attention!!

Questions??

Corneal Sequestrum

Treatment options

• Medical management
  • Sloughing of plaque
  • Timing unknown
  • Discomfort
  • Risk of perforation

• Keratectomy
  • Preferred
  • Controlled removal
  • Graft placement if necessary
  • Good visual outcome
Cranial Cruciate Ligament Ruptures – Extracapsular Techniques
Kevin Benjamino, DVM, DACVS-SA

Objectives
- Provide overview of the pathogenesis of cranial cruciate ligament (CrCL) ruptures in dogs
- Review case selection for extracapsular techniques
- Provide techniques for extracapsular stabilization for CrCL tears in the carefully selected patient

The incidence of cranial cruciate ligament ruptures affecting dogs in veterinary medicine is extraordinarily high and is the most frequently made diagnosis in dogs of all sizes. While we tend to see predilection in female, spayed large breed dogs (Labrador Retrievers, etc.) the incidence of is growing in dogs of all sizes. While this is the most common orthopedic condition, one would think that there would be more agreement in regard to the management. This is always debate surround the management of this condition from surgical methods to non-surgical therapy. This lecture is designed to give an overview of the pathogenesis of cranial cruciate ligament ruptures and provide some insight into the extracapsular techniques used to surgical stabilize the stifle. Which so many options, there is no wonder there is no one “cookie cutter” technique. Multiple factors must be considered when determining what is the best option for both the patient and owner.

To begin, one must have a basic understanding of the anatomy of the stifle and what the CrCL acts to counteract (force). The CrCL is an intra-articular ligament that courses from the caudomedial part of the lateral femoral condyle to the cranial intercondylar area of the tibia. The caudal cruciate ligament has a path that opposes the CrCL. The CrCL can be functionally divided into two segments – craniomedial band and the caudolateral band (name implies the position of the segments). While both segments are taut in extension, the craniomedial band is taut in flexion and the caudolateral band is lax in flexion. It is with some degree of frequency that we see partial CrCL tears, in which one of the bands (typically the cranial medial band) is ruptured. The function of the segments of the CrCL is as follows: cranial translation is offset by both bands, as is hyperextension. Internal rotation is offset by the craniomedial band.

When cranial translation is described in the canine patient, it is with some err because the it is not just a cranially directed force. The force generated when the limb is engaged is both a proximal and cranial force, which is termed a shear force. This shear force is directly related to the tibial plateau angle (TPA). Dogs (in comparison to other species) have a steep TPA, in general 25-30 degrees. TPAs in excess of 30 degrees is not uncommon with some dogs having limb deformities causing excessive angles (>40 degrees). In most cases, the TPA is a conformational/developmental issue, however previous proximal tibial growth plate injuries can predispose the dog to unilateral excessive TPAs. It has been shown that the force exerted on the CrCL in relation to an increasing TPA is not a linear relationship, however it is an exponential relationship when greater than 25 degrees. This relationship must be taken into consideration when the surgical technique is selected.

Factors that can cause a CrCL tear in the canine are multifactorial and most dogs present with chronic changes versus acute injury as it occurs in the human. Factors evaluated and deemed to be associated with CrCL tears in the dog are tibial plateau angle, ischemia/hypoxia of the extra-synovial CrCL, repeated trauma/overuse, structural changes (such as to the collagen), biochemical and cellular changes to the extracellular matrix. Other potential causes are obesity, hormonal influence, etc. There has been research attempting to evaluate the causative factors of CrCL rupture in the dog. In general, there are three types of tears that can occur in the canine: complete rupture and two variants of partial tears. Some refer to the groupings of partial tears as competent (remaining attached ligament give stability) and incompetent tears (remaining attached ligament does not give stability).

Once a CrCL tear is diagnosed, the next step is to determine what the best action is for the patient and the client. This lecture is not designed to debate conservative therapy versus surgical therapy or to look into the validity of “fringe” therapies. Surgery (in the appropriate anesthetic candidate) is the preferred management due to multiple factors such as progressive degenerative changes, continued trauma/injury to the other intra-articular structures such as the medial meniscus and cartilage surfaces. When attempting to make the decision of what type of procedure, one can divide the procedures into two basic groups: 1. Extracapsular techniques and 2. tibial osteotomy techniques.
The more research is carried out, the general consensus is that the osteotomy techniques (in particular the tibial plateau leveling osteotomy (TPLO)) allow a more complete recovery and the best chance at a normal gait. That being said, the extracapsular techniques still have their place in practice, and it is up to us to select the appropriate cases and owners for these techniques.

The surgical decision making is invariably based on three facets: 1. Anatomical concerns, 2. Patient concerns, and 3. Owner concerns. The anatomical concerns must be evaluated, as these can really set us up for success or failure – the tibial plateau angle must be evaluated radiographically. An excessive slope will cause excessive force on the repair and cause a higher complication rate. Whether the CrCL rupture is partial or complete can weigh in on the decision. Concurrent disease processes such as a medial patellar luxation (MPL) can also sway the clinician toward a particular technique. There are numerous ways to combine the therapy for both a CrCL rupture and patellar stabilization; this will be surgeon dependent.

Some patient concerns that may influence the decision-making process are age, activity of the dog, goals for recovery (i.e. agility, performance, or family pet), and concurrent disease such as obesity and endocrine disorders. All of these concerns must be thought through to put the patient in the best chance for recovery. Other considerations would be owner related to include their ability to restrict and rehabilitate their pet. Also, in the decision making is the financial aspect of these procedures.

The extra-capsular stabilization technique has been a mainstay in management of CrCL tears and has undergone many augmentations. The standard technique is generally performed with nylon suture/leader line and is placed on the lateral aspect of the joint and anchors from the fabella and courses cranially to the proximal tibial tuberosity. While this technique can be very effective in the smaller patient, it does have an increased failure rate as the weight and activity level of the patient increases. Another potential predisposing risk factor is an increased tibial plateau angle (greater than 25 degrees). As the angle increases, the force exerted on the implant increases.

It is known that in response to the suture being placed, the body will further develop scar tissue in a periarticular fashion, which will further stabilize the stifle. Due to continued tension placed on the implant, the suture will, at some point break or elongate. This is likely due to the placement of the implant and its variable length as it goes through various ranges of motion. Even though the implant may stretch or break, there is commonly enough scar tissue present that stabilizes the stifle.

The relatively recent introduction of isometric points for implant placement by Don Hulse and others has definitely improved our results with extracapsular repair and has been the hallmark of techniques involving Arthrex Fiberwire, Arthrex Tight Rope, and InTrauma IsoLock implantation. The intention of the using the isometry theory is that the strain place on the implant is the same in all phases of motion (compared to earlier techniques). This, in part, should decrease the rate of implant failure and improve the mobility of the stifle joint. Please reference the images in the lecture that label the femoral and tibial sites of implantation, however the F2-T3 or F2-T2 sites allow for least amount of strain on the implant.

The above-mentioned techniques that utilize the isometric point placement tend to have an increased success rate (in the appropriate patient) and decreased implant failure, due to decreased stress on the implant. Please see the lecture for the description of the implantation. This lecture is not intended to replace the courses offered by multiple companies and the author would recommend that these courses be attended prior to implementation of the techniques. More recently there have been some peer reviewed studies that compared extracapsular repairs (ECR) to the tibial plateau leveling osteotomy (TPLO) and the tibial tuberosity advancement (TTA) and have shown the TPLO to be better at producing a more normal gait (compared to the normal limb) compared to the other techniques. This being said, the extracapsular techniques can be a very good procedure for a certain group of dogs (smaller and lower activity) and owners, when finances are considered into the decision-making process. When the procedure is performed correctly, and the correct case is chosen, a very good outcome can be expected.
References:

Cranial Cruciate Ligament Ruptures – Osteotomy Techniques
Kevin Benjamino, DVM, DACVS-SA

Objectives
- Provide overview of the pathogenesis of cranial cruciate ligament (CrCL) ruptures in dogs
- Review case selection for osteotomy techniques
- Describe the differences between the common osteotomy techniques – tibial plateau leveling osteotomy (TPLO) and the tibial tuberosity advancement (TTA)

The incidence of cranial cruciate ligament ruptures affecting dogs in veterinary medicine is extraordinarily high and is the most frequently made diagnosis in dogs of all sizes. While we tend to see predilection in female, spayed large breed dogs (Labrador Retrievers, etc.) the incidence of is growing in dogs of all sizes. While this is the most common orthopedic condition, one would think that there would be more agreement in regard to the management. This is always debate surround the management of this condition from surgical methods to non-surgical therapy. This lecture is designed to give an overview of the pathogenesis of cranial cruciate ligament ruptures and provide some insight into the extracapsular techniques used to surgical stabilize the stifle. Which so many options, there is no wonder there is no one “cookie cutter” technique. Multiple factors must be considered when determining what is the best option for both the patient and owner.

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The more research is carried out, the general consensus is that the osteotomy techniques (in particular the tibial plateau leveling osteotomy (TPLO)) allows for a more complete recovery and the best chance at a normal gait.

The surgical decision making is invariably based on three facets: 1. Anatomical concerns, 2. Patient concerns, and 3. Owner concerns. The anatomical concerns must be evaluated, as these can really set us up for success or failure – the tibial plateau angle must be evaluated radiographically. An excessive slope will cause excessive force on the repair and cause a higher complication rate. Whether the CrCL rupture is partial or complete can weigh in on the decision. Concurrent disease processes such as a medial patellar luxation (MPL) can also sway the clinician toward a particular technique. There are numerous ways to combine the therapy for both a CrCL rupture and patellar stabilization; this will be surgeon dependent.

Some patient concerns that may influence the decision-making process are age, activity of the dog, goals for recovery (i.e. agility, performance, or family pet), and concurrent disease such as obesity and endocrine disorders. All of these concerns must be thought through to put the patient in the best chance for recovery. Other considerations would be owner related to include their ability to restrict and rehabilitate their pet. Also, in the decision making is the financial aspect of these procedures.

The osteotomy procedures have become more of the gold standard for CrCL stabilization over the past 20 – 25 years when Barclay Slocum introduced the tibial plateau leveling osteotomy to mainstream veterinary community. Over time both the procedure has been honed and the implants have been improved upon, however the concept has remained the same. Over the last 10-15 years, the tibial tuberosity has gained popularity within the veterinary community as another good option. Both procedures address the cranio-proximal shear force directly and in general have a favorable outcome.

The tibial plateau leveling osteotomy (TPLO) was designed to counteract the shear force present in the CrCL deficient stifle by anatomically changing the tibial plateau angle (TPA). The idea is that if the TPA is reduced (goal of 6 degrees) adequately, this will neutralize the force that was exerted on the formerly intact CrCL. In the canine patient this seems to work due to the shear force being the major force that is counteracted by the intact CrCL. In essence, the TPLO creates a joint that does not need the CrCL. How the TPA is reduced is via a circular cut positioned under the tibial joint surface. The TPA is rotated and stabilized with a plate and screws to allow “fracture” healing. In some recent studies this procedure seems to offer the best option in regard to return to function at 6 and 12 months post-operatively.

The tibial tuberosity advancement (TTA) is a different procedure that introduced the concept of advancing the tibial tuberosity and in effect the patellar tendon attachment cranially in order to engage the quadriceps to counteract the cranio-proximal shear force. The advancement is performed by a proximo-distal bone cut into the tibial tuberosity and applying a wedge or cage that will move the tuberosity cranially a predetermined length. In addition to the cage, a plate and screws placed as a tension band is placed. The shear force is neutralized in a normal standing angle.

Both of the abovementioned procedures are thought to be superior to other techniques and have become available for dogs of varying sizes, even down to the toy breed dogs. Also, both procedures allow the surgeon to correct for other issues such as a medial patella luxation with minor alterations of the osteotomy. Both procedures tend to have some level of invasiveness (both require cuts into bone). These procedures do require advanced coursework and training to perform at a proficient level and come with an investment both in time and finances. The clinician must be prepared to undergo a learning curve which will mean some complications along the way.

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References:
Objectives
- Review the current treatment options for preemptive procedures that address hip laxity
- Review the current treatment options for salvage procedures that address hip laxity
- Provide an accurate expectation for outcomes of the common hip dysplasia procedures

Chronic hip dysplasia still a common disease process that affects our canine patients of all sizes, despite efforts to educate the general public and breeders as to its hereditary nature. Hip dysplasia refers to laxity that affects the coxofemoral joint and can vary in severity. It is generally thought that the contributing factors that affect the overall stability or laxity present within the joint include the ligament of the femoral head, joint capsule, dorsal acetabular rim, and supporting musculature such as the gluteal muscles.

While hip dysplasia is a genetically linked disease process, the environment certainly contributes to the expression of this disease. Environmental factors would include patient growth, obesity, and rate of development. All of these factors during the first 12 to 18 months of growth can greatly influence the gene expression. Hip dysplasia is generally considered to be biphasic in its presentation in the canine patient. Young patients that are affected are usually uncomfortable due to the degree of hip laxity present and older patients are usually affected by the progressive amount of degenerative changes present (i.e. peri-articular osteophytes, cartilage wear, etc.). There is certainly overlap between these two manifestations of hip dysplasia.

The focus of this lecture is to provide current guidelines for the surgical treatment of hip dysplasia and give the clinician confidence when making these recommendations to their clients. It is imperative for the veterinary community to re-establish early screening for hip dysplasia as a way to provide the absolute best options for their patients and to improve overall patient care. The main surgical options can be divided into two groups: 1. Pre-emptive surgical procedures and 2. Salvage (end stage) procedures. In order to be a candidate for preemptive procedures (juvenile pubic symphysiodesis and double pelvic osteotomy), the patient must be identified as a juvenile prior to the development of degenerative changes. The advantage of these types of procedures is that the goal is to maintain the patient’s own coxofemoral joint and reduce the amount of laxity. The salvage procedures (total hip replacement and femoral head ostectomy) can be done when the patient is uncomfortable, and quality of life is compromised.

Juvenile Pubic Symphysiodesis (JPS)

The juvenile pubic symphysiodesis is a preemptive procedure that in theory exploits the growth potential of the pelvis. The main goal is to fuse the pubic symphysis, thus allowing the rest of the pelvis to continue growing. This allows the dorsal acetabular rim to increase its coverage of the femoral head, decreasing subluxation. The timing of this procedure is very important. The ideal age is approximately 4 months of age (no greater than 6 months) in order to take advantage of the open growth plates and growth potential. The unfortunate part is that it is rare to identify the appropriate candidate due to the small time window. The procedure itself is a straightforward one as the pubic symphysis is exposed and generally cauterized to initiate early closer. Risks associated with this procedure are minimal as no implants are used and the size of the pelvic canal is maintained.

Double Pelvic Osteotomy (DPO)

The double pelvic osteotomy is another preemptive procedure that attempts to improve coverage of the dorsal acetabular rim by directly rotating the acetabulum laterally. An older version of this procedure is the triple pelvic osteotomy (TPO) which utilizes cuts along the pubic, ischium, and ilium and a pre-angled plate placed on the ilium. The DPO utilizes only cuts in the pubis and ilium and maintains the ischium. The advantage of the DPO versus the TPO is decreased complications such as narrowing of the pelvic canal, urethral compromise (due to placement of the ischial cut), decreases implant failure due to a more stable pelvis.
The selection criteria to select the proper DPO candidate can be limiting, however by adhering to these criteria one can improve the success of this procedure. The criteria are listed below:

1. Age – usually 5 -12 months
2. Joint laxity present (positive Ortolani)
3. Clinical signs are mild (more severe clinical signs generally fail other criteria)
4. Absence (or near absence) of degenerative changes to the hip on radiographs
5. Measurement of the dorsal acetabular rim (0-7.5 degrees) measured on radiographs
6. Goniometer measurements of angle of subluxation (10-20 degrees) and angle of reduction
7. Distraction Index (PennHip style radiographs) 0.5 – 0.9
8. Arthroscopic changes of ≤ grade II cartilage wear

In the properly selected patient this procedure can be very successful at maintain a more normal coxofemoral joint long term. The best practice is to be screen patients at an early age, ideally at 6 months of age as the treatment window is narrow (but larger than the JPS). For those patients exhibiting radiographic evidence of laxity and having a positive Ortolani evaluation further investigation into candidacy should be recommended. This procedure has a very good success rate in the properly selected patient and can avoid the need for future procedures as will be mentioned below.

Total Hip Replacement

The total hip replacement procedure has become the gold standard procedure of surgical treatment for hip dysplasia in the skeletally mature patient. Once thought of for the large breed dog, there is now a wide array of implants for varying sizes of patients. The total hip replacement (THR) provides replacement of both the acetabular and femoral head aspects of the joint. The most commonly used implants are produces by BioMedtrix and Kyon companies and range from cemented implants to press fit (biologic ingrowth) implants. The Kyon implants combine a biologic ingrowth technology with screw stabilization in the femoral component.

Criteria for case selection is very wide and include most cases with hip dysplasia and degeneration joint disease since both surfaces are replaced. For the skeletally mature patient this procedure allows for the most functionally normal hip. The overall success rate is between 90-93%. Of the patients that have bilateral pathology only about 15-20% of patients require both sides to be addressed, meaning the other 80-85% of cases compensate well when only one hip replacement is performed. Potential risks would include hip prosthesis luxation, femoral fracture, infection, subsidence of the femoral stem, among other complications.

Most pets with a THR require an 8-12 week recovery process and must follow a fairly restrictive post-operative process. The overall success should be very good in those cases that do not experience complications. In those patients’ experiencing complications, some are amenable to further surgery or medical management and some may need the implants removed, reverting the hip joint to a femoral head ostectomy.

Femoral Head Ostectomy (FHO)

A true salvage procedure, the FHO is a procedure that involve removal of the femoral head and neck. The joint capsule is repaired, as this is incised, and the hip becomes a “false” joint. The articulating surface is removed decreasing the discomfort that was previously present. Unfortunately, since the joint is functionally removed there are chronic changes that remain persistent such as decreased range of motion and muscle atrophy. It is very important that the patient is put through an extensive rehabilitation program post-operative to maximize the results of this procedure. In most patients, complete weight bearing on the surgery limb will not be achieved, however our hope is that the patient is more comfortable than the pre-operative limb. The indications for the FHO are hip dysplasia, femoral neck/acetabular fractures, luxation, etc.

Medical Management

Medical management for hip dysplasia (or conservative/non-surgical management) can be used in patients that are less severe or are not ideal candidates for surgery for various reasons. The goal of non-surgical management is multi-fold to decrease inflammation, improved muscle mass/muscle balance and to attempt to maintain cartilage that may still be present.
The major modes of therapy include drugs, supplements, intra-articular injections, rehabilitation and other modes such as acupuncture, etc. These modes of therapy can have varying levels of success for our canine patient and are similar for other joints that experience degenerative changes. More detail regarding the above options will be outlined in the accompanying lecture.

Overall, chronic hip dysplasia is a very treatable issue in our canine patients. It is important that every patient is evaluated thoroughly, as not all experience the same level of discomfort and recommendations may vary from patient to patient. Both physical examination, complete history, and radiographs are required to get a complete overview of the patient’s situation. The author feels that early (juvenile) screening is imperative to allow the clinician to make the best treatment plan for the patient. Also, during the physical examination, complete evaluation of the pelvic limbs is needed to assess for any other orthopedic conditions.
Medial Patella Luxation Management in the Small Breed and Large Breed Dog
Kevin Benjamino, DVM, DACVS-SA

Objectives
- Improve the clinician's overall assessment of patients (small breed) affected by medial patella luxation
- Describe the use of pre-operative radiograph when assessing both the femur and tibia in cases of medial patella luxation
- Review common surgical procedures used for stabilization of patella luxations

Medial patella luxation in the small breed dog is a very common orthopedic condition. In many cases (80-85%) this condition is bilateral in nature and the cause is mostly congenital/hereditary, while traumatic medial patella luxation can occur this comprises a small percentage of cases. The most common dogs affected are the small and toy breed dogs, however other breeds and medium/large dogs can be affected with the etiology being very similar – hereditary. Also, in many skeletally mature dogs with medial patella luxation (MPL), concurrent cranial cruciate ligament rupture can be seen. Careful assessment is needed for both diagnosis and treatment of these patients.

Medial patella luxation is a noted in patients where the patella does not sit appropriately within the trochlear groove and tracts medial to the medial femoral trochlear ridge. The degree of luxation and positioning of the distal femur and proximal femur dictate the grade of luxation. In the end of the day, the femoral and tibial conformation dictate the placement of the patella, due to the forces applied to the patella. Listed below is the grading scale used for these patients.

Patella luxation grading scale:

**Grade I/IV** – The patella may be luxated manually with the joint in extension. Reduction occurs when digital pressure is released. Many times, an incidental finding with occasional lameness.

**Grade II/IV** – Patella is easily manually luxated, especially with rotation of the foot. Reduction occurs with flexion and opposite rotation of the foot. The patella luxates spontaneously intermittently.

**Grade III/IV** – The patella is permanently luxated, manual reduction is possible, but reluxation occurs when pressure is released, or the stifle is flexed. Lameness ranges from weight bearing lame to non-weight bearing.

**Grade IV/IV** – The patella is permanently luxated and cannot be manually reduced. The limb is held non-weight bearing if unilateral or the patient moves in a crouched posture if bilateral.

The assessment of patients with medial patella luxation is very important. Most patient will have a varying degree of lameness and in grade I and II patients the lameness may not be appreciated during a gait evaluation. In these patients it is very important to assess the muscle mass in the hind limbs as this may give the clinician a clue as to which limb (or both) are affected. The classic gait is an intermittent non-weight bearing lameness combined with a normal gait, however this is more likely in the grade I and II patient versus grade III and IV.

The next physical examination assessment is direct palpation of the stifle and patella. It is imperative that the clinician assess for stifle instability (cranial drawer) as it is not uncommon for patients to have a concurrent cranial cruciate ligament rupture. During palpation of the patella, it is important that the limb is placed in both flexion and extension, as the patella will be more relaxed in extension of the stifle. Both medial and lateral pressure needs to be applied to the patella to assess for instability. Also, it is very important to applied internal rotation force, as well as external rotation force of the tibia during assessment. This will exacerbate a patella luxation. Based on the degree of luxation a grade can be applied for further characterization. In some patients that are anxious, sedation can be used during the examination.
The next step to diagnosis and further characterization of patella luxations would be sedated radiographs. The author views these as being an important part of assessing the true issue surrounding the patella, which looks at patellar alignment and anatomical variations of the femur and tibia. In some respect, the patella is the consequence or the effect (cause and effect model) of abnormal angulation of the femur and/or tibia, as this places abnormal stress on the attachments of the patella. The main attachments of the patella include the quadriceps mechanism, patellar ligament, and the parapatellar ligaments. In many cases the distal femur is implicated as a cause for excessive medial force on the patella – femoral varus deformity. Normal varus angles have been identified in larger breed dogs. Other structural deformities of the femur include external rotation of the distal femur and proximal femoral changes that alter the normal version (anteversion/retroversion) of the femoral head and its articulation with the acetabulum.

The patella can also be implicated as playing a role on medial patella luxation. One can see rotational deformities and tibial valgus/varus deformities. In many cases, at the very least there is some medialization of the tibial tuberosity present. Another reason to take radiographs of these patients is to assess the positioning of the patella in relation to the femoral trochlear groove (alta – higher and baja – lower). Based on the above accurately positioned radiographs are recommended in all patients with medial patella luxation, especially those with a grade III or higher, if not all patients. This can also show evidence of possible cranial cruciate ligament disease (excessive periarticular osteophytes, excessive joint effusion, and cranial translation of the tibia).

The standard recommended views include the following: lateral of the tibia to include the stifle and hock (standard tibial plateau leveling osteotomy radiographs), lateral projection of the femur including the coxofemoral joint and stifle – care is taken to align/superimpose the distal femoral condyles in both views. The other views recommended include a cranial-caudal projection of both the femur and tibia, in separate projections. This is used to assess both bones in the frontal plan – looking for varus/valgus and rotation deformities. For a more accurate assessment of femoral torsion a “sky-line” view can be taken as demonstrated in the lecture, however this is a particularly challenging projection. The author recommends these pre-operative radiographs in order to give the surgeon a plan to be executed while in surgery and it certainly allows the clinician to ability to counsel the owner appropriately as to which procedures likely need to be performed. In patients with excessive angular changes, corrective tibial and/or femoral osteotomies may be needed. This is more common in the medium and larger breed dogs, however, can be needed in the small and toy breed dogs.

The most common procedures to be performed in the patient with a medial patella luxation are described in detail within the lecture. The scope of the lecture is to focus on the most common procedures and to provide guidance as to when most advanced procedures may be needed, such as corrective osteotomies. The selection of the common procedures are made on a case-by-case basis for the individual patient.

The first point of assessment is the femoral trochlear groove (after evaluation of the intra-articular surfaces). It is necessary to access the depth of the groove and look for wear patterns on the medial trochlear ridge. Radiographs can demonstrate a shallow groove when evaluating the lateral stifle projection. If the groove has adequate depth, then the groove does not need to be altered (this is not generally the case). There are three main techniques used to augment the trochlear groove. The most common techniques are the block recession/trochleoplasty and the wedge recession/trochleoplasty. Both of these techniques try to salvage the cartilage surface for the patella to track. The block trochleoplasty is more challenging in the small patient and if a pneumatic saw is not available. In most small breed dogs, the wedge trochleoplasty is the easiest to performed and this is performed via a Hobby saw and rasp/file. In any technique the width of the recession needs to be a little wider than the widest point of the patella. The depth of the groove should be deep enough to allow the patella to sit appropriately with the subchondral plate sitting below the trochlear ridge (better seen on radiographs). The last technique would be the sulcoplasty. When this is performed the cartilage is not maintained and the cartilage and bone are removed, and a rasp/file is used to smooth out the underlying surface. The hope with this procedure is for fibrocartilage to develop, and in many cases this occurs, however there are some cases where scar tissue or other fibrous material develops in the groove. More recently, work has been done on the patellar groove replacement (PGR) by Kyon and this shows promise, especially in cases where there is no cartilage present.
Another grouping of standard procedures includes soft tissue augmentation. The most common would be a medial desmotomy/medial joint capsule release and lateral imbrication. In most cases, a similar medial joint incision is made much like on the lateral aspect. This can decrease the medial force exerted on the patella. Lateral imbrication can be performed to exert more force laterally on the patella. It is important that appropriate suture is used (and size) and suture pattern used, such as “vest-over-pants” or mattress suture patterns. By doing this procedure allow, relaxation is very common. The author typically performs these techniques after address the trochlear groove and tibial attachment.

The last of the common techniques to address medial patella luxation involves address the attachment site of the straight patellar tendon. In many cases the tibial tuberosity is more medial than desired. The goal of this procedure (tibial tuberosity transposition) is to lateralize the attachment point. The reason to move the tibial tuberosity is to allow quicker healing via the metaphyseal bone present. The tibial tuberosity is cut from proximal to distal, with care taken to not fully cut the distal cortex. If the distal cortex can be spared and the proximal portion of the tibial tuberosity lateralized the cut segment with be much less susceptible to complications such as fracture. The proximal segment is generally mobilized 3-5 mm, depending on the size of patient and the amount of correction desired. The tibial is stabilized by the use of a K wire and possibly tension band. There are some newer techniques that show promise for faster healing and less risk to the patient that exploit the elasticity of bone. Care must be taken when performing this procedure as well as exercise restriction for about 6 weeks post-operatively to allow this bone cut to heal appropriately.

The identification and surgical management can be both challenging and rewarding for the clinician. The complex of medial patella luxation is multi-factorial in most patients and care must be taken to think through each case as an individual case versus a “one size fits all” mentality. When one reaches this point in thinking, complications and risks can be minimized and success maximized. As mentioned above there are cases that may benefit from more aggressive procedures that address more of the underlying issues such as femoral and tibial deformities and these too can be rewarding.
Perfect Your Examination: Pelvic Limb Lameness
Kevin Benjamino DVM, DACVS-SA

Objectives
- Aid in developing a systematic approach to evaluating issues with the pelvic limb
- Review the physical examination from the digits to the hips
- Review of the basic neurologic evaluation

Introduction
One of the key ways to arrive at an accurate diagnosis is the successful completion of a physical examination. It is the examination that allows us to localize injury and disease. While not all examination findings are pathognomonic for a specific disease process, the presence of pain, swelling, etc. can lead us to our next step in the diagnostic process. In order to reach a diagnosis, there is a series of steps that must be taken. As the clinician gains experience and confidence, this series of steps may be lessened, however the physical examination must never be omitted. The physical examination is just as (or more) important as the clinical history and diagnostic tests. This lecture is designed to give a systematic review of common injuries as they pertain to the hindlimb and is in no way exhaustive. Also, fracture types and repairs are not reviewed – this only reviews the evaluation of the limb.

Physical Examination
When one starts their physical examination the absolute first step is a gait evaluation. In many cases the gait examination should be carried out by a technician or owner and the clinician should evaluate the animal both walking away and towards, as well as from both sides. This allows the clinician to start by identifying the problematic limb(s). It is important that the patient be walked at varying speeds as this may exacerbate a particular condition. From a gait evaluation, one can discern whether this is a neurologic or orthopedic ailment and develop potential differential diagnoses even based off of the gait analysis alone.

Once the affected limb is identified, evaluate the patient at a stand and first evaluate the unaffected contralateral limb (if unilateral disease). This assessment gives us a “control” for comparison. The physical examination starts first by evaluating for any obvious swelling to joints or long bones. Next, start with the distal-most aspect of the limb – the digits. Evaluate the nail beds for changes – swelling, drainage, or abnormal nail growth. Palpate the digits, as well as, flex and extend the phalangeal joints. In this location, palpate the sesamoid bones for signs of pain and discomfort.

It is important for the examiner to begin at the digits and evaluating each for abnormalities. Once these are evaluated the next major region would be the metatarsal bones. It is important to evaluate each for evidence of pain or swelling. Fractures can occur at this location; however, the history generally will support this finding. A more common injury at this location would be tarso-metatarsal luxation/subluxation. This injury, trauma induced, occurs when the supporting ligaments are torn, or can propagate from proximal metatarsal fractures. Most of these injuries will also coincide with swelling in this area. Other fractures in this area – tarsal bone and calcaneus can occur and usually have a similar traumatic history. If trauma is suspected in this region, it is important that radiographs are taken of this area. It is strongly recommended that the patient is sedated or anesthetized for full evaluation and radiographs. When ligamentous injuries are suspected, stressed radiographs are necessary for a diagnosis to be made. The views should include stressed viewed in both the lateral and cranial-caudal projections.

Another region that can cause lameness due to pathology is the common calcaneal tendon and nearby structures. In short, the common calcaneal tendon consists of three tendons that insert on the tuber calcanei. The three tendons are the gastrocnemius tendon (largest), the common tendon of the biceps femoris, semitendinosus, and gracilis muscles, and the tendon of the superficial digital flexor muscle. Any or all of these tendinous structures can be damaged. Avulsions from the tuber calcanei can occur, in which case usually the above structures minus the superficial digital flexor tendon (SDF) are avulsed. Partial tears/avulsions can also occur. These injuries can be either due to acute trauma or chronic in nature.
Common calcaneal tendon tears generally occur in sporting and hunting dogs either due to trauma or chronic, repetitive injury. Both the Doberman Pinscher and Labrador Retrievers have been over-represented for chronic injuries with no inciting cause and can be bilateral. In chronic lesions, some patients that will have a “bear claw” appearance to the digits due to the over-riding force of flexion from the intact SDF tendon. Swelling is always present and can be compared to the opposite limb. If one sees these classic signs, but the common calcaneal tendon palpated normally, careful evaluation (and radiographs) of the proximal insertion of the gastrocnemius needs to occur. Tears/avulsions proximally can happen in the canine patient.

Some patients can develop a luxation of the SDF tendon. This can sometimes be a more challenging injury to diagnose and is usually associated with sizeable swelling over the common calcaneal tendon. Unlike common calcaneal tendon tears, this swelling is fluctuant, which develops from the synovial bursa that exists. Lateral luxation is more common than medial. This condition is seen more commonly in the Shetland Sheepdog breeds but is not exclusive to them. Lameness can definitely range in severity. Palpation and manipulation of the SDF should be performed in extension and flexion.

Another nearby joint that needs to be evaluated for irregularities is the tibio-tarsal joint. Acute traumatic and chronic traumatic injuries can occur causing varying degrees of lameness. In acute trauma cases, normal positioned radiographs can appear normal, necessitating sedated stressed films (medial and lateral stress). At times comparing radiographs from the contralateral limb is very helpful. The main ligamentous structures are the medial and lateral collateral ligaments that attach to the tibial malleolus (medial) and fibular malleolus (lateral). The ligaments attach distally on the tarsus and proximal metatarsal region and have both a short and long segment (understanding is important when repairing). In chronic repeated trauma, injury can be seen, generally affecting the medial collateral ligament – short band. Radiographs will generally show osteoarthritic changes to the tibio-tarsal joint. Another condition that can be seen in the tarsus is osteochondritis dissecans of the medial talar ridge. This is a congenital abnormality and in the juvenile patient may be seen as a radiolucent defect on radiographs and in the mature patient as degenerative changes to the joint. There is also joint effusion present in most juvenile patients and fibrosis in adults.

Moving proximally, from the tarsal joint, it is important that the tibia is palpated for any signs of discomfort along the metaphyseal and diaphyseal regions. The same palpation should be performed for the femur. Common conditions affecting these regions would be inflammatory conditions such as panosteitis (juvenile patients) and neoplasia (bi-modal in terms of age ranges). There are certainly other, less common, issues that can occur. The proximal tibia is a common place for physeal fractures to occur in the juvenile patient, including tibial tuberosity avulsion fractures and tibial plateau fractures. Surgical stabilization is recommended for optimal results and decreasing the risk of angular limb deformities.

Conditions of the stifle (intra- and extra-articular) make up the most common injuries and conditions of all joints. Traumatic injuries of the stifle joint can occur and are somewhat beyond the scope of this lecture. They are typically multi-ligamentous in nature and require advanced surgical techniques. When assess the stifle in the high trauma patient, it is best to do so under sedation and evaluate the stifle in all phases of motion and cranial-caudal, caudal-cranial, medial and lateral positions.

It is important to carefully evaluate the stifle joint when a patient presents for hind limb lameness. The most common condition affecting the canine patient is injury to the cranial cruciate ligament. In most canine patients this condition is a chronic in nature. The etiology of cranial cruciate ligament tears has been well evaluated and studied. Most patients will have a variable amount of lameness, from mild weight shifting of the limb to non-weight bearing lameness. Some of this depends of the acuteness of the injury, degree of tearing of the ligament, and other concurrent injuries such as medial meniscal tears. Cranial cruciate ligament ruptures affect dogs of all ages and breeds. Historically it has been documented to be over-represented in the Labrador Retriever and Rottweiler breeds, however all breeds are affected. The cranial cruciate ligament can be either partially or completely torn. Patients who develop a complete tear have an increased risk of concurrent meniscal injury. Dogs that develop a cranial cruciate ligament rupture do have an increased risk of rupturing the contralateral limb at some point in their life and a smaller number of patients will present with concurrent bilateral cranial cruciate ligament tears.
Evaluation for a cranial cruciate ligament tear is a technique that will be needed throughout any small animal veterinarians' career. It is important to develop a systematic technique. Palpation of the joint is the first step in evaluating for joint effusion and using the contralateral joint for comparison. In patients where the ligament is torn (even partial) there will be discomfort on hyperextension of the stifle. If the meniscus is involved, pain on hyperflexion is a common finding. Also, in some patients, the limb will be extended when the patient assumes a sitting position, due to discomfort when placing the stifle in flexion. One of the hallmark signs associated with cranial cruciate ligament ruptures is called cranial drawer. Since the cranial cruciate ligament offset cranial tibial motion, in patients that have a ligament rupture, the tibia can be manipulated and moved in a cranial direction in relation to the femur. It is important to perform this technique in various ranges of motion. Partial tears may only have motion in flexion. Another similar technique used to evaluate for a cranial cruciate ligament rupture is looking for tibial thrust. This is where the examiner is looking for motion of the tibia, exploiting tibial shear force due to the dog’s increase tibial plateau angle. The stifle and hock are place in 90 degree angles and proximal force is exerted from the distal limb below the hock. If the cranial cruciate ligament is intact then no movement will be appreciated. If a tear is present, then the tibia will move cranial and proximal. These techniques confirm this condition and radiographs allow us to rule out other condition, show secondary changes, and allow us to measure the proximal tibia for certain stabilization techniques.

For further evaluation of the stifle one should also assess for less common injuries such as damage to the caudal cruciate ligament and damage to the collateral ligament structures. Both will have motion in their respective directions – caudal cruciate ligament rupture will propagate caudal motion of the tibia in relation to the femur, collateral ligament ruptures will allow instability and joint widening. Radiographs are strongly recommended when diagnosing these conditions.

Another common condition to affect our canine patients is medial patella luxation (MPL). The most common signalment is the young/juvenile small breed and toy breed dogs. However, older patients can have a medial patella luxation, as well as patients of varying sizes. While this condition can develop due to trauma, the most common form is a result of genetics. It is necessary to identify these patients as they should be removed from breeding programs. About 90% of patients with a patella luxation will have a medial patella luxation and 10% will have a lateral patella luxation. When discussing these issues (more will be reviewed in the patella luxation lectures), the luxation is the effect and the irregular limb alignment is the cause, except in the true trauma induced luxation case. What this means is usually there are limb angulation issues that predispose the patient to a patella luxation, has the patella attaches proximally to the quadriceps which attach at the hip and distally the patella attaches to the straight patellar ligament that attaches to the tibial tuberosity. Any variance in these attachment points can cause luxation. It is typical for the femur to have some degree of distal varus, however some patients with excessive varus will be predisposed to medial patella luxation. Likewise, if a patient has tibial torsion and/or external rotation of the proximal tibia, this also will predispose to medial patella luxation.

It is necessary to place the stifle in all ranges of motion and place stress on the patella in both the medial and lateral direction. A true luxation will allow the patella to move beyond the trochlear ridge in either direction. It is important that equal pressure is placed in both directions. Medial patella luxation is generally graded on a scale of 1-4 and defined by the ease of luxation, ease of reduction, and anatomical positioning of the distal femur and proximal tibia. On evaluation of these patients, it is important to both watch their gait and get a characterization of their gait by the owner. The typically gait is marked by intermittent non-weight bearing lameness, however in more chronic cases, more continuous weight bearing lameness is possible.

After thorough evaluation of the stifle and patella, palpation of the femur should be performed. Much like the tibia, care should be taken for abnormalities and painful areas. The osseous conditions of the diaphysis of the femur are similar to the tibia. It is in this region (medial thigh) that one can palpate for muscle discomfort that can arise of iliopsoas injury. Also, the muscle groups of the gracilis and semitendinosus can undergo changes and contract due to previous trauma. Palpation will elicit pain and the muscle belly will become taunt and firm with scar tissue.

The next joint for evaluation is the coxofemoral joint. More detail is devoted to causes of pathology in the hip dysplasia lectures, this will focus on examination techniques. For hip evaluation, the ideal scenario is for both an awake and sedated examination, both allowing the clinician to fell for different issues. Most chronic hip changes result in weight bearing lameness versus non-weight bearing.
Acute injuries to the hip joint will generally result in a more severe degree of lameness and many times result in a non-weight bearing lameness. The most typical traumatic injuries to affect the hip would be luxation and fracture of the femoral neck, head, and/or acetabulum. It is important for the clinician to assess gait and directly palpate the hip joint.

For patients experiencing a luxation of the coxofemoral joint it is important to look at the positioning of the limb. There are two types of hip luxation – craniodorsal (most common) and caudoventral. Most luxations occur due to some type of trauma, however there are reports of spontaneous (atraumatic) luxations. Craniodorsal luxations result in positioning of the limb elevated off the ground and the distal limb positioned towards midline. Caudoventral luxations will result in the limb being in an adducted position (distal limb placed more laterally). Another tool the clinician can use is palpation of the greater trochanter. In the normal patient, a triangle can be made between the wing of the ilium, greater trochanter, and point of the ischium (tuber ischii). When the coxofemoral joint is luxated in a craniodorsal direction the greater trochanter can be palpated dorsally (triangle is lost). Lastly when evaluating for a luxation, range of motion is assessed. Pain is elicited in phases of motion, as well as reduced range of motion.

Palpation of the hip joint and pelvis should be performed in order to assess for fractures. Range of motion should be attempted in the patient to assess for fractures associated with this joint as well. A rectal examination should also be performed in all patients where pelvic trauma is suspected. There are multiple reasons for this examination: fractures to the ischium and pubis can be palpated, assessing for rectal tears, and assess for anal tone/neurologic compromise.

In patients that have suspected hip dysplasia, a thorough examination of the coxofemoral joint is needed. The first step to evaluation is assessing the patients gait and degree of lameness and limb(s) affected. Once this is determined, careful evaluation of range of motion with both external and internal rotation is performed. This evaluation can be performed in the awake patient in most cases. For complete evaluation the patient will need to be sedated to look at subluxation and reduction of the hip. The most common examination performed is the Ortolani test which evaluates for subluxation and reduction of the hip joint. The actual associated angles can be useful when looking for candidacy of certain procedures (double pelvic osteotomy). The Barlow test is a similar type of examination. While the patient is sedated, radiographs should also be performed in order to make the final diagnosis of hip dysplasia.

As mentioned in the thoracic limb evaluation course notes, a thorough neurologic examination should also be performed in patients, as both go hand-in-hand. It is important for the clinician to be able to differentiate between the two types of impairment that can occur. The complete neurologic examination should include proprioceptive evaluation, reflex assessment, proper withdrawal and proper sensation to the limb. Also, evaluation for spinal hyperpathia, cutaneous reflexes, and anal tone is important. The more one performed a complete orthopedic and neurologic examination the better the skill set. Repetition is key for the development.
Objectives
- To provide guidance for performing a repeatable examination of the forelimb
- To provide key “tricks” aiding in the examination process
- Aid in assimilating examination findings with diagnostic tests to provide an accurate diagnosis

Introduction

One of the key ways to arrive at an accurate diagnosis is the successful completion of a physical examination. It is the examination that allows us to localize injury and disease. While not all examination findings are pathognomonic for a specific disease process, the presence of pain, swelling, etc. can lead us to our next step in the diagnostic process. In order to reach a diagnosis, there is a series of steps that must be taken. As the clinician gains experience and confidence, this series of steps may be lessened, however the physical examination must never be omitted. The physical examination is just as (or more) important as the clinical history and diagnostic tests.

Physical Examination

When one starts their physical examination the absolute first step is a gait evaluation. In many cases the gait examination should be carried out by a technician or owner and the clinician should evaluate the animal both walking away and towards, as well as from both sides. This allows the clinician to start by identifying the problematic limb(s). It is important that the patient be walked at varying speeds as this may exacerbate a particular condition. From a gait evaluation, one can discern whether this is a neurologic or orthopedic ailment and develop potential differential diagnoses even based off of the gait analysis alone.

Once the affected limb is identified, evaluate the patient at a stand and first evaluate the unaffected contralateral limb (if unilateral disease). This assessment gives us a “control” for comparison. The physical examination starts first by evaluating for any obvious swelling to joints or long bones. Next, start with the distal-most aspect of the limb – the digits. Evaluate the nail beds for changes – swelling, drainage, or abnormal nail growth. Palpate the digits, as well as, flex and extend the phalangeal joints. In this location, palpate the sesamoid bones for signs of pain and discomfort.

Once the phalangeal joints are evaluated, full evaluation of the carpus is performed. Full extension and flexion is evaluated. Also, assess for carpal swelling, both unilateral and bilateral. Bilateral carpal swelling can indicate a systemic issue such as immune mediated polyarthritis (IMPA). Many high-energy/working dogs and geriatric patients will have evidence of osteoarthritis at the level of the carpus. This can occur by chronic insult to the carpus. It is not uncommon to see mild decrease on hyperflexion of the joint and this may or may not be a clinical issue. Always compare the affected to the non-affected site.

Moving past the carpus, long bone palpation of the radius and ulna is very important. Especially depending on the age of the patient. In juvenile patients we can see panosteitis and in the aged patient (not exclusively) we can see primary bone tumors such as osteosarcoma. Fracture of these bones will generally be obvious due to the instability produced. Angular deformities will also be obvious during gait and standing.

The next site moving proximal is the elbow. This is a complex joint to evaluate. Again, signalment and history is very important. It is important to put the elbow in hyperflexion and hyperextension. The most common elbow disease that is seen is the hereditary process called elbow dysplasia. Elbow dysplasia is a complex of abnormalities affecting the elbow joint. When evaluating a patient for this disease process, it is important to apply pressure to the elbow on the medial side of the joint, which is in the area of the medial coronoid. Reaction associated with discomfort is a hallmark sign in the young and older patient affected by elbow dysplasia and medial compartment disease/fragmented medial coronoid process. Also, with medial pressure applied and the elbow flexed, internally rotate the carpus (supination). This movement also elicits pain in the dog with a fragmented coronoid process.

It is also important that you apply normal pressure and palpate over both the medial and lateral epicondyles. This is especially true in puppies with acute onset of lameness, as fractures can occur in this region.
Olecranon fractures are rare, however, you will feel instability at the olecranon due to the tension from the triceps attachments. Bicondylar (Y fractures) can occur in the juvenile and adult patients; instability will be palpated, and the full extent of the injury will be noted on radiographs.

Most disease processes associated with the humerus will either be traumatic (fractures) and/or neoplastic. Primary bone tumors (osteosarcoma) will generally occur at the proximal humerus and the distal radius, however other locations have been reported and seen in clinical practice. If neoplastic changes are noted within the diaphyseal regions of the bone, there is a higher chance for the lesion to be metastatic and a full workup searching for a primary mass is needed. If one palpates thickening of the metaphyseal regions with discomfort, radiographs and/or CT scan would be indicated.

The shoulder can be an intimidating joint for evaluation. Normally the canine (and feline) scapulohumeral joint has a very defined and limited range of motion. There is the predominate cranial (extension) and caudal (flexion) motion and very limited lateral and medial motion. The medial and lateral ranges of motion (<40 degrees) are sometimes difficult to assess. This may require sedation for proper assessment of glenohumeral (rotator cuff) ligament tears/trauma. Measurement of lateral movement angles can help with the diagnosis of glenohumeral ligament tears; however, the next step would be either MRI or arthroscopy for a definitive diagnosis.

In the young canine patient, we are often times looking for examination evidence of osteochondritis dissecans (OCD) of the caudal humeral head. Pain will generally be evident on hyperflexion (primarily) and hyperextension. The presence of pain would indicate that radiographs and/or CT be pursued as the next step. Most OCD lesions are noted on lateral radiographs as a small lucency at the caudal humeral head.

In the young to middle aged athletic patient, we also see other sports related musculotendinous injuries. One of the more common injuries seen occur with the biceps and supraspinatus tendons. These two tendons cross each other in the cranial aspect of the shoulder joint and insert opposite of each other. The biceps tendon crosses the shoulder from distal to proximal and attaches on the supraglenoid tubercle. The supraspinatus muscle originates on the cranial aspect of the scapular spine and attaches to the greater tubercle of the humerus. It is responsible for external rotation of the shoulder, as well as extension of the shoulder joint. The physical examination can be pathognomonic for this disease process. A musculoskeletal ultrasound and/or MRI can aid in further diagnosing this condition. This injury has a very good outcome with surgical intervention.

Infraspinatus injuries and contracture is a very rare injury, however physical examination diagnosis can be very straightforward. This will occur in active, bird/hunting dogs typically, but is not limited to this class of dogs. Fibrosis and contracture will generally occur at some point after the initial injury. In hindsight, owners many times recall when the patient was lame following activity and that the lameness resolved over a few days with rest and NSAID therapy. In patients with infraspinatus contracture the common exam finding is that the dog develops external rotation of the shoulder joint and the patient is resistant to internal rotation. The muscle originates caudal to the scapular spine and attaches to the greater tubercle of the humerus. It is responsible for external rotation of the shoulder, as well as extension of the shoulder joint. The physical examination can be pathognomonic for this disease process. A musculoskeletal ultrasound and/or MRI can aid in further diagnosing this condition. This injury has a very good outcome with surgical intervention.

Once an orthopedic examination is completed, it is always necessary to assess the neurologic status of the patient. Assessment of the neurologic status of the limb and cervical region (looking for discomfort) is very important. While a patient may be lame, this can be due a cervical disk rupture, neoplasia, or other neurologic disorders affecting the cervical/axillary regions. Neurologic injuries such as brachial plexus avulsions can also occur. When evaluating for potential neurologic causes of lameness, a cranial nerve examination should also be performed. Depending on neuro-localization, cranial nerve deficits can be seen such as Horner’s syndrome.
It is very important that one develops a very systematic orthopedic and neurologic examination of the forelimb. This approach will allow the clinician to gain confidence in their abilities. The use of the opposite limb for a “control” model and the effectiveness of performing these evaluations in both standing and lateral recumbency are very important. The orthopedic examination should be the foundation of the diagnostic work-up of forelimb lameness.
Management of canine OA requires development of a plan that is thorough and tailored to the individual patient and client. The Comprehensive Care Quad was developed by this author to ensure a systematic approach to developing an OA treatment plan for canine patients. Not every treatment strategy will be employed, but each category must be addressed in order to offer the wide range of management techniques available at this time.

1. Pain Management
   - The foundation of pain management is use of a non-steroidal anti-inflammatory (NSAID), including cox-inhibiting products or Galliprant. These medications have proven efficacy, and when used appropriately, have a strong safety profile. Dogs should not be denied proven analgesia based on age (too young or too old). If traditional cox-inhibiting NSAIDs are contra-indicated, most dogs are able to tolerate Galliprant. When NSAIDs are insufficiently managing pain, other medications such as amantadine, gabapentin, acetaminophen, and codeine may be added.
   - Needles: There are currently 3 common pain management techniques for OA administered by injection:
     i. Adequan- this is an FDA approved product that is labeled as a disease modifying osteoarthritic drug. It is most effective at preserving articular cartilage when used early in the OA disease process. Its mechanism of action also provides pain relief through decreasing inflammation in a mechanism distinct from NSAIDs. It is administered intra-muscularly or subcutaneously (NOT intra-articularly).
     ii. Intra-articular therapy- this includes administration of steroids, hyaluronic acid, platelet rich plasma, and/or stem cell therapy directly into the effected joint. There is research to support each of these techniques, and intra-articular therapy should be a routine part of managing OA in dogs. Novel intra-articular therapies are currently being developed, including radioactive tin (Tin 117m), which may offer long-term pain relief.
     iii. Acupuncture- this is a non-pharmaceutical technique that may offer adjunctive pain relief for many dogs.
   - Surgery: Depending on the effected joint, there are many surgical techniques that are indicated to manage OA. Some techniques offer excellent long-term prognosis, such as total hip replacement, while others are considered salvage procedures for end stage OA (FHO, arthrodesis). Others yet are indicated to help manage symptoms, but require additional comprehensive treatment to be successful (subtotal coronoidectomy). The point being, surgery is often indicated as an adjective therapy to manage the pain associated with OA.

2. Nutritional Management
   - Weight management is the single most important aspect of managing OA. Clients should be counseled early in the dog’s life about the importance of their dog maintaining a lean body condition (4.5/9). Veterinarians should be prepared to make specific diet recommendations, including caloric requirements for weight loss and maintenance.
   - Nutritional supplements: these include omega 3 fatty acids, cannabis, and many other products that may help support joint health. Veterinarians should be prepared to have an evidence-based discussion with pet owners on the value of nutritional supplements. While the research is somewhat limited in this category, the majority of pet owners will use a joint supplement, even if not prescribed by the veterinarian. Therefore, this author urges veterinarians to make specific recommendations for products that they know and trust.
3. Lifestyle Management

- Activity modification - Dogs with OA should engage in regular, low impact exercise such as walking (ideally 20-60 minutes per day), swimming and scent detection such as “Noseworks.” High impact activity such as running, jumping, and ball-play should be avoided.
- Home environment - The dog’s home environment needs to be modified to accommodate their needs, including ensuring non-slip flooring, including on stairs, and using ramps and steps if needed. Adequate bedding is essential. Other modifications may include raising the food bowl or limiting access to certain areas of the house that are “OA friendly.”
- Assistive devices - Harnesses, slings, support wraps and orthotics may all have a place in management of OA.

4. Rehabilitation Techniques

- Formal physical rehabilitation can be very helpful for dogs with OA. Consultation with a veterinarian certified in canine rehabilitation (CCRT, CCRP) is strongly recommended. Rehabilitation techniques that are helpful for dogs with OA include targeted therapeutic exercise, manual therapy and therapeutic modalities such as cold/compression, PEMF and photobiomodulation.

For more information on these treatment options and associated references see [www.caninearthritis.org](http://www.caninearthritis.org).
An individualized and comprehensive plan of care for a patient with OA is all well and good, but how do we know if it’s working? How do we know when to change course? Prior to initiating an OA treatment plan, baseline measurements should be taken, which include body weight, body condition score, lameness evaluation, and establishment of an OA Stage using the COAST system. In order to establish the OA Stage, a clinical metrology instrument (CMI) must be used. This is a client questionnaire that is designed to evaluate the dog’s functional abilities and level of pain in the home environment. There are several CMIs that have been validated and have been shown to be able to detect response to treatment. Examples include: Canine Brief Pain Inventory and Canine Orthopedic Index. Another example of a CMI is the Client Specific Outcome Measure. Veterinarians should become familiar with CMIs and adopt one to use in practice.

In addition to the aforementioned baseline measurements, it is crucial to discuss realistic and achievable goals with clients. Working toward these goals will be the aim of any treatment plan. Once starting an OA treatment plan, dogs should be re-assessed every 1-4 weeks initially, particularly if weight loss is part of the plan. In the first month, clients will need accountability in order to develop new habits that go along with their plan, such as regular walks and incorporating therapeutic exercises into daily routines. CMIs should be repeated every 4 weeks. A positive improvement should be seen within 2-4 weeks for any NSAID; if no improvement is seen, a different product may be needed.

Weight loss will take weeks to months to achieve, depending on the amount of weight loss needed. Clients can become discouraged with the slow progress that this effort takes; however, it is crucial to continue to hold the client accountable and provide continued support and motivation. The expected rate of weight loss should be about 1% per week; if this is not seen, you may need to change the diet or revisit the conversation of treats and total calories with the client.

This author recommends at least once a month rechecks during the first 3 months of starting an OA plan; if after 3 months the patient has improved and is on a maintenance course, the next appointment should be set for no longer than 3 months to ensure that the client remains compliant. Thereafter, examinations should be no less frequent that every 6 months. CMIs should be repeated at each visit.

For more information on CMIs, comprehensive plan development, and associated references see www.caninearthritis.org.
Osteoarthritis (OA) effects an estimated 20% of dogs and is the leading cause of chronic pain in canine patients. There is widespread misconception among veterinarians and pet owners that OA is a condition that only effects older dogs. In fact, at least half of cases of canine OA are diagnosed in dogs over the age of 8 years of age. Yet another staggering statistic is that 50% of cases of OA go undiagnosed. This may be due to a combination of pet owners and veterinarians not being aware of the early signs of OA, and importantly, understanding what causes OA in dogs.

Humans (and potentially cats) develop primary OA; this is a degenerative condition of joints that results due to “normal wear and tear,” which develops as we age. Dogs most often develop secondary OA, due to an underlying developmental or traumatic injury to the joint. The most common cause of OA in dogs is Developmental Orthopedic Disease (DOD), including hip dysplasia, elbow dysplasia and medial patella luxation. By definition, DODs are present in juvenile dogs. Identifying these dogs at risk of OA early in life offers the best opportunity for pro-active care.

There are at least 8 common DODs that lead to OA in dogs:

1. Hip Dysplasia (HD)
   Hip dysplasia can be diagnosed in very young dogs. This author suggests that all puppies, particularly those breeds at risk of HD, should be tested for an Ortolani sign when they are 16 weeks of age. If the Ortolani exam is positive, this indicates hip laxity, i.e., the dog has hip dysplasia. A PennHip radiographic exam can be performed at this age as well, which provides an objective measurement of hip laxity and can compare the severity of laxity to other dogs of the same breed. There are 2 surgical procedures that can only be performed in juvenile dogs with HD: juvenile pubic symphisisiodesis (JPS) and triple (or double) pelvic osteotomy (T/DPO). Clients of dogs with HD should be counseled on weight management, activity recommendations, optimal age for spay/ neutering, and pain management.

2. Fragmented medial coronoid process (FMCP, a form of elbow dysplasia)
   FMCP is the most common form of elbow dysplasia. Dogs with this condition typically present between 6-9 months of age with unilateral or bilateral forelimb lameness. CT or arthroscopy is usually needed to confirm the diagnosis. Consultation with a surgeon is recommended at this time to discuss potential surgical interventions. However, no matter what surgery is performed, long term OA management will be needed.

3. Ununited anconeal process (UAP, a form of elbow dysplasia)
   UAP is a relatively infrequent form of elbow dysplasia, but can easily be diagnosed based on a flexed lateral radiograph in dogs over the age of 5-6 months. Consultation with a surgeon is recommended at the time of diagnosis to discuss potential surgical interventions. However, no matter what surgery is performed, long term OA management will be needed.

4. Osteochondrosis (OC/OCD) of the elbow
   Like other forms of elbow dysplasia, OC/OCD typically presents in dogs between 6-9 months of age. Consultation with a surgeon is recommended at the time of diagnosis to discuss potential surgical interventions. However, no matter what surgery is performed, long term OA management will be needed.
5. **Osteochondrosis of the shoulder**
OC/OCD of the shoulder presents very similarly to elbow dysplasia. A single lateral radiograph of the shoulder is typically sufficient to diagnosis the condition. Consultation with a surgeon is recommended at the time of diagnosis to discuss surgical interventions. Typically, surgical treatment results in an excellent long-term prognosis. However, the patient should be monitored closely long term as OA will develop, but may not become clinically significant.

6. **Osteochondrosis of the tarsus**
Like other DODs, this condition typically presents in dogs under 1 year of age. The OC/OCD lesion is typically present on the medial trochlear ridge, and firm swelling of the medial tarsus can often be palpated. Radiographs are often sufficient for diagnosis, though CT may be needed in some cases to confirm a diagnosis. While surgery can potentially be pursued, it does not offer an excellent prognosis and long-term management of OA is essential.

7. **Medial patella luxation (MPL; or lateral patella luxation, LPL)**
Patella luxation is typically due to congenital malalignment of the quadriceps mechanism and abnormal biomechanical forces acting on the patella and femoral trochlea. Patella luxation is graded 1-4 based on the severity of luxation, and many cases can progress in severity. MPL has been shown to lead to OA of the stifle, and is also a predisposing cause for cranial cruciate ligament rupture, which will lead to significant OA if not treated surgically. Not all cases of M/LPL will require surgery, but discussion of comprehensive management of OA and at least semi-annual monitoring is recommended.

8. **Avascular necrosis of the femoral head (Legg-Calve Perthes Disease)**
This condition typically develops in miniature and small breed dogs. A disruption in blood supply to the femoral neck and head leads to irregular growth of the hip joint, which will result in OA. The condition is typically painful, leading to significant lameness and discomfort with manipulation of the hip. Radiographs are sufficient for diagnosis, and femoral head and neck ostectomy (FHO) offers an excellent prognosis.

For more information on these conditions, including diagnosis, treatment, and references, see [www.caninearthritis.org](http://www.caninearthritis.org).
What’s your CCQ? Developing a comprehensive care plan for dogs with OA using the CCQ
Kristin Kirkby Shaw, DVM, MS, PhD, CCRT, DACVS-SA, DACVSMR
Animal Surgical and Orthopedic Center &
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CARE: Canine Arthritis Resources & Education
www.caninearthritis.org

Osteoarthritis (OA) effects between 20-50% of dogs and is the leading cause of chronic pain in canine patients. Chronic pain is among the most common reasons for euthanasia. Therefore, early and comprehensive management of OA and associated pain is essential for preserving longevity and quality of life in canine patients.

Historically, dogs with OA were diagnosed with the condition when they reached their senior and geriatric years, and recommendations of rest and anti-inflammatories were prescribed. In this day and age, we can do much better, beginning with diagnosing OA much earlier in the life of the dog. This affords many more opportunities for altering the course of the OA and extending a healthy and active life.

“Multi-modal management of pain” has grown up and has a new name: Comprehensive Care. A comprehensive plan for OA should include 4 key areas, which include many treatment options within each group. This author has developed the Comprehensive Care Quad to ensure systematic and thorough discussion of OA management options with clients. The Comprehensive Care Quad presents a new approach to designing individualized, multi-modal treatment plans that include: Pain Management, Nutrition & Weight Management, Activity/Environmental Modifications, Rehabilitation Techniques. Within each quad are numerous treatment techniques that can be selected based on the individual patient and client’s needs.

Not all treatments will be started at the same time. Patients should be monitored frequently (weekly to monthly when initialing an OA program) and the plan adjusted based on response to treatment, as measured by functional abilities, client questionnaires, and physical exam.

For more information on these treatment options and associated references see www.caninearthritis.org.
Fear Plus Pain or Pain Plus Fear? Banish Them Both!
Robin Downing DVM, MS Bioethics, DAAPM, DACVSMR, CVPP, CCRP

Divinum Est Opus Sedare Dolorem  
(Divine is the Work to Subdue Pain) - Galen

Always approach feline pain from a comprehensive perspective. Begin at the beginning with a thorough examination that includes a neurologic exam, soft tissue palpation, joint ROMs, and gait assessment. Include a metabolic profile so as not to miss important co-morbidities.

Treat the treatable - - and treat all the treatable. Make a plan and work the plan, and recognize that chronic pain is best addressed from a **MULTIMODAL** approach, and it is no longer appropriate simply to throw an NSAID at the patient. Break the pain cycle as quickly and effectively as possible **before** initiating physiotherapy/tissue manipulation. Multimodal management of chronic pain means multi-tasking, and the client is an absolutely essential partner in the process, or the process is doomed to fail.

Creating a fear-free experience for cats is a huge part of our obligation to them!

Pre-exam medication options:
Buprenorphine  
0.05mg/kg  
Delivered by owner at home @ 1 hour before the visit  
Be sure to explain HOW to deliver it into the cheek pouch  
It will NOT mask chronic cat pain!

Gabapentin  
100mg PO @ 1 – 2 hours pre-exam  
It will NOT mask chronic cat pain!

Some cats do best with BOTH gabapentin AND buprenorphine.

Acepromazine (1mg/ml) – 0.01mg/kg + hydromorphone (2mg/ml) – 0.05mg/kg - - same syringe & delivered SQ  
Can add ¼ of the midazolam dose used for induction if needed - - also in the same syringe.  
Deliver as the cat arrives, wait 20 minutes, will NOT mask chronic pain!

So, what is the relationship among pain, stress, and distress? While animals do not anticipate or fear their own DEATH, they most certainly DO anticipate and fear **PAIN**, rightly attempting to avoid pain whenever possible. The pain cats may choose to avoid may include the following:
IBD  
OA  
Periodontal disease

Pain, anxiety, stress, and distress can become a self-perpetuating cycle.

Let me share some cases:  
Coco Chanel  
Ragdoll, FS, 5kg, 10 years  
Reclusive, impossible to pet, non-interactive with other cats  
Never seen when company came to the house  
Veterinary visits under general anesthesia  
Biopsy confirmed IBD  
Liquid, projectile diarrhea  
Occasional vomiting  
Easily angered  
Adequan®  
Gabapentin  
Feliway® diffusers, wipes, and travel spray
Sybil
DMH, FS, 3.2kg, 15 yrs
Chronic herpes
**HATED** car travel
Poop, pee, and puke in the carrier with every veterinary visit
Feliway® wipes & travel spray
NurtureCalm® Collar
100mg gabapentin before travel

Ganache
Siamese cross, MC, 6kg, 5 yrs
Should weigh 5kg
Aggressive with other cats in household
Anxious/aggressive at veterinary visits
Back pain:
  - T/L
  - L/S
  - SI joints
  - Tail base
Feliway® diffusers & wipes
Microlactin feline chews – 300mg BID
Every 8 week cervical chiropractic adjustments

Flower
At rescue:
DSH, FS, 7.3 kg (16# !!!), 9 yr
Could not be touched
Attacked visitors to home
Mats over 80% of body
Excoriated perianal/perivulval areas
Now:
  - 3.4 kg (7.9#) (Hills Metabolic®)
  - Gregarious, interactive, playful
Microlactin feline chews – 300mg BID

Rooney
DMH, MC, 2.8kg
CRD
OA – lowback, L/S, SI joints
**21½ YEARS OLD!**
Adequan® weekly
SQ Ringers – 60mls SID
Microlactin feline chews – 300mg BID
Gabapentin – 100mg BID
HPD® k/d + Mobility
Weekly cervical Chiropractic adjustment

Pain, Anxiety, & Fear (oh my!)
With unacceptable behaviors (“bad actors”), ALWAYS think about pain FIRST! Remember that anxiety exacerbates pain, and pain exacerbates anxiety. Look at the WHOLE patient. Ask LOTS of questions and LISTEN carefully to clients for clues. Ask for pictures and videos. Adequate resources are paramount, but comfort is critical. It is not enough to offer one or two fear-free techniques. Reducing fear may require reducing pain.
Digital Thermal Imaging: Unlocking the clues to patient health

Jennifer F. Johnson VMD, CVPP

Digital Thermal Imaging - Clues to Pain

The use of thermal images may help to easily and noninvasively unlock the clues to continue our daily clinical pain investigation. Digital thermal imaging (DTI) is a measurement of the radiated energy from target tissues. The thermographic images are viewed with the camera’s display function, showing emitted infrared radiation, which is perceived as heat.

Thermal imaging is based on the concept that physiological changes in the body lead to changes in blood flow. The resulting superficial temperature changes due to blood flow can be detected with the thermographic camera and measured, both qualitatively and quantitatively with medical-specific software. In fact, medical software is what separates clinical thermal imaging equipment from industrial thermography. Medical thermal imaging should have high resolution (minimum of 640x480/ AAT Annual Meeting, Sept 2018) and be able to easily identify the change in temperature when we are comparing areas on a patient.

Thermal imaging technology is not a new modality, it was first used in veterinary medicine in the 1960s, but recent improvements in the equipment have produced a superior image with user-friendly veterinary specific software (1). Digital Thermal Imaging utilizes a completely safe, non-invasive thermographic camera, which detects radiant energy being emitted from the body and converts it to an image we can see. Changes in blood flow directly correlate with inflammation. Where there is increased circulation there will be in increase in the thermal gradient. The opposite is also true: with chronic disease, scarring, atrophy, nerve damage or disuse, areas depict cooler, or with a decrease in the thermal gradient. Many studies have been published reporting evidence that thermal imaging is helpful in identifying areas of disease. In 2014, Grossbard and the surgical group at Long Island Veterinary Specialists published a study looking at type I thoracolumbar disc disease (TLIVDD) in dogs. Their conclusions were that infrared thermography was 90% successful in differentiating between normal dogs and dogs with IVDD and 97% successful in identifying the abnormal disc space location in dogs that had IVDD (2). The same group also looked at canine stifle disease. Digital thermal imaging was compared to radiology, ultrasonography, MRI and CT for evaluation of the stifle and it was found to be useful in early identification of stifle injuries, offering an advantage of objectivity as well as allowing the visualization of physiologic changes within the anatomical region of interest before the onset of structural change (3).

The Digatherm(R) digital thermal imaging tablet has a thermal sensitivity of less than 0.02 degrees Celsius, providing 15-20 times more accuracy than human fingertips (4). The images and software show qualitative results with a variety of color palettes as well as quantitative results with temperature measurements which can be evaluated via the software.

The key to this technology is symmetry. Images can be compared to contralateral anatomy to help to distinguish areas of concern. The significance of asymmetry is a 1°C temperature change in more than 20% of the area of interest. Because of the difficulty of accurate pain assessment in companion animals - and certainly palpation of cats and dogs can be confounding depending on their personality and attitude - DTI can be helpful as a physiological screen of every patient that comes into the office. The idea that thermal images can be added to one’s collection of vital signs makes sense. Collecting images can be as easy as snapping a photo and can be evaluated quickly, just like one would evaluate temperature, pulse and respiration. If an image suggests an area of increased or decreased thermal gradient, this is an area to be further investigated. But isn’t just about identifying ‘fever’. DTI will detect and create a visual picture of any primary, secondary or tertiary area of the body in need of further evaluation. Besides the obvious musculoskeletal implications, thermal gradients can be appreciated with differences in oral/tooth root disease, vascular disease, oral, urogenital and abdominal disease. In our feline patients, images can be obtained, and are most valuable, when the patient can wander the room with no restraint and
handling. In addition to imaging lateral, dorsal, cranial and caudal views, we can also image foot prints on
the floor to assess any limb weight-bearing or gait abnormalities, which we term an orthostatic analysis.

Publications Using Thermography

Studies continue to evaluate thermal imaging in clinical settings and the results continue to be promising.
Specifically, with cats, thermal imaging has been useful for the evaluation of cats with hyperthyroidism,
finding it was 80.5% successful in differentiating hyperthyroid cats from normal thyroid cats. The same
study then looked at cats after I-131 treatment, finding that thermal imaging was 92.8% successful in
identifying cats that had thyroxine concentration in the normal reference range 1-month post iodine
treatment (4).

A more exciting study related to feline pain is the use of thermography to evaluate cats with
thromboembolic disease (FATE). Cats presenting with hind limb paralysis were imaged and the thermal
gradients between ipsilateral limbs were measured, finding that thermal differences have an excellent
specificity and high sensitivity for FATE diagnosis. The authors concluded that “infrared thermography
seems to be a promising useful, easy, non-invasive and rapid method for detecting aortic
thromboembolism in cats, particularly in emergency situations” (5).

A robust preliminary study comparing thermography and palpation with owner pain assessment
questionnaire, involved 103 feline patients presented to the Veterinary Teaching Hospital at the University
of Helsinki. The study concluded that the owner questionnaire was only successful in identification of
cats with severe pain, while thermography and palpation was more valuable in the identification of cats
with mild pain. The ease of use of thermography compared to palpation was noted, suggesting that
thermography is even more valuable as a tool for tense cats reluctant to palpation (6).

1. Grossbard BP et al. (2014), Medical Infrared Imaging (Thermography) of Type I Thoracolumbar
2. Infernuso T, Loughin CA, Marino DJ, Umbaugh SE, Solt PS. (2010), Thermal Imaging of Normal
   1:1098612X17732485.
   examination and modified questionnaire as an instrument to assess painful conditions in cats.
Feline Pain – Increasing Your Tools
Jennifer F Johnson VMD, CVPP

Introduction
Every day, veterinarians are faced with this fact; our cat patients are in pain! The identification and treatment of pain in our patients is key to their longevity and quality of life.

Consequence of Untreated Pain
Studies of pain in humans and animals repeatedly show the significant consequences of unalleviated pain, most notably the increase of mortality in patients suffering from pain. Pain is responsible for a myriad of chemical and neurologic changes which, if untreated, can lead to dire pathophysiologic consequences involving multiple organ systems. The neurohumoral responses to pain and stress trigger a cascade of events, including changes in cortisol, catecholamines, and other hormones. This stress response to injury and subsequent immune system stimulation precipitates the release of cytokines augmenting the systemic inflammatory response syndrome (SIRS). Unchecked, SIRS will lead to multiple organ dysfunction syndrome. The cause or extent of the pain and trauma to the body is not the issue, what will be the ultimate cause of morbidity and mortality is the body’s response to the disease. It has been said that stress kills. One could better surmise that unalleviated pain, no matter what the cause, is a prime component of patient death.

Measuring Success in a Clinical Setting
Veterinarians must consider pain measurement in every patient and strive to quantify pain along with the three other vital signs, temperature, pulse, respiration. With continued pain assessment, one can determine the success of treatment in an unbiased and equivalent fashion. In veterinary medicine, we must continue to collect data, we must continue to collect clinical evidence, and we must exercise care to document responses or non-responses to treatment in the most unprejudiced way possible. To this end, it is important to review reasonable ways to document pain management protocol results to carefully quantify the results of any pain management protocol.

Feline-Specific Pain Scales
Lord Kelvin stated, “If you cannot measure it, you cannot improve it”. Using a practice-specific pain scale for evaluation of patients before, during, and after treatment is critical for measuring treatment success. Owner-education is key - The International Veterinary Association of Pain Management provides excellent feline-specific client education handouts to members. www.ivapm.org

There are many published references available to help you design and implement a protocol in your hospital for pain assessment, including subjective, objective, and semi-objective tools. It is helpful to consider implementing a practice specific pain assessment scale where all veterinarians and nurses are consistent. The Glasgow Composite Measure Pain Scale – Feline, is a short and simple evaluation, allowing consistency and striving for objectivity (Figure 1). Recent research was presented from the University of Montreal, showing progress on the creation of a validated Feline Grimace Scale (1). The most exciting feline-specific tool for pain assessment was developed by the Comparative Pain Research and Education Centre at North Carolina State University College of Veterinary Medicine. In the spring of 2018, the educational website: https://painfreecats.org/ was published and includes the Feline Musculoskeletal Pain Index (FMPI) the first feline-specific validated tool for pain assessment. This website can be shared with clients and staff members and is a wonderful resource for discussion relating to feline pain.

The Problem
Feline practitioners know that individual cats may have very different responses to pain, most of the time the clinical observations can be very subtle. This leads most clinicians to use palpation as the primary tool for pain assessment in cats, which can be confounded by potential aggressive behavioral changes in cats that are in pain and resent palpation (2). The second issue relates to the failure of objective evaluation when using pain scales. Common rhetoric states that pets don’t have a placebo effect – however the same cannot be said about pet owners, or even caregivers. In 2012, while collecting data analyzing dogs with osteoarthritis, Michael Conzemius DVM, PhD concluded that a placebo effect in owner evaluations occurred 56.9% of the time, while even more concerning – the placebo effect in veterinary – or caregiver evaluations - occurred between 40-45% of the time (3). This caregiver placebo effect can be confounding to clinicians as we try first, to identify pain, and then, subsequently try to analyze the success or failure of a particular treatment. There continue to be limitations in the use of pain scales and clinicians are constantly looking for combinations of objective tools to use to evaluate painful areas. With the more frequent use of Digital Infrared Thermography, veterinarians may have found another tool for pain assessment.
Digital Thermal Imaging - Clues to Pain

The use of thermal images may help to easily and noninvasively unlock the clues to continue our daily clinical pain investigation. Digital thermal imaging (DTI) is a measurement of the radiated energy from target tissues. Thermal imaging technology is not a new modality, it was first used in veterinary medicine in the 1960s, but recent improvements in the equipment have produced a superior image with user-friendly veterinary specific software (4). Digital Thermal Imaging utilizes a completely safe, non-invasive thermographic camera, which detects radiant energy being emitted from the body and converts it to an image we can see. Changes in blood flow directly correlate with inflammation. Where there is increased circulation there will be an increase in the thermal gradient. The opposite is also true: with chronic disease, scarring, atrophy, nerve damage or disuse, areas depict cooler, or with a decrease in the thermal gradient. Many studies have been published reporting evidence that thermal imaging is helpful in identifying areas of disease. In 2014, Grossbard and the surgical group at Long Island Veterinary Specialists published a study looking at type I thoracolumbar disc disease (TLIVDD) in dogs. Their conclusions were that infrared thermography was 90% successful in differentiating between normal dogs and dogs with IVDD and 97% successful in identifying the abnormal disc space location in dogs that had IVDD (4). The same group also looked at canine stifle disease. Digital thermal imaging was compared to radiology, ultrasonography, MRI and CT for evaluation of the stifle and it was found to be useful in early identification of stifle injuries, offering an advantage of objectivity as well as allowing the visualization of physiologic changes within the anatomical region of interest before the onset of structural changes (5). The Digatherm(R) digital thermal imaging tablet has a thermal sensitivity of less than 0.02 degrees Celsius, providing 15-20 times more accuracy than human fingertips (6). The images and software show qualitative results with a variety of color palettes as well as quantitative results with temperature measurements which can be evaluated via the software.

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Feline Specific Publications Using Thermography

Studies continue to evaluate thermal imaging in clinical settings and the results continue to be promising. Specifically, with cats, thermal imaging has been useful for the evaluation of cats with hyperthyroidism, finding it was 80.5% successful in differentiating hyperthyroid cats from normal thyroid cats. The same study then looked at cats after I-131 treatment, finding that thermal imaging was 92.8% successful in identifying cats that had thyroxine concentration in the normal reference range 1-month post iodine treatment (7).

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Multimodal Pain Management for Cats – Photobiomodulation and Pulsed Electromagnetic Field Therapy

Photobiomodulation

Blocking various biochemical and physiologic responses along the pain pathway is the goal of all pharmacological and adjunct pain relief. Pharmaceuticals have been developed to block specific processes of the pain pathway to
great success, but there is good evidence to suggest that photobiomodulation (PBM) also creates biochemical changes along the pain pathway to substantially and effectively produce pain relief. By increasing serotonin and beta endorphin, PBM works to modulate the pain response and decrease the need for exogenous opioids (10, 11). Nitric oxide release from the cell after laser stimulation will increase cellular metabolism and increase vascular blood flow by vasodilation (12). Photobiomodulation also has a direct analgesic effect on nerves. By application of photonic energy to injured nerve cells, the resting potential increases closer to the normal transmission voltage. At the same time, the speed of transmission of the nociceptive signal is decreased by blocking the depolarization of C-fiber afferents. Slowing transmission and increasing the amount of nociception it takes to convert a pain signal are more helpful for acute nerve pain, but multiple studies have shown the remarkable ability of laser therapy to promote nerve cell regeneration by sprouting new axons and repairing nerve cells. injury. Using therapeutic photobiomodulation to increase the speed of nerve cell healing is a key component of laser therapy pain relief by reducing allodynia and neuropathic pain (13,14).

Armed with positive success anecdotes from colleagues, many of today’s modern veterinary practices have invested in a therapy laser and consider it to be ‘standard of care.’ Clinical evidence continues to support the use of laser for acute and chronic painful conditions, but with the caveat that the wavelength, dose, and application technique must be appropriate for successful outcomes (15). The therapeutic dose of energy required for effective pain management is related to the amount of energy that can penetrate into cells to cause the photobiochemical response. Dosing is dependent on the tissue type and the depth of the tissue that you need to penetrate. Current literature recommends doses ranging from 2-15 J/cm². For success, one must combine the appropriate dose with the proper application. The frequency of treatment is dependent on the type of painful condition being treated. Routine surgical incisional pain may only require one treatment immediately after the procedure, and perhaps, if pain persists, a second treatment the next day. More complex acute pain conditions require an aggressive treatment daily for the initial resolution of pain, and then treatments of tapering frequency until the condition is resolved. Chronic pain conditions require the longest treatment plan for best outcome, beginning with frequent treatments (3-4 times a week) for the initial reduction in pain, and then tapering slowly as continued response is measured. Most chronic cases, especially those that include chronic osteoarthritis pain, require continued maintenance therapy, usually once every 4-6 weeks. When considering the addition of PBM to a multimodal pain management plan, clinicians should consider its use for more than just osteoarthritis or any acute musculoskeletal pain. Laser therapy can be quite useful for common visceral pain conditions such as pancreatic pain, dental pain and urinary tract pain (16,17). PBM treatments are well-tolerated by feline patients. Cats appear quite comfortable during a procedure, where limited patient restraint can be applied, and the treatment times are not excessive.

**Pulsed electromagnetic field therapy (PEMF) - a client friendly addition to fight pain**

There are some drawbacks with laser therapy in a clinical setting, notably that successful treatment requires a skilled applicator, usually in the hospital setting. While the vast majority of patients tolerate and even appear to love and appreciate their therapy laser session, often it is difficult for clients to be compliant with follow up treatments and in the case of our feline patients, many clients will opt-out of repeated visits to your veterinary hospital. Pulsed electromagnetic field (PEMF) therapy may provide a pain-relief modality that can work synergistically with PBM, providing pain relief to our patients at home, in a portable and safe fashion. PEMF is a non-invasive treatment that applies pulsed, non-thermal, electromagnetic fields in tissue to promote healing. These devices are approved by the FDA to treat fractures, post-operative pain and edema, osteoarthritis, and plantar fasciitis. The mechanism of action shows that targeted PEMF results in increased concentrations of nitric oxide, which increases vasodilation and decreased inflammation (18,19). Targeted PEMF has been studied for the reduction of pain in patients with knee arthritis, showing significant reductions in pain (20). Most recently, PEMF was studied in dogs with acute, severe sensorimotor complete IVDD injury and it was found to significantly reduce incision-associated pain in dogs post-surgery and enhance proprioceptive placement (21). PEMF has been used to treat bone fractures, inflammation, arthritis, pain, swelling and chronic wounds. The ease of use of PEMF devices makes them attractive in a veterinary setting. In 2018, Gaynor et al. published a retrospective, compiling previous research in the use of PEMF specifically in veterinary medicine, to conclude that “PEMF, as part of multimodal treatment, may improve veterinary clinical outcomes” (22). The cat-friendly application of PEMF needs to be emphasized. The device can be prescribed to the client for use on the patient at home, where treatment applications can be performed, without stress, multiple times daily as prescribed. The Assisi Loop™ system comes in two sizes with a unique loop construction, allowing it to be fastened to a bandage or wrap (Loop-Aid) to stay in place over the treatment area, even if the patient is moving about. The therapy can penetrate through bedding and bandages and wraps, allowing various manipulations for successful application. Most successful feline applications can be accomplished using a bed or carrier with the loop, so that the dose can be applied to the target tissue as effectively as possible (23).

As our knowledgeable and proactive millennial pet owners are more compliant with preventive care, our patients are living longer lives. With longevity comes different health problems, namely the increase in pain and chronic painful conditions that affect our feline patient population. The general practice has an opportunity to improve their pain management portfolio by incorporating more robust pain assessment tools as well as non-pharmacologic analgesic
modalities, to provide more complete pain management and better patient care. By utilizing digital thermography to help identify areas of concern and then adding laser therapy and PEMF therapy to your analgesic toolbox, practitioners can achieve great success in managing pain, while creating an added pain management profit center in practice.

Figure 1

Glasgow Composite Measure Pain Scale: CMPS - Feline

Guidance for use

The Glasgow Feline Composite Measure Pain Scale (CMPS-Feline), which can be applied quickly and reliably in a clinical setting, has been designed as a clinical decision making tool for use in cats in acute pain. It includes 28 descriptor options within 7 behavioral categories. Within each category, the descriptors are ranked numerically according to their associated pain severity and the person carrying out the assessment chooses the descriptor within each category which best fits the cat's behavior/condition. It is important to carry out the assessment procedure as described on the questionnaire, following the protocol closely. The pain score is the sum of the rank scores. The maximum score for the 7 categories is 20. The total CMPS-Feline score has been shown to be a useful indicator of analgesic requirement and the recommended analgesic intervention level is 5/20.
Glasgow Feline Composite Measure Pain Scale: CMPS - Feline

Choose the most appropriate expression from each section and total the scores to calculate the pain score for the cat. If more than one expression applies choose the higher score.

LOOK AT THE CAT IN ITS CAGE:

Is it?

**Question 1**
- Silent / purring / meowing 0
- Crying / growling / groaning 1

**Question 2**
- Relaxed 0
- Licking lips 1
- Restless / covering at back of cage 2
- Tense / hunched 3
- Rigid / hunched 4

**Question 3**
- Ignoring any wound or painful area 0
- Attention to wound 1

**Question 4**
- a) Look at the following caricatures. Circle the drawing which best depicts the cat’s ear position?

![Caricatures]

**Question 5**
- a) Look at the shape of the muzzle in the following caricatures. Circle the drawing which appears most like that of the cat?

![Caricatures]

APPROACH THE CAGE, CALL THE CAT BY NAME & STROKE ALONG ITS BACK FROM HEAD TO TAIL

**Question 5**
- Does it?
  - Respond to stroking 0
- Is it?
  - Unresponsive 1
  - Aggressive 2

IF IT HAS A WOUND OR PAINFUL AREA, APPLY GENTLE PRESSURE 5 CM AROUND THE SITE. IN THE ABSENCE OF ANY PAINFUL AREA APPLY SIMILAR PRESSURE AROUND THE HIND LEG ABOVE THE KNEE

**Question 6**
- Does it?
  - Do nothing 0
  - Swish tail / flatten ears 1
  - Cry / hiss 2
  - Growl 3
  - Bite / slash out 4

**Question 7**
- General impression
  - Happy and content 0
  - Disinterested / quiet 1
  - Anxious / fearful 2
  - Dull 3
  - Depressed / grumpy 4

Pain Score ... /20

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References

4. Grossbard BP et al. (2014), Medical Infrared Imaging (Thermography) of Type I Thoracolumbar Disk Disease in Chondrodystrophic Dogs. Veterinary Surgery, 43: 869-876.

NOTES:
Introduction

Every day, veterinarians are faced with this fact: our patients are in pain. The identification and treatment of pain in our patients is key to their longevity and quality of life.

Consequence of Untreated Pain

Studies of pain in humans and animals repeatedly show the significant consequences of unalleviated pain, most notably the increase of mortality in patients suffering from pain. Pain is responsible for a myriad of chemical and neurologic changes which, if untreated, can lead to dire pathophysiologic consequences involving multiple organ systems. The neurohumoral responses to pain and stress trigger a cascade of events, including changes in cortisol, catecholamines, and other hormones. This stress response to injury and subsequent immune system stimulation precipitates the release of cytokines augmenting the systemic inflammatory response syndrome (SIRS). Unchecked, SIRS will lead to multiple organ dysfunction syndrome. The cause or extent of the pain and trauma to the body is not the issue, what will be the ultimate cause of morbidity and mortality is the body’s response to the disease. It has been said that stress kills. One could better surmise that unalleviated pain, no matter what the cause, is a prime component of patient death.

Measuring Success in a Clinical Setting

Veterinarians must consider pain measurement in every patient and strive to quantify pain along with the three other vital signs, temperature, pulse, respiration. With continued pain assessment, one can determine the success of treatment in an unbiased and equivalent fashion. In veterinary medicine, we must continue to collect data, we must continue to collect clinical evidence, and we must exercise care to document responses or non-responses to treatment in the most unprejudiced way possible. To this end, it is important to review reasonable ways to document pain management protocol results to carefully quantify the results of any pain management protocol.

Canine and Feline-Specific Pain Scales

Lord Kelvin stated, "If you cannot measure it, you cannot improve it". Using a practice-specific pain scale for evaluation of patients before, during, and after treatment is critical for measuring treatment success. Owner-education is key - The International Veterinary Association of Pain Management provides excellent client education handouts to members (www.ivapm.org). There are many published references available to help you design and implement a protocol in your hospital for pain assessment, including subjective, objective, and semi-objective tools. It is helpful to consider implementing a practice specific pain assessment scale where all veterinarians and nurses are consistent. It may be valuable to consider two distinct pain measurements metrics, one for in-practice use and a second validated tool for client assessment and measurement.

The Glasgow Composite Measure Pain Scales for Canine and more recently, Feline, is a short and simple evaluation, allowing consistency and striving for objectivity (Figures 1 and 2). Recent research was presented from the University of Montreal, showing progress on the creation of a validated Feline Grimace Scale, which has been added to the feline short scale (1).

For owner evaluation, the use of a species-specific validated tool is the most accurate. The Canine Brief Pain Inventory (CBPI) was developed in 2008 by Dr Dottie Brown and is available at http://www.vet.upenn.edu/research/clinical-trials/vcic/pennchart/cbpi-tool. The most exciting feline-specific tool for pain assessment was developed by the Comparative Pain Research and Education Centre at North Carolina State University College of Veterinary Medicine. In the spring of 2018, the educational
website: https://painlesscats.org/ was published and includes the Feline Musculoskeletal Pain Index (FMPI) the first feline-specific validated tool for pain assessment. This website can be shared with clients and staff members and is a wonderful resource for discussion relating to feline pain.

**Quality of Life Scales**

Some veterinary pain management experts believe that in the case of chronic pain, evaluation of “Quality of Life” and the use of QOL scales, may be more helpful in the long-term assessment of pain in patients. In fact, in hospice situation, one can use the HHHHMM scale to create a valuable dialogue with owners, where Hurt, Hunger, Hydration, Hygiene, Happiness, Mobility and More good days than bad, can be evaluated for each patient on a scale from 1-10, with a total of over 35 being acceptable quality of life for continued hospice care. The issue with traditional pain assessment metrics is when we are looking at acute versus chronic pain conditions. It is simple to use a metric for acute pain and tell the clinician to “do something”/give pain relief at a certain number, but in the case of chronic pain, it is much more difficult to quantify the change in a way that is meaningful to the patient, owner or clinician. Health-related quality of life assessments have been used in human medicine for over 30 years, and have been valuable in analysis of non-verbal patients. NewMetrica, in the UK, has developed an owner-assessment HRQL scale, which provides valid metrics to clinicians via a web interface, where they can follow owner reporting over time. For canines, it measures energy, happiness, activity and calmness, while for felines, it measures vitality, comfort and emotional well-being (www.newmetrica.com).

**The Problem**

Small animal veterinary practitioners know that individual patients may have very different responses to pain, most of the time the clinical observations can be very subtle. This leads most clinicians to use palpation as the primary tool for pain assessment in cats, which can be confounded by potential aggressive behavioral changes in cats that are in pain and resent palpation (2). The second issue relates to the failure of objective evaluation when using pain scales. Common rhetoric states that pets don’t have a placebo effect – however the same cannot be said about pet owners, or even caregivers. In 2012, while collecting data analyzing dogs with osteoarthritis, Michael Conzemius DVM, PhD concluded that a placebo effect in owner evaluations occurred 56.9% of the time, while even more concerning – the placebo effect in veterinary – or caregiver evaluations - occurred between 40-45% of the time (3). This caregiver placebo effect can be confounding to clinicians as we try first, to identify pain, and then, subsequently try to analyze the success or failure of a particular treatment. Each validated pain tool and HRQL assessment needs to be used “as-written”, to reduce response bias that occurs so often with client as well as clinician caregiver reporting. Some common reasons for subconscious bias stem from the fear of impending euthanasia, the excuse for euthanasia, and the social desirability related to a particular response. It is only with a well-rounded combination of assessments can veterinary practitioners expect to find meaningful success in patient comfort and pain relief (4).

SHORT FORM OF THE GLASGOW COMPOSITE PAIN SCALE

Dog's name ____________________________
Hospital Number _________ Date   /   /   Time
Surgery Yes/No  (delete as appropriate)
Procedure or Condition __________________________________________

In the sections below please circle the appropriate score in each list and sum these to give the total score.

A. Look at dog in Kennel

Is the dog?

(i)  (ii)
Quiet  0 Ignoring any wound or painful area 0
Crying or whimpering 1 Looking at wound or painful area 1
Groaning 2 Licking wound or painful area 2
Screaming 3 Rubbing wound or painful area 3
Chewing wound or painful area 4

In the case of spinal, pelvic or multiple limb fractures, or where assistance is required to aid locomotion do not carry out section B and proceed to C
Please tick if this is the case □ then proceed to C.

B. Put lead on dog and lead out of the kennel.

When the dog rises/walks is it?

(iii) (iv)
Normal 0 Do nothing 0
Lame 1 Look round 1
Slow or reluctant 2 Flinch 2
Stiff 3 Growl or guard area 3
It refuses to move 4 Snap 4
Cry 5

C. If it has a wound or painful area
Including abdomen, apply gentle pressure 2 inches round the site.

Does it?

(v)

Normal 0
Lame 1
Slow or reluctant 2
Stiff 3
It refuses to move 4

D. Overall

Is the dog?

(vi)

Happy and content or happy and bouncy 0
Quiet 1
Indifferent or non-responsive to surroundings 2
Nervous or anxious or fearful 3
Depressed or non-responsive to stimulation 4
Comfortable 0
Unsettled 1
Restless 2
Hunched or tense 3
Rigid 4

Total Score (i+ii+iii+iv+vi+vi) = _______

© University of Glasgow
Short Form of the Glasgow Composite Pain Scale (SF-GOPS) with assessment record sheet, to be used by dog owners

### Date:

- **If Total Score is greater than 5 (4 when not carrying out section B) give analgesics**

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- **If Total Score is greater than 5 (4 when not carrying out section B) give analgesics**

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Glasgow Composite Measure Pain Scale: CMPS - Feline

Guidance for use

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Glasgow Feline Composite Measure Pain Scale: CMPS - Feline

Choose the most appropriate expression from each section and total the scores to calculate the pain score for the cat. If more than one expression applies choose the higher score.

LOOK AT THE CAT IN ITS CAGE:

Is it?

**Question 1**
- Silent / purring / meowing
- Crying/growling / groaning

**Question 2**
- Relaxed
- Licking lips
- Restless/cowering at back of cage
- Tense/crouched
- Rigid/hunched

**Question 3**
- Ignoring any wound or painful area
- Attention to wound

**Question 4**

a) Look at the following caricatures. Circle the drawing which best depicts the cat’s ear position?

![Caricatures](image)

b) Look at the shape of the muzzle in the following caricatures. Circle the drawing which appears most like that of the cat?

![Caricatures](image)
APPROACH THE CAGE, CALL THE CAT BY NAME & STROKE ALONG ITS BACK FROM HEAD TO TAIL

**Question 5**

Does it?
- Respond to stroking 0

Is it?
- Unresponsive 1
- Aggressive 2

IF IT HAS A WOUND OR PAINFUL AREA, APPLY GENTLE PRESSURE 5 CM AROUND THE SITE. IN THE ABSENCE OF ANY PAINFUL AREA APPLY SIMILAR PRESSURE AROUND THE HIND LEG ABOVE THE KNEE

**Question 6**

Does it?
- Do nothing 0
- Swish tail/flatten ears 1
- Cry/hiss 2
- Growl 3
- Bite/lash out 4

**Question 7**

General impression
Is the cat?
- Happy and content 0
- Disinterested/quiet 1
- Anxious/fearful 2
- Dull 3
- Depressed/grumpy 4

Pain Score … /20

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How Drugs and Non-drugs interact with endogenous pain physiology:
FETCH 2019
Bonnie D. Wright, DVM, DACVAA
Mistralvet.com

Know your enemy and know yourself
and you can fight a hundred battles without disaster
Sun Tzu

There is no doubt that the experience of pain transforms an individual’s life, both while it is ongoing and after the acute effects have passed. Pain alters healing, activity, social interactions and the kinesthetics of movement. Pain will always undergo some aspects of amplification and deamplification- allowing it to persist or gradually reduce over time. While the subject of pain processing is startlingly complex, an understanding of the basic framework allows the clinician to design a more appropriate, individualized analgesic treatment for their patient in pain.

When contemplating the physiology of pain signaling there is a continuum between acute pain and chronic pain that defies attempts to confine these definitions to discrete time frames. Physiologists studying ‘acute pain’ define this as pain that does not actually cause damage to tissue. Clearly, this is a very different scenario from what a clinician would describe as acute pain, which very often involves surgical incisions, manipulation of deep, visceral type tissues and always, consequent inflammation. For this discussion, I will not attempt to separate acute pain processing from chronic pain processing, but rather see acute pain as phasic, and chronic pain as a progress through the entire spectrum that becomes tonic, at least for a while.

In the complexity of clinical medicine, it is not possible to predict where any given individual will land along the road of pain amplification. Many will fully recover from acute injury and return to a state indistinguishable from a non-injured counterpart (unless you were to investigate the pain map that was created on their glia). Others will manifest life-long changes and these individuals will tend to move differently and manifest accumulating dysfunction as their kinesthetics, myofascial system and nervous system find alternative methods of processing.

Simple pain begins at the site of injury, and may begin as a proportional response to the level of stimulus (the pain reported is proportional to the intensity of the injury). Peripheral nerve terminals are established in close association with resident mast cells and capillaries. These triads sense changes in pH, temperature and proteins as well as transducing high intensity mechanical stimuli. Unlike the other sensory receptors, pain receptors do not fatigue in response to repeated stimulation. Rather, with repeated stimulation, changes occur that include: increased sensitivity, budding of new receptive terminals, and recruitment of previously quiescent terminals (silent nociceptors). Likewise, when significant neuronal activity occurs (with or without local tissue damage) the associated mast cells and capillaries escalate the release of inflammatory mediators, recruit white blood cells and promote metabolic activity in the region.

Once a signal is sufficient to trigger the high-intensity receptors (pain receptors) the signal travels up the axon. The axon may be myelinated (A-delta fibers) or unmyelinated (c fibers). In the peripheral nervous system, the primary afferent will synapse in the dorsal horn of the spinal cord in lamina 1, 2, 3 or 5. Other sensory fibers synapse in similar regions (especially 3, 4 and 5), allowing interaction between signaling pathways carrying different types of sensory information.
Local Anesthetics/Na Channel blockers
Anti-inflammatories (NSAID or steroid- local or systemic)
Anti-histamines
Ice, heat, TRP channel modifiers
Touch, acupuncture, laser

Before plunging into a discussion of the dorsal horn it is important to recognize some major way-points for pain signal modification along the path of axonal flow. All along the axons of peripheral nerves are ion channels, receptors and the cellular machinery for energy production and electrical gradient maintenance. Contemplating the sheer magnitude of a nerve fiber (picture a horse sensory nerve body in the DRG extending up to the spinal cord and down to the coronary band) provides an understanding of how much activity occurs along this pathway. Imagine the amount of transportation that must occur along the filaments within the axon fibers. Thus, the axonal entity is not inert, but rather subject to modification during prolonged pain states.

Touch, acupuncture, laser
Local Anesthetics/Na Channel blockers
Ice, heat, TRP channel modifiers

Likewise, the dorsal root ganglion (DRG) holds the cell bodies for peripheral pain fibers. There is a tendency to think about these cell bodies as if they were fans in the bleachers- watching the action go by without having any impact on the outcome. Not so, as becomes immediately evident when we consider the role of the cell body. With increased activity along a nerve fiber the cell body becomes very active, synthesizing proteins and receptors, packaging them and sending them long distances along axonal filaments to be placed at nerve terminals, the dorsal horn and along the axons themselves. Ion channel populations change dramatically during chronic pain states, and all of these changes begin with the cell body in the DRG. It is also very interesting to recognize that the DRG is the region in the CNS that is least protected by the blood-brain-barrier. Thus the cell body becomes privy to circulating proteins, drugs, inflammatory mediators and toxins that are excluded from the bulk of pain processing.

Lidocaine (IV route)
Anti-inflammatories (NSAID or steroid)
Acetaminophen (centrally acting COX modification)
Anti-epileptics (gabapentin, pregabalin, etc.)

Returning to the dorsal horn of the spinal cord, the first order neuron devolves the electrical signal from the painful stimulus into a chemical one. Keep in mind that ions still play a major role in this step, with calcium being required to release neurotransmitters into the cleft. The signal is carried across the cleft between first and second order neurons by diffusion of these proteins. The major players in passing this information across the synapse are glutamate and substance P. Many other proteins, receptors and ion channels contribute to the complexity of this process. When repeated stimulation occurs additional channels, proteins and receptors become active. These may serve to facilitate transfer of signals or to dampen transfer of signals. I will discuss the most relevant details in the discussion about currently available therapies directed at this aspect of pain processing.

Adding to the complexity, recognize that there are likely more than just two nerve-endings at the synaptic cleft being stimulated in our discussion. The signal is likely to also pass to interneurons, rapid projecting neurons and glial receptors that live in the same immediate neighborhood. The glial system and other support cells (mast cells, resident macrophages, etc)
have recently been recognized as potent and active contributors to pain signaling. Likewise, interneurons can serve in inhibitory, excitatory and recruiting functions.

- **Narcotics (opioids)**
- **Serotonin/norepinephrine modifiers**
- **NMDA antagonists**
- **Alpha-two modifiers**
- **Cannabinoids**
- **Centrally acting anti-inflammatories (NSAIDS, steroids, acetaminophen)**

After neurotransmitters diffuse across the cleft they bind to specific receptors (such as AMPA and NK1 respectively) which in turn initiate electrical depolarization of the second order neuron. This second order neuron, or projection neuron, will carry the signal up to the brainstem. The spino-cervico-thalamic tract is one of the major paths for somatic pain signaling in domestic animals and it crosses midline in the cervical region- arriving in the thalamus with minimal synaptic modification. The spinoreticular tract is the second important pathway for pain signaling, especially important in carrying deep or visceral pain. It tends to undergo extensive branching and synaptic modification as it ascends both sides of the spinal cord. This difference in ascending tracts helps to explain some of the physiologic and pharmacological differences between somatic and visceral pain. Interestingly, the spinoreticular tract also sends some signals directly through the limbic system- giving a structural reason for the common experience that visceral pain lends a greater sense of misery than superficial somatic pain.

**Spinally administered drugs**

Once pain a pain signal has arrived in the thalamus or reticular system it is distributed to a variety of regions in the cortex, limbic system, midbrain, etc. The nucleus raphe magnus (NRM) and nucleus reticularis gigantocellularis in the medulla receive signaling and provide descending inhibition utilizing serotonin and norepinephrine. The hypothalamus releases endorphin and initiates a cascade of opioid- dependent inhibitory mechanisms.

- **Narcotics (opioids)**
- **Serotonin/norepinephrine modifiers**
- **NMDA antagonists**
- **Alpha-two modifiers**
- **Cannabinoids**
- **Anxiety modifiers (environment, sedatives)**
- **Centrally acting anti-inflammatories (NSAIDS, steroids, acetaminophen)**
- **Acupuncture, massage, exercise**

The mechanistic explanation of pain signaling that has emerged around the first synaptic transfer (dorsal horn of the spinal cord) does not translate easily to the complexity found in the CNS. Pain signals synapse in the limbic system, allowing emotional state and memory to affect processing. They synapse in regions that alter autonomic activity, thereby increasing or decreasing physiological functions, levels of consciousness, etc. The milieu of output from the CNS, is therefore less distinct. This is frustrating as a scientist, but perhaps more fascinating as a clinician as it allows entry of concepts such as ‘quality of life’ and ‘comfort’.

In particular, the glial network of the spine and CNS has been gathering significant respect as a long-term modifier of pain. The glial cells become activated by activity within the synapse, and rapidly spread this excitement (non-synaptically) from cell to cell, across midline, and facilitating the intensity of the signal. These changes may persist far longer than the wind-up that occurs in
the nerves, and can contribute to conditions such as neuropathic pain, opioid tolerance and addiction, and reduced healing within the nervous system.

- **NMDA antagonists**
- **Opioid antagonists**
- **Glial antagonists**
- **Acupuncture, exercise**

Muscle and fascia live alongside the neurological framework and become implicated in changes that occur. With excessive neuronal activity associated muscles spasm and enter an ‘energy crisis’. An initial sustained release of calcium due to muscle splinting and activation results in sustained sarcomere contracture. Increased metabolic rate of these muscle groups is compounded by local ischemia due to the contracted state of the muscle inhibiting local blood flow. Energy in the form of ATP is required to move intracellular calcium back into the sarcoplasmic reticulum at the end of the contraction, which is no longer possible in the sarcomeres anoxic state, thus the high intracellular calcium remains, sustaining the contraction.

In addition to be painful of their own right, chronic muscle contraction (trigger points) contribute to changes in movement and put additional strain on joints and/or spinal segments served by the contracted muscle bellies. Over time this ‘myofascial restriction’ help to create co-morbid conditions that accumulate into a multi-faceted pain experience. Furthermore, in our non-speaking patients, a knowledge of these patterns can create a standard ‘pain map’ that helps us to identify the likely sources of pain, or at least implicate typically-associated groups (shoulder/neck and iliopsoas/caudal lumbar).

- **Acupuncture with trigger-point deactivation**
- **Local anesthetics**
- **PRP- regenerative medicine**
- **Exercise/ motion/ Stretching**
- **Massage**
- **Heat/Ice**
- **Laser/ shock-wave**

Pain assessment is critical end-point to this conversation about physiology. Through a combination of body language in motion, stance and transitions to recumbency a vast amount of information can be gathered. This is followed by inquisitive but gentle touch. The myofascial patterns that develop in painful conditions are predictable, repeatable and can help create a map that describes the underlying pathology. Heat is often present along these myofascial kinetic chains, and both touch and thermal imaging can be used to discern these patterns. Often, the process of treating pain with physical medicine techniques will show marked changes in the heat patterns from the initial exam (either amplifying or cooling), so rechecking for heat after a session is a valuable added perspective.
Physiology of Non-Pharmacological Pain Medicine
FETCH 2019:
Bonnie Wright, DVM, DACVAA
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Description:
Review the biochemical underpinnings of non-pharmacological therapies such as thermal therapies, acupuncture and laser therapies, electro-magnetic therapies. Discuss the biochemical underpinnings of exercise, and tissue stretch

While a sizable portion of the ‘western medicine’ taught in Veterinary and Medical School has a pharmacologic base, a growing awareness of physiologically based medical interventions has arrived. Physiology provides a complex, multi-faceted tapestry for both pain sensing and modification of the transmitted signal. The overlap between the nervous system and other endogenous, autonomic systems (such as immune state, GI motility, etc.) that has long been recognized in osteopathic medicine is now becoming main-stream and evidence base is ballooning. Understanding the physiology of nervous system wiring is the first step toward understanding methods to intervene in these endogenous systems, and help modify them towards homeostasis. The relationship of many of these modalities with vagal nerve stimulation and interactions opens a growing field of evidence to help describe the mechanisms for these interventions. (Browning K, 2017)

While pain is a major target, several other conditions applicable to veterinary practitioners are also gaining an evidential basis:
- Analgesia (PENG GAO, 2015)
- Neurologic injury mitigation and return to function (Qaseem A, 2017)
- Gastro-intestinal motility disorders (Li H, 2015)
- Orthopedic trauma and return to function (Sullivan D, 2016)
- Immune and other homeostatic systems (seizures, immune-mediated conditions) (Kim SK, 2010)

To elaborate on treatments for each of these categories in subsequent lectures today, an understanding of the physiologic underpinnings of physical medicine is required. Physical medicine techniques exploit endogenous physiology in numerous ways:

Temperature related modalities
- Ice (physical) or menthol/peppermint (chemical)-
  TRP M8 channels on afferent nerve endings (McCoy DD1, 2011 Jun;300(6))
  Activation provides a continuum of cold-analgesia through cold-pain (noxious), as well as voltage-mediated modifications (electrical stimulation and TENS). (Knowlton WM, 2011 Jan 1;12(1))
  In addition to modification of blood-flow, inflammation and swelling (Millis, Jan;45(1)) cryotherapy has a discrete biochemical effect on the modification of pain. Most veterinary studies just focus on degree of cooling relative to time (20 minutes appears ideal in most tissues). To understand this biochemistry, we will review the afferent
nociceptor, which responds to chemical, mechanical and thermal sources of pain. Transient receptor protein channels (TRP) are ion channels that are distributed on afferent nerve endings, and conduct specific sensations. Ice is generally considered for acute injuries (post-op up to day 3), but due to analgesic effects, may be used chronically as well.

**Heat:** Physical heat (warm packs, hydrocollator packs, etc) or capsaicin (chemical)

Heat is used to increase blood flow, increase collagen distensibility, and possibly provide mild analgesia (Millis, Jan;45(1)). Ideal temperature has not been elucidated, but a period of 10 minutes is recommended for application (1.5 cm max. depth to surface heating, regardless of application time- for deeper heating see therapeutic ultrasound). In general, heat is used more frequently for chronic conditions, with fibrous, or musculotendinous restrictions, or to help drain edema after it has formed (after approximately the third day after an injury).

TRP V1 channels on afferent nerve endings sense heat. This can be pro-inflammatory or anti-inflammatory (via IL-6 in muscle tissue). (Obi S, 2017 Mar 1;122(3)). In general, heat is not considered directly analgesic at the level of nerve-endings (but see discussion on tissue distensibility, which is likely to provide analgesia to soft tissue restriction secondary to fibrosis and fibrous tissue healing, as well as muscle spasm/trigger points)

Therapeutic ultrasound – Enhances tissue distensibility and wound healing. Heats deeper tissues rather than surface (up to 3 cm). Hair should be clipped (data shows poor heating through hair, regardless of gel). Consider use for deeper heating of muscle and tendon groups. (Millis, Jan;45(1)) Regardless of depth, heating wanes after 10 minutes post-U/S (so need to move to stretching and soft tissue work within 10 minutes).

**Tissue deformation** is a powerful modulator of intrinsic healing in soft tissues such as skin, muscle, ligaments, tendons, fascia, cartilage and periosteum. When tissue is deformed, growth factors and a variety of proteins and neurotransmitters are released, leading to changes in pain processing, metabolic processes, inflammation, blood flow and healing capacity. (HM, 2014) Various techniques, such as: provider-applied techniques (acupuncture, massage, myofascial trigger point release), individual motion (stretching, exercise, physical therapy) and mechanical devices (extra-corporeal shock-wave therapy ESWT), depend heavily on these physiological networks.

**Electro-magnetic Therapies** are often used in addition to the tissue deformation and neuromodulation mediated therapies such as acupuncture and physical rehabilitation techniques. This can be done with the use of electrical stimulation through needles (electro-acupuncture), tissue adhesive on shaved skin (TENS), and PEMF (pulsed electromagnetic field) devices (loops, beds, blankets). This field of treatment is based upon both the ability of living cells to produce electrical fields (as measured by ECG, EEG and bio-impedence), as well as respond to exogenously applied electrical fields. Direct healing effects have been documented, and are attributed to mitochondrial activation (also recognized as a mechanism of action of laser), chemical changes (mediated by receptors including CGRP, NGF and sP), mechanotransduction, and thermal effects. These changes, as with tissue deformation, have been found at the cellular, tissue, spinal segmental and central nervous system levels. (Mayor, 2007)

**Neuromodulation** Neuromodulation changes neurotransmitters at the skin, along axons, in the spinal cord, interneurons, brain and even in the supportive structures of the glia. These changes can be short term (immediately improving comfort and function) but can also be long-standing (using plasticity of the nervous system to make structural and permanent changes in pain processing and nerve function). Neuromodulation is intrinsic to the analgesic actions of physical medicine techniques. (PENG GAO, 2015) A vast amount of data is available for this topic, especially in research species such as rabbits, rodents, and sometimes, cats and dogs. Non-pharmacological forms of neuromodulation decrease the amount of opioids required for acute pain, as well as many of the side effects of these medications. (Fan AY, 2017 Nov;15(6)) The healing effects of physical medicine...
modalities on the nervous system should not surprise us (as we know that the nervous system functions in a use-dependent pattern), and yet it receives absurdly little air-time.

**Peripheral:** There is a triad of peripheral components: cellular, vascular and neuronal cross-talk to amplify or deamplify peripheral signals, and increase or decrease the receptive fields by modifying the population of peripheral nerve endings. This can be utilized by peripherally applied medications (capsaicin, topical NSAIDS and lidocaine, injected local anesthetics).

**Spinal** Primary afferent fibers transmit impulses from the periphery through A\(\delta\) and C fibers to the dorsal horn of the spinal cord (lamina 1-3 & 5 primarily). Touch fibers terminate more ventrally in the dorsal horn, in lamina 3-5. Note the overlap in lamina 5. This region is replete with wide dynamic range neurons (WDR) than can transmit a variety of signals. If an impulse such as touch, needle penetration or proprioceptive input (from shaking a burned finger, for instance) bombards the dorsal horn in this region, it causes a transient overload, contributing to a decrease in transmission and amplification of pain signals leaving the region. Additionally, punctate stimulation of afferent fibers accentuates spinal cord mechanisms of analgesia using enkephalins, endorphins, serotonin, norepinepherine, purines, glutamate, neurokinin, cannabinoid, ion channel modifiers, modification of transcription, and through additional modification of associated cell types such as interneurons, microglia and astrocytes.

**Central:** Ascending from the spinal cord, the information from a\(\delta\) fibers are carried to the brainstem for rapid dissemination including alerting and autonomic changes. C fiber information are delivered to the limbic system, evoking the emotive qualities of pain and misery. Descending modulation includes use of endogenous chemicals such as enkephalins, endorphins, serotonin, norepinepherine, and endo-cannabinoids. Most of the commonly thought-of analgesic drugs (opioids, dexmedetomidine, ketamine) work through these endogenous systems. Empiric data on the autonomic and emotive aspect of acupuncture therapy have supported the physiologic assumption that this association with the limbic system could be exploited to a patient’s advantage.

**Viscero-Somatic inter-relationships:** The spinal cord carries all types of nerve fibers, including autonomic, proprioceptive and motor nerve groups. Due to inevitable overlap of input and output from regional spinal segments, there is a great deal of spill-over onto the neurologic framework by both pain and acupuncture. This causes the phenomenon of viscero-somatic and somato-visceral projections, where pain from an internal organ can be referred to the periphery, and vice versa. Likewise, treatment of a regional spinal segment is likely to have effects on related structures that are served by the same spinal segment, but not available for treatment due to their location deep within the structure. This relationship opens up a huge variety of conditions that can benefit from neuromodulatory input, such as electrical stimulation PEMF therapies and acupuncture.

**Musculo-tendinous function:** Much of this section relates to using these systems with deliberate physiotherapy, keeping form and function in mind to avoid as much tissue loss, and encourage earlier return to strength and function. Muscles show decrease in mass (atrophy) after only two weeks of reduced use, and a single week of immobilization. Type 1 fibers (slow-twitch- static support and proprioceptive) are the most vulnerable. In general, recovery of muscle mass and function takes longer than period of disuse. Tendons and ligaments: size, fibril structure and organization, collagen biodynamics, HA, chondroitin and water content are all negatively affected by disuse. The tendon-bone interface is the most vulnerable, and the longest to recover (6 weeks of immobilization requires >18 weeks of remobilization). Myofascial trigger points and myofascial strain patterns: Muscle and fascia live alongside the neurologic framework and become implicated in changes that occur with injury, illness and disease. This can occur in patterns associated with common compensatory muscle groups, or simply in a spinal-segmental association with the irritable regions of the nervous system. This can be used to aid in diagnosis, monitor treatment success, and contribute to treatment success.
With excessive neuronal activity associated muscles spasm and enter into an ‘energy crisis’. An initial sustained release of calcium due to muscle splinting and activation results in sustained sarcomere contracture. Increased metabolic rate of these muscle groups is compounded by local ischemia due to the contracted state of the muscle inhibiting local blood flow. Energy in the form of ATP is required to move intracellular calcium back into the sarcoplasmic reticulum at the end of the contraction, which is no longer possible in the sarcomeres anoxic state, thus the high intracellular calcium remains, sustaining the contraction. In addition to be painful of their own right, chronic muscle contraction (trigger points) contribute to changes in movement and put additional strain on joints and/or spinal segments served by the contracted muscle bellies. Over time this ‘myofascial restriction’ help to create co-morbid conditions that accumulate into a multi-faceted pain experience. Therapies for myofascial trigger points and strain patterns include acupuncture, laser, heat, stretching, active and passive range of motion, muscle-spindle release mechanisms, mobilizations, massage, and injection or topical application of local anesthetics

**Cartilage and Joints:**

Disuse also causes reduction in bone strength and cartilage health and function. Perfusion of non-vascular tissues, such as articular cartilage and intervertebral disks, is thought to be a dynamic process- with fluids being moved through motor activity and compression/traction cycles (such as walking). Knee loading protects cartilage by decreasing presence of osteoclasts in a rodent study. (Li X, 2016)

**Metabolism, blood flow and immune modulation:**

Vagal nerve input is a powerful modifier of the immune system, and may influence CNS excitability (seizure mitigation). (Browning K, 2017) Acupuncture, electrotherapy and exercise are all modifiers of vagal nerve activity. Blood flow is also altered by various forms of physical medicine, and provides dynamic changes in local immune control, deep organ perfusion relative to peripheral stimulation, and delivery of chemical signals for both pain neuromodulation and tissue healing.

**Exercise:** Physical exercise is analgesic, in addition to providing soft tissue stretching and lubrication. (Naugle K, 2012). Exercise is associated with more rapid recovery after trauma, including brain and spinal cord injuries.

**References:**


The goal for this lecture is to have a case-based discussion utilizing the concepts of multi-modal assessment and treatments for specific pain conditions - to include pharma and non-pharma. The attendees will be asked for the particular cases that will be discussed. Please come with some neuro, orthopedic, geriatric and other complex pain cases that you want to start treating on Monday. The focus today will be for patients managed at home (not hospitalized patients).

The menu options for diagnosis include:
- Observation prior to touching
- Palpation of joints, muscles, fascia and heat/cool
- Practical neurologic assessment
- Other imaging: discussion of radiographic, thermal imaging, musculo-skeletal ultrasound, or more advanced imaging
- Response to treatment

The menu options for pharma include:
- NSAIDs and EP4 receptor inhibitors (can discuss steroids here as well)
- Oral opioids or tramadol
- Anti-epileptic drugs
- NMDA antagonist drugs
- Serotonin and/or norepinephrine modifiers
- Cannabinoid drugs
- Adequan/pentosin protocols
- Topical products: lidocaine and NSAID patches and topical Trp channel modifiers
- Joint injections: steroids, HA, biologicals, exotic options (Botulinum, RTX)

The menu for supplements include:
- Anti-inflammatory and anti-oxidant supplements (PUFAs, curcumin, boswellia, bioflavonoids, ahwagandha, devil’s claw, other phyto-medicinals
- Joint health supplements (glucosamine, chondroitin, GLM, ASUs, milk protein)
- Animal derived products (Myristol, EVA, milk protein, collagen)

The menu items for non-pharma include:
- Ice
- Heat
- Massage, touch
- External pressure (coaptation, thunder shirts, bracing)
- Acupuncture and trigger point release
- Exercise (isotonic versus isometric, basic recommendations and advancements)
- Machine based (Laser, PEMF tools, TENS, shock-wave, hand massagers)
Step 1: Assessment

1) History... what to find out...

2) Myofascial palpation- how and common patterns to uncover

3) Heat assessment- location and changeability during session

4) Strength/function of musculoskeletal system

5) Neurological function

6) Concurrent conditions

Step 2: Individualizing the four legs of treatment:

1) Pharmacological

2) Supplement or biological

3) Non-pharma: externally applied

4) Motion/exercise/physical rehabilitation

Step 3: Modifications- how soon, and how to modify
Step 1: Assessment

1) History... what to find out...

2) Myofascial palpation- how and common patterns to uncover

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6) Concurrent conditions

Step 2: Individualizing the four legs of treatment:

1) Pharmacological

2) Supplement or biological

3) Non-pharma: externally applied

4) Motion/exercise/physical rehabilitation

Step 3: Modifications- how soon, and how to modify
Doc How will I know it’s time?
Helping owners with assessing quality of life

Mary Gardner, DVM
Lap of Love Veterinary Hospice - Nationwide

Learning Objective:
Knowing when is “time” is not an easy thing to do unless a pet is in the active stage of suffering – at that point, it is usually clear to all involved. But most pet owners do not want to have their pet get to the point of extreme suffering. But when do you make that decision? This presentation will give attendees tools and tips to help guide owners through the decision process and to provide them with guidelines to do what is best for the pet and the family.

Proceedings:
Melinda’s phone call to me started off as most of our calls do, with lots of heartfelt tears. It was clear Melinda needed support and additional education through this tough time. Chance, her 4 year old male Staffordshire Terrier, greeted me at the door for our in-home hospice consultation, clearly unconcerned that he has both severe mitral and tricuspid valve insufficiency, along with atrial fibrillation. Melinda understood the gravity of his condition and was well-coached by the cardiologist. Her most pressing issue however, as with most of our clients, is knowing When to make that final decision. It’s the most important question we are asked as doctors and although our clients want a specific timeline, more personalized patient and client information is needed to most comprehensively evaluate quality of life (QOL) and reach an educated, informed, and supported choice that fits not only their pet’s medical condition but also the family’s wishes. “Quality of Life” applies not only to the pet; it applies just as much to the family!

The most commonly used objective measurements for quality of life by veterinarians are mobility, appetite, pain, and proper voiding. I certainly do not disagree with any of these but the presence of quality of life based on these items should not be answered with a “yes or no,” but rather “if… then”.

There are numerous objective QOL scales available that do a wonderful job addressing these, and other, clinical signs of the pet but, in my opinion, leave out the other 50% of the equation; the family’s time, emotional, physical and (when appropriate, financial) budgets. This is why I always start hospice consultations with open-ended questions. I need to get an idea of what the family values most in their pet’s daily life, where their “stop point” is in relation to the pet’s disease condition, and what their idea of a “good death” is for their pet.

The goal is not to evaluate the QOL for the family (although I feel owners want and deserve my opinion) but rather to help them uncover their own thoughts, feelings, and boundaries for their pet surrounding end of life decisions. These questions help me gauge the family’s time, emotional, physical and (when appropriate, financial) budgets:

1. Have you ever been through the loss of a pet before? If so, what was your experience (good or bad, and why)? (Side bar: “Have you ever been through this before?” is usually the first thing I ask. I find that families experiencing quality of life evaluation for the first time generally need more hand-holding and more direct language about the process ahead. They tend to wait for that hand-written letter from their pet saying “I’m ready now, Mom.” This is not just my observation, it is what I hear from these pet owners time and again after the loss of their pet; “I can’t believe I waited that long.”

2. What do you hope the life expectancy of your pet will be? What do you think it will be?

3. What is the ideal situation you wish for your pet’s end of life experience? (at home, pass away in her sleep, etc.)

4. Do you hold any stress or anxiety about any of these issues? (This section is meant to help identify the main concerns the family has.)

   • Pet suffering
   • Desire to perform nursing care for pet
• Ability to perform nursing care for pet
• Pet dying alone
• Not knowing the right time to euthanize
• Coping with loss
• Concern for other household animals
• Concern for other members of the family (i.e., children)

After some discussion, it was clear Melinda most valued the physical companionship Chance brought her. He followed her everywhere, even when it was clear his breathing was labored. She was aware that his condition could deteriorate rapidly at any time, leading to death in minutes to hours at best (a condition I categorize as “imminent”). Knowing the significant anxiety that accompanies dyspnea and the happiness her presence brings him, Melinda placed great value on the quality of death for Chance. Her worst fear was coming home after work to find that he passed away on his own, not knowing if he was in pain or stress during that death phase. Melinda’s stop-point came a couple weeks later when Chance no longer followed her to the next room; she knew it was time. She wanted to be with him and to lean on the support of family at that crucial moment, which is why we met at Chance’s favorite spot on the beach at sunset the next day to peacefully say good-bye.

Ideally, every family’s budgets and boundaries align with the disease process at hand. For Melinda it did, but this is not always the case. The family that places greatest weight on both the happiness of the pet in addition to avoiding an emergency situation at all costs needs to understand the significant risk they run by waiting too long with imminent conditions…. This determines what clinical signs should be weighted most heavily to evaluate quality of life. We have to start moving away from the standard “call me when he stops eating”! Appetite truly does not concern me for the 85 lb Labrador that has severe osteoarthritis. This dog may never stop eating and the family must not rely on this clinical sign to ever manifest itself. The little Yorkie with congestive heart failure that suddenly refuses food, however, definitely concerns me. Each disease process has its own set of clinical signs that should be weighted most heavily.

If the pet is declining in health and there are no additional diagnostics or treatments the family is either willing or able to explore, then quality of life is either an imminent concern or will be some point soon. If the family’s emotional, time, physical or financial budgets are being drained there is a subjective time period in which euthanasia is an appropriate decision to make. This period could be hours, days, weeks, or even months. Before this specific period, I will refuse to euthanize since there is clearly a good quality of life. After this period, however, I will insist on euthanizing due to suffering of the pet. During this larger subjective time however, it is truly dependent on the family to make whatever decision is best for them under the guidance of a supportive medical team. Some owners need time to come to terms with the decline of their pet while others want to prevent any unnecessary suffering at all. Everyone is different. After all, owners know their pet’s personality better than anyone, even the vet!
Chance was clearly a happy boy that loved his mom dearly, watching her every move and following her to the kitchen, just 15 feet from where I was sitting. Melinda, a 25 year old professional, found Chance in the Florida Everglades as a puppy during a college field trip. He grew up with Melinda during her first years as an adult and now helps her feel secure while living alone. She has given Chance the very best quality of life thus far but with such a life-limiting and condition, is facing the difficult and inevitable loss of her boy. Although tired and breathing more rapidly than normal, Chance is happy. He has no perception of what “heart failure” means and no emotional reaction to his physical condition. He is living in the moment (isn’t that what we love about our pets anyway!?). The drawback is that once in pain, animals cannot sense an ending to their hurt. As humans, we can take a pill knowing that the headache will eventually subside but animals have no perception of their suffering ending. This key point is at the heart of quality of life evaluation; how do we measure happiness and prolong it as long as possible.

Pain and Anxiety
Pain in animals is another important topic that all pet owners should be well versed on. It’s the main topic I discuss during my in-home hospice consultations. Myself, and many other professionals, believe that carnivorous animals, such as cats and dogs, do not “hide” their pain, rather pain simply doesn’t bother them the same way it bothers humans. Animals do not have an emotional attachment to their pain like we do. Humans react to the diagnosis of cancer much differently than Fluffy does! Fluffy doesn’t know she has a terminal illness, it bothers us more than it bothers her. This is vastly different than prey animals like rabbits or guinea pigs, who must hide their pain to prevent carnivorous attacks. If you’re interested in learning more about pain and suffering in pets, grab Temple Grandin’s book “Animals in Translation” and read chapter 5.

When discussing the decision to euthanize, we should be just as concerned about anxiety in our pet as we are about pain. Personally, I feel that anxiety is worse than pain in animals. Think about the last time your dog went to the vet. How was his behavior? Was he nervous in the exam room? Did he give you that look that said “this is terrible!”? Now think back to when he last hurt himself. Perhaps scraping his paw or straining a muscle after running too hard. My dog rarely looks as distraught when she’s in pain as she does when she’s anxious. It’s the same for animals that are dying. End stage arthritis patients begin panting, pacing, whining, and crying, especially at night time. Due to hormonal fluctuations and other factors, symptoms can usually appear worse at night. The body is telling the carnivorous dog that he is no longer at the top of the food chain; he has been demoted and if he lies down, he will become someone else’s dinner. Anti-anxiety medications can sometimes work for a time but for pets that are at this stage, the end is certainly near.

Waiting Too Long
An interesting trend that I did not expect when starting my hospice practice is that the more times families experience the loss of a pet, the sooner they make the decision to euthanize. Owners experiencing the decline or terminal illness of a pet for the first time will generally wait until the very end to make that difficult decision. They are fearful of doing it too soon and giving up without a good fight. Afterwards, however, most of these owners regret waiting too long. They reflect back on the past days, weeks, or months, and feel guilty for putting their pet through those numerous trips to the vet or uncomfortable medical procedures that did not improve their pet’s quality of life. The next time they witness the decline of a pet, they are much more likely to make the decision at the beginning of the decline instead of the end.

What about a natural death?
Yes, there are those pets that peacefully fall asleep and pass naturally on their own, but just as in humans, this is rare. Many owners fear their pet “passing alone” while others do not. Occasionally I am asked to help families through the natural dying process with their pet. For different reasons, these families are against euthanasia. I explain everything I possibly can, from how a natural death may look, how long it may take, what their pet may experience, etc. Inevitably, almost all of these families regret doing this. Most of them comment afterwards “I wish I would not have done that, I wish she didn’t have to suffer.” A natural death can be difficult to watch, especially for non-medically oriented people. Most people can watch a human family member in pain much more easily than they can their pet. To an extent, we can talk other humans through physical pain or discomfort. Humans can perceive an ending to their pain (via medication or even death) but there is little emotional comfort we can offer a pet that is suffering, they simply cannot perceive an ending to that pain. Families take this guilt difficulty and I do my very best to not only readily suggest euthanasia when appropriate, but prepare families for a “worst-case” scenario should they chose to wait.
Weigh Your Options Carefully
If the most important thing to you is waiting until the last possible minute to say goodbye to your baby, you will most likely be facing an emergency, stress-filled, sufferable condition for your pet. It may not be peaceful and you may regret waiting too long. If a peaceful, calm, loving, family-oriented, in-home end of life experience is what you wish for your pet, then you will probably have to make the decision a little sooner than you want. Making that decision should not be about ceasing any suffering that has already occurred, but about preventing suffering from occurring in the first place. Above all, our pets do not deserve to hurt.

I’ve heard from countless pet owners that the death of their pet was worse than the death of their own parents. This might sound blasphemous to some, but to others it’s the cold truth. Making the decision to euthanize a pet can feel gut-wrenching, murderous, and immoral. Yes, those are strong words, but that is what our pet families experience. They feel they are letting their pet down or that they are the cause of their friend’s death. They forget that euthanasia is a gift, something that, when used appropriately and timely, prevents further physical suffering for the pet and emotional suffering of the family. Making the actual decision is the hardest part of the experience and I’m asked on a daily basis, “Doc, how will I know when it’s time?” Let me shed some light on this difficult discussion.

Quality of Life Scale
When evaluating quality of life, personalized patient and client information is needed to reach an educated, informed, and supported choice that fits not only their pet’s medical condition but also the family’s wishes. In short, quality of life applies not only to the pet; it also applies to the family!

Pet’s Quality of Life

Score each subsection on a scale of 0-2:

- 0 = agree with statement (describes my pet)
- 1 = some changes seen
- 2 = disagree with statement (does not describe my pet)

1. Social Functions
   a. Desire to be with the family has not changed.
   b. Interacts normally with family or other pets (i.e., no increased aggression or other changes).

2. Natural Functions
   a. Appetite has stayed the same.
   b. Drinking has stayed the same.
   c. Normal urination habits.
   d. Normal bowel movement habits.
   e. Ability to ambulate (walk around) has stayed the same.

3. Mental Health
   a. Enjoys normal play activities.
   b. Still dislikes the same things. (i.e., still hates the mailman = 0, or doesn’t bark at the mailman anymore = 2)
   c. No outward signs of stress or anxiety.
   d. Does not seem confused or apathetic.
   e. Nighttime activity is normal, no changes seen.

4. Physical Health
   a. No changes in breathing or panting patterns.
   b. No outward signs of pain. (See Resources Below)
   c. No pacing around the house.
   d. My pet’s overall condition has not changed recently.
Results:

1. 0 - 8 = Quality of life is most likely adequate. No medical intervention required yet, but guidance from your veterinarian may help you identify signs to look for in the future.
2. 9 – 16 = Quality of life is questionable and medical intervention is suggested. Your pet would certainly benefit from veterinary oversight and guidance to evaluate the disease process he/she is experiencing.
3. 17 - 36 = Quality of life is a definite concern. Changes will likely become more progressive and more severe in the near future. Veterinary guidance will help you better understand the end stages of your pet’s disease process in order to make a more informed decision of whether to continue hospice care or elect peaceful euthanasia.

Resources:

1. AAHA/AAFP Pain Management Guidelines for Dogs and Cats, [www.aahanet.org/Library/PainMgmt.aspx](http://www.aahanet.org/Library/PainMgmt.aspx)

Family’s Concerns

Score each section on a scale of 0-2:

- 0 = I am not concerned at this time.
- 1 = There is some concern.
- 2 = I am concerned about this.

_I am concerned about the following things:_

1. Pet suffering
2. Desire to perform nursing care for your pet
3. Ability to perform nursing care for your pet
4. Pet dying alone
5. Not knowing the right time to euthanize
6. Coping with loss
7. Concern for other household animals
8. Concern for other members of the family (i.e., children)

Results:

1. 0 - 4 = Your concerns are minimal at this time. You have either accepted the inevitable loss of your pet and understand what lies ahead, or have not yet given it much thought. If you have not considered these things, now is the time to begin evaluating your own concerns and limitations.
2. 5 - 9 = Your concerns are mounting. Begin your search for information by educating yourself on your pet’s condition; it’s the best way to ensure you are prepared for the emotional changes ahead.
3. 10 - 16 = Although you may not place much value on your own quality of life, your concerns about the changes in your pet are valid. Now is the time to prepare yourself and to build a support system around you. Veterinary guidance will help you prepare for the medical changes in your pet while counselors and other health professionals can begin helping you with anticipatory grief.

Basic Quality of Life Assessments

Let’s face it – some people just need an easy way to evaluate a pet’s quality of life. I’m not saying I agree with this method, but for some, this is all they can mentally handle during these delicate days.
The most traditional method is when you ask a family to record the top 5 favorite things of the pet and when they stop doing 3 or more of them, it is ‘time’. My apprehension to this method is that it does not take into consideration the pet’s ailment.

One twist I like to add to this is adding something that the pet hates to that list. There are certain things that just ‘bug’ our pets – and when they stop caring for those things, it can be a sign that they are simply tired and do not have the energy to ‘care’. My own dog hated the Goodyear blimp that flew over our house. The week he passed – he didn’t make a peep at it coming into his air space.

Another uncomplicated way to track quality of life is to get two jars – one labeled ‘good day’ and the other ‘bad day’. Have the owner put a penny in the appropriate day jar based on the pet’s behavior, habits, daily functions, etc. Then after a few weeks – you can see if the pet is having more bad days than good and it is probably appropriate to recommend euthanasia.

A much better quality of life scale was created by Alice Villalobos, DVM and is called The HHHHMM Scale. This takes into consideration hurt, hunger, hydration, hygiene, happiness, mobility, and more good days than bad. It can be downloaded by following this link: [http://www.pawspice.com/downloads/QualityofLifeScale.pdf](http://www.pawspice.com/downloads/QualityofLifeScale.pdf)

**Advanced Quality of Life Assessments**

After helping thousands of families with determining when is ‘time’ – I have realized that much of that assessment is ruled by the pet’s ailment. As mentioned above – the pet in heart failure is very different than a pet with arthritis. The questions that you evaluate are very different. Appetite in arthritis is not as important as it is in heart failure. Respiratory effort is vital in heart failure while not so much (except for painting due to pain) in arthritis.

Due to this – the questions I have owners ask everyday is based on the ailment. Lap of Love has created an online interactive tool that owners can use to evaluate their pet’s quality of life. They create their pet’s profile and choose from a variety of ailments. Based on the ailment selection, the questions and parameters they evaluate are different.

This tool is free for vets and the public at large and can be found at [www.pethospicejournal.com](http://www.pethospicejournal.com)

Using this scale in conjunction with the family’s quality of life has helped many owners feel empowered over their decisions – whether to continue or euthanize their pets.

Suggestions on using any quality of life scale:

1. Complete the scale at different times of the day, note circadian fluctuations in well-being. (We find most pets tend to do worse at night and better during the day.)
2. Request multiple members of the family complete the scale; compare observations.
3. Take periodic photos of your pet to help you remember their physical appearance.

**Summary**

How I wish the answer to the question ‘when is time’ was simple and clear cut – however, it is not. It is our duty to assist owners with end of life decisions and to help end and prevent suffering of animals. There are many ways to help families explore quality of life questions but the one way that is an injustice to our profession is if you simply say, ‘Call me when it’s time’. Owners need more than this and animals deserve more.
Euthanasia under pressure – when the S hits the fan

Mary Gardner, DVM
Lap of Love Veterinary Hospice - Nationwide

We are not taught to be good at death. No one taught me how to walk into an exam room for a euthanasia, what to say to a crying teenager, or whether or not to hug the old man that just lost the last piece of his late wife. But even moreso – we are not trained well on how to handle the worst situations – like when the S hits the fan.

So what could hit the fan?

1) You can’t find a vein
2) The pet has a vocal reaction to the sedation or the euthanasia itself
3) The pet has a physical reaction to either
4) You have the emotionally challenging clients

Many veterinarians get concerned when they can’t find a vein – but knowing all the routes possible will help relieve the pressure of always obtaining IV access.

The AVMA Euthanasia Guidelines allow for other routes of pentobarbital administration (with unconscious sedation only):

1. Intra-cardiac – If needed, gently place your hand over the thoracic cavity and say “I’m going to give this in a central vein that will bring it directly where it needs to be.” Shield needle and syringe from the family with your other hand. Aim more cranial and ventral than you think and leave room in the syringe for air and/or blood. 1 mL per 10 lb is recommended. (Cooney 2012)

2. Intra-renal – this is a standard protocol for cats by many in-home euthanasia veterinarians. Say “I’m going to give the second injection through the abdominal cavity into a large vessel, it generally takes anywhere from a few seconds to a couple minutes.” 80% will pass before you have finished giving the full injection. Give 3 mL per 10 lb in the cortex, even in the smallest of kidneys (most skilled practitioners will recommend 6 mL). (Cooney 2012)

3. Intra-hepatic – if needed, this is a good alternative to intra-peritoneal as it causes death in 2-5 minutes. Explain “I’m going to give the injection near a highly perfused organ, he’s going to pass away in just a few minutes.” Use 2 mL per 10 lb and aim cranially just under the xyphoid process. (Cooney 2012)

4. Intra-peritoneal (pre-sedation not required at this time) – there is some evidence that abdominal irritation from barbiturate injection (Wadham 1997), but IP is still a good alternative, especially for fractious cats. 3 mL per 10 lb is recommended (Cooney 2012)

A natural passing doesn’t always happen very quickly either. I have had many frantic phone calls from people wanting me to rush to their home because they wanted a natural passing for their pet but the process is taking too long or not very peaceful. The pet might start having a seizure, they may start to choke or they may have difficulty breathing. This is not easy to watch or let your pet go through and people need to be prepared for this. What happens if your pet is alone during this traumatic time? I wouldn’t want to be alone during my final moments, so families with pets that are near the end should have someone with them at all times to make sure they are not suffering.

Points to cover with families during the dying process:

- It can happen at any time – day or night
- The dying process can take hours
• The pet may stretch, put their head back, yawn, seize and even vocalize
• Afterwards, they will first have a period of muscle relaxation (including the release of urine and feces) followed my rigor mortis
• Plan for aftercare – provide the family with information on how to handle the body after including information on burial, crematory information or a local emergency clinic that will arrange cremation.

Sometimes the ‘S’ that hits the fan is about how you feel the family is reacting to the situation. I have found there are 4 main types of client reactions/behaviors outside the ‘norm’ (if there can be a norm during such a sensitive time).

Please note:
• these are just general classifications. Not many clients will fit into one tidy box! But recognizing and reading clients’ general needs, wishes, and goals can help us to better serve them and give them the experience they want.
• ALL clients are grieving. They cared enough to call us, and that means something.
• Individuals display their grief in different ways
• All clients need and deserve our respect and care
• There may be different client types within one family unit
• Don’t take any behavior personally; it really is not about you.
• Meet the client where they are: guide them and respect them.
• Remember: your goal is to provide their pet with a peaceful passing at home. And that is exactly what you are doing.

1) THE ACTIVE COMMUNICATOR (AC)
• The most (obviously) interactive type
• May tell a lot of stories about their pet or just want to talk in general
• May appear the warmest of the types
• Generally, love all that you do and make sure to TELL you that
• Likely to be open and show their grief
• Although many are talkative, not all are. The less talkative AC types will be able to clearly communicate how much interaction they want and need.
• Be aware: This type of client can sometime keep you at the appointment longer than needed, (or for a longer period than you have) simply because they want to connect.
• Best ways to approach:
  • Actively listen to their stories about their pets, ask questions, and “go with the flow” as far as conversation is concerned
  • Remember to guide the appointment along and watch the time. The goal is to help this person feel heard and for them to feel connected to you

2) THE VIGILANT OBSERVER (VO)
• Often quieter than the active communicator, but usually pleasant
• Watchful and very interested in the process of what you are doing
• May ask a lot of technical questions, be concerned, or want to know about the process after euthanasia (i.e. details about body transport and cremation)
• May have had a previous “bad” experience with euthanasia
• May be anxious and worried about the process
• Likely to show their grief
Typically grateful to you, most especially if the appointment went better than they expected.
Be aware: Can become controlling in a negative way or try to lead the appointment.
Best ways to approach:
Explain more details as you see fit: this client may need more information than usual in order to feel more comfortable
Reassure this client throughout the process

3) THE PRIVATE MOURNER (PM)
- This individual wants minimal interaction with you; does not share stories or talk much
- Prefers you communicate only what is needed about the appointment
- For the PM, silence is golden
- May request no further contact or decline emails and remembrance items
- Although may be experiencing strong grief, may be less likely to demonstrate it or may prefer to show grief privately
- Be Aware: Can appear aloof or like they do not connect with you
- Best Ways to Approach:
  - Be comfortable in the quiet: do not talk extra unless you can improve upon the silence
  - Explain the process as you would for anyone but respect their desire for less verbal communication otherwise.

4) THE STRAIGHTFORWARD CONSUMER (SC)
- May approach the visit as a business transaction
- Shorter visits often suit them best
- May seem less attached to pet than others (although this may not be the case at all!)
- May decline further contact or memorial items
- Most likely to choose to NOT be present for all or part of the process
- Unlikely to show a lot of emotion in front of you. If does show emotion, may be more apt to try to hide it, or feel embarrassed/shrug it off
- Be Aware: You may not feel as connected to this type of client, but this does not mean they aren’t grateful to you for helping pet.
- Best Ways to Approach:
  - Refrain from judgment or taking the interaction personally
  - Treat pet and client with kindness and respect as you would any other

In this lecture we will cover all the routes of euthasia and some other ‘s’ hitting fan situations!

References:

Resources:
Treatment and Care of the Geriatric Veterinary Patient, Gardner & McVety, Wiley Publishing
Veterinarians have a variety of tools to keep puppies and kittens healthy as they grow, and we are well prepared to help our aging patients as they reach their senior years. The care and management of a geriatric pet, however, is very different for both the patient and the owners alike. As pets reach advanced ages and enter into this last life stage, owners are faced with a myriad of physical and emotional concerns (for both the pet and themselves). There is so much more that can be done within the veterinary profession to properly recognize this geriatric stage, keep the patient comfortable, and help owners deal with their delicate, aging family members and the toll that it can take on them.

When thinking about the human side of medicine, Geriatricians have deal with three major things: 1) Polypharmacy, which includes all the different drugs the patient has to take, 2) Dementia, which is not only a massive problem in humans but also a big problem in the veterinary world. 3) Caregivers, helping them with their geriatric family members. This is actually one of the things we deal with the most with our geriatric pets. The difference in humans between a senior and a geriatric is that a senior is 65. It does not distinguish how they’re acting or the physical abilities they may have. It’s just a number. A person who is geriatric or has geriatric syndrome, basically means they’re susceptible to adverse outcomes and increased risk. When your grandmother gets admitted to the hospital because she fell and broke her hip, you worry about other things like her possibly catching pneumonia.

A person may be considered geriatric if they meet three of the following criteria. They’re experiencing weight loss, slowed mobility, fatigue, exhaustion and/or low levels of activity. Of these, weight loss and exhaustion seem to develop later in life. So, similarly in veterinary medicine, when we see skinny old dogs with mobility issues we worry about them.

LIFE STAGES:

When we think about our patients there are four life stages that we cater to. The puppy/kitten stage, the adult stage, the senior stage and the geriatric stage. We tend to not segregate senior from geriatric but there are some big differences. A nine-year-old lab that comes in wagging his tail is considered senior but the wobbly 12-year-old lab that comes in the door is different. Before and during this last stage, there is much we can do to help pets live a comfortable life as a geriatric.

We’re seeing a lot of older pets now due to better medicine, better nutrition and the strong bond that we share with them that has changed over time. So, we’re going to see a lot more of the geriatric stage then we ever did before. When we think about the age of a pet in the geriatric stage we think of a Great Dane that’s 9 years old. But a smaller dog may start to be considered at 12 years old or more. In general, we might consider the last 15% of the lifespan of a pet to be in the geriatric stage, whatever size they may be. A cat’s geriatric stage can be considered even later in life.

The AVMA reported that 30% of their client base had pets between 6-10 years old and another 15% had pets over the age of 10. That’s about 45% of your client base considered having pets considered senior and geriatric. We tend to concentrate on marketing to puppies and kittens and adults.

VetSuccess, a data intelligence company, has data from thousands of vet clinics around the country. They report that the percentage of pets in a clinic’s database within the puppy/kitten life stage are only 5%. The adult stage is around 30%. The senior stage is around 15% and the geriatric stage comes in at 48%. So, make sure to keep in touch with those senior and geriatric patients.

When we look at the percentage of pets that we euthanize that we have seen in the past 18 months, only 23% of cats 12 years and older have been seen by their vets within that time frame. That’s 77% of your
client base of pet owners with cats not being seen in the last 18 mos. Only 30% of dogs that are 10 years and older have been seen within the past 18 months. That’s 69% of your total client base that have dogs.

When you think of the number of patients in your database that are geriatric, that’s a lot of missed opportunity to help and revenue. Pets that are in the geriatric stage are the ones that we need to see the most. And the family need the visits as well.

**Practical Care in the Home**

A home visit can be massively helpful when assisting a family with a plan to manage their pet. But for many veterinarians, that is just not something that can be done. You can ask families to bring in videos and pictures of the environment to help you look for trouble spots, identify areas of opportunity or confirm that the family is doing a great job with their pet!

Mobility is the most common ailment we manage at Lap of Love – particularly with the larger breed dogs (but it does effect cats as well). Over half of the homes I enter have the main living area covered in tile flooring – with no solid traction areas for the dog to walk securely on! This is the ice rink of death for a dog with mobility issues. You will be amazed at what a simply ‘run way’ of yoga mats or bath mats can do for a dog! Harnesses that can be worn at all times is also a massive benefit. Yes – a beach town is good – but try getting that under a big dog in time to assist them. Traction booties, Pawfriction, toe Grips are all highly recommended products.

Safety is paramount with these order grey muzzle pets. Think about stairs (even 1 or 2 can cause a tragic tumble), pools or lakes, areas they can get stuck in and also young children that may not understand the concept of a pet in pain.

**Care for the Client**

Not until I dealt with the struggles of caring for my own geriatric pet, did I fully appreciate the toll it placed on me. You love your pet, you will do what you need to do – but it’s hard physically and emotionally. With our pets we also have the burden of deciding to euthanize when appropriate. And that in itself is a heart wrenching decision and pet owners need our guidance.

**Caregiver Burden:**

A cross sectional observational study was done by Spitznagel, Jacobson, Cox, Carlson entitled “Caregiver burden in owners of a sick companion animal: a cross-sectional observational study” . This was the first study that examines the toll of caregiving on pet owners. It measured the mental health of owners by monitoring levels of depression, stress and anxiety as well as Quality of Life enjoyment for the owner. (Comparable measurements have been studied in human caregiving relationships to assess similarities)

The results showed that caregivers of terminally or chronically ill pets had:

1) Greater level of caregiver burden and stress
2) Greater perceived stress
3) Greater symptoms of depression and anxiety
4) Lower on indicators of quality of life and enjoyment

Symptoms of Pet Caregiver Burnout are:

- Withdrawal from friends, family, and other loved ones
- Loss of interest in activities previously enjoyed
• Feeling blue, irritable, hopeless, and helpless
• Changes in appetite, weight, or both
• Changes in sleep patterns
• Getting sick more often
• Feelings of wanting to hurt yourself or the person for whom you are caring
• Emotional and physical exhaustion
• Irritability

We need to appreciate this feeling of burden and find ways to support them through education, respite care, helping them with ways to manage their pet, etc.

**Anticipatory Grief:**
The death of a pet is, for many, the worst personal loss they have ever experienced. Complicate the event with the possibility of euthanasia and the emotions can be too much for some pet parents to bare. How, when, and why veterinary professionals can make a difference at such an important time is essential to maintaining not only the human-animal bond, but the doctor-client bond as well. There is no better time to show your clients you care than by helping through the difficult journey of pet loss.

**In Summary:**
As a profession we have been well educated and equipped for caring for the senior pet. For those fragile, advanced aged geriatric pets there is an opportunity to provide better care as they enter their golden years and support the families as they struggle alongside their pet.

**Resources:**
Geriatric Questionnaire: [https://www.lapoflove.com/Education/Geriatric_Veterinary_Patients](https://www.lapoflove.com/Education/Geriatric_Veterinary_Patients)

Products to recommend: [https://www.amazon.com/shop/drmarygardner](https://www.amazon.com/shop/drmarygardner)

Treatment and Care of the Geriatric Veterinary Patient, Gardner & McVety, Wiley Publishing
The euthanasia you don’t want to do

Mary Gardner, DVM
Lap of Love Veterinary Hospice

To become a veterinarian, we take an oath to not only stop suffering but prevent it from occurring. Most veterinarians accept that performing a euthanasia on a pet that is gravely ill is something that we must do to uphold that oath and prevent the further suffering for a pet.

But there are some euthanasias that tend to pull on our moral or ethical heartstrings. The euthanasia that keeps us up at night, the one we regret we ever did or the one that you hope you never are faced with. Often we label these as ‘convenience euthanasia’ but that may be a broad umbrella that many are put under when in fact it’s not ‘convenient’ at all.

I find the definitions that Dr Dani McVety put together to be helpful to categorize these euthanasias:

**Euthanasia Definitions**

- **Convenience euthanasia** is a very subjective term. We use this phrase when euthanasia is requested for a pet that would otherwise be deemed adoptable under most circumstances and the family is unwilling to explore these options. For example, “my pet doesn’t match the decor in my home any more” (yes, I’ve heard this). Personally, I do not offer convenience euthanasia in my practice, we offer support and resources to re-home these pets.

- **Non-medical euthanasia** is a term I use when describing a request that is not related to the medical stability of the pet. This is a broad term that includes behavior issues (such as aggression or improper elimination in the home), in addition to emotional or lifestyle changes of the family that precludes the pet from experiencing a quality of life.

- **Non-imminent medical euthanasia** is a term that describes situations like the 12 year old cat. These conditions may be manageable or even curable under the right circumstances, but for whatever reason, those circumstances do not exist. This includes the parvo puppy that may survive with intensive care, the 5 year old intact female with a pyometra, or the young cat with a broken leg. Without the right resources and conditions (which may be too expensive), this pet would potentially suffer greatly. Rarely will I turn down this type of euthanasia request.

- **Medical euthanasia** describes most of the euthanasias that occur in our clinics; a choice that is made when the quality of life of the pet is deemed unsustainable by both the family and the veterinarian.

As mentioned, most veterinarians are ‘ok’ to perform a medical euthanasia – and that is the most common that we face. But the other three do pop up.

I think it’s most important to first listen to the family and understand the full story to the situation. They may be facing personal challenges that make the situation even more severe than normal. Know what your local shelter and rescue situation is – are they overwhelmed, do they have a high kill rate, do they take behavior animals, etc. Know that adoptable and treatable are very subjective terms. And what we as veterinarians may find acceptable, most other humans do.
Having a guideline to follow when faced with these situations will be helpful so that you’re not put off guard. I myself have done a handful of euthanasias that I wish I had not done – but after each one, I made my own personal rules or boundaries so if I am faced with them again, I will have better options available.

Dr McVety also has the following ‘rules’ which are something to consider:

**Non-Medical & Convenience Euthanasia Rules to Live By**

- Do not euthanize a pet that you do not feel comfortable euthanizing. Period. (But say “no” carefully, keeping these other rules in mind.)

- Always help the family explore alternative options and think about how those options will effect the family and the pet down the road. Remember that a shelter is the deadliest place for a pet to be. Write them down, discuss them, think about what effect those alternatives have on OTHER animals in society.

- If you are comfortable euthanizing, even if you don’t completely agree, you must help the family understand that although this is difficult for you (and them), you care greatly for them, their pet, and that this is the best decision that can be made given the circumstances. You do not want them to feel judged, which could lead to a lifetime of guilt.

- Do not get involved in cases if you don’t plan to help, you will do more harm to our profession by judging and berating clients that if you simply hand them a number to a different veterinarian (preferable), or at least the local shelter or rescue organization.

In the lecture we will go over case studies representing the most common types of euthanasias you just don’t want to do.
FOCUSING ON GERIATRIC PATIENT MANAGEMENT

Our abilities to recognize and manage pain, anxiety, hygiene, and other symptoms that may limit quality of life has advanced in recent years and our profession is seeking ways to identify these unique client and patient needs, communicate effectively, set realistic expectations, and help guide pet parents with the care and management of their aging geriatric companion animal. These tools can empower the veterinarian to embrace the geriatric pet, know how to handle the symptoms that plague them as well as assist owners with the care and management.

The goal of proper and effective geriatric pet care is to enhance the quality of life for the pet and the owners, empower them to properly care for their pet during this delicate life phase, and maintain the strength of human-animal bond.

During geriatric consultations, which can often times really be hospice consultations, we deal a lot with pain. Weakness, anxiety/fear, panting, pacing and hygiene can all be problems the pet is having. They may be isolated because they’re separated from their families or they’re having trouble handling being alone for even just a few hours a day.

You often times hear that “Old age is not a disease”. Well we may not be able to diagnose everything the pet is experiencing but we know that they do struggle. And the problems that they get as they get older do affect their quality of life and their family’s quality of life.

When thinking about older pets there are three main categories we think of:

1. Old but healthy- They don’t really have any major disease processes going on with them but normal old age changes.
2. Old with subclinical organ dysfunction- They might come in for a dental but also have kidney malfunction.
3. Old with overt disease. There are often multipole comorbidities. Your blind, diabetic patient that you see that’s arthritic and urinating around the house.

Geriatric Wellness Plan

In our clinics we have wonderful puppy and kitten packages, but remember that they only account for about 5% of our client base. Similar to wellness plans for younger patients, clinics can create Geriatric Wellness plans to encourage owners to consistently bring their pets in for exams. Bundling services and avoiding services that may not be necessary at this life stage is the foundation. An example of bundling services is offering 4 visits per year for a discounted rate (i.e., if your typical office visit cost is $45– offer 4 visits for a discounted rate of $135 instead). At the geriatric stage, diseases and symptoms progress fast; thus, warranting the need for multiple visits a year. Bundled service discounts are a great way to maximize compliance for pets in need by incentivizing for a visit every quarter. Allow 30 min for your consultations and provide a geriatric questionnaire prior to their appointment. Find grey muzzles! Start by flagging geriatric pets in your practice management software and running reports every six months.

Offering unique services is another component of a Geriatric Wellness Plan. For instance, geriatric pet sitting, monthly “sanitary shaves”, Fear-Free nail trims, laser therapy, physical therapy, and geriatric boarding/day care are a few ideas that can be incorporated into the plan.
At this stage in life, many pets will also need specialized accessories or products to help manage their daily activities. This can be done by offering a retail space within the clinic, or if that is unfeasible, simply by providing information sheets to clients on useful items and where to order them.

It’s important to remember that it’s not just how old they are but HOW are they? How are they doing? How are they being managed? How are the families doing?

**Education and Marketing**

Lack of education or simple awareness of issues will keep clients away from clinics.

Creating the content for education is the first step. The main areas to focus on (individualized for each species) are:

- The aging process (it’s not just 7 yrs to our 1 yr) and symptoms
  - Geriatric Questionnaire http://lapoflove.com/Education_/Geriatric_Questionnaire.pdf
  - The Top 10 ailments/symptoms pets struggle with. For example:
    - Mobility
    - Senility
    - Sleep Disturbances
    - Energy levels
    - Pain
    - Heart Problems
    - Kidney Problems
    - Dental Problems
    - Vision Loss
    - Cancer
- Assessing Quality of Life
- Specialized Services offered in your clinic (laser, acupuncture, rehabilitation, processes different for the geriatrics)
- Titers vs Vaccines
- Caregiver Fatigue and Support
- Saying goodbye

**Awareness**

Many owners are in the dark when it comes to the options they have available to them – but if they aren’t coming into your practice how can you tell them? The solution is you must make them aware with marketing!

- Website: Most clinic websites have information on puppies and kittens and also senior – but rare are the websites that have information on geriatrics or quality of life assessments.
- Newsletters: If your clinic sends newsletters, include articles about the grey muzzles
- Blogs and Vlogs: Dedicate at least 30% of those to geriatric issues.
- Paid Ads: If you are spending money on ads for vaccines, push some of that budget to ‘grey muzzle’ ads
- Social Media: Dedicate posts to the geriatrics on all your social media outlets.

**Changes in Your Clinic:** Let it be known to all that enter your clinic that you cater to the Geriatrics.

- Have a ‘Grey Muzzle’ parking spot
- Specialized boarding with daily texts/pictures, personalized service (owners are very worried about leaving their geriatric pet in anyone’s care), including yoga mats or bathmats in the rooms, etc.
- Have policies and standards that are specific to the geriatrics.
Holding techniques, anesthesia procedures/monitoring, in-patient care, venipuncture, etc. are all different with the geriatric pets.

- Everyone in the clinic should know the differences of an advanced aged body and what your processes are for them.

In Summary:

The clinic can be a scary place for a pet – especially a fragile one. Making sure that they are physically and mentally safe, handled well/carefully, and treated respectfully is key. Sadly we are missing out on helping a huge pet population and we can do so much good if we actually saw them. Create a marketing plan to get them in to see you – then treat those grey muzzles well, the families will be so grateful.

Resources:

Blunt Dissection Podcast by Dr Dave Nicol – Episode 16 focuses on geriatric anesthesia techniques. [https://www.drdavenicol.com/blunt-dissection-podcast](https://www.drdavenicol.com/blunt-dissection-podcast)

Geriatric Questionnaire: [https://www.lapoflove.com/Education/Geriatric_Veterinary_Patients](https://www.lapoflove.com/Education/Geriatric_Veterinary_Patients)

Products to recommend: [https://www.amazon.com/shop/drmarygardner](https://www.amazon.com/shop/drmarygardner)

Treatment and Care of the Geriatric Veterinary Patient, Gardner & McVety, Wiley Publishing
Top age-related ailments to educate clients about

Mary Gardner, DVM
Lap of Love Veterinary Hospice

THE AGING PROCESS

Aging is the inevitable decline in the body's resiliency both mental and physical. Over time, cell production decreases, leaving fewer cells which are less capable of repairing wear and tear on the body. The immune system is compromised and therefore more susceptible to infections, less proficient at seeking out and destroying mutant cells, many older pets succumb to conditions they could have resisted in their youth.

The aging process is incredibly complicated and it can be difficult to distinguish between changes that are the result of ‘age’ and those that come with common medical conditions.

Below are some of the more common age related issues that plague our geriatric patients:

Eyes:
Lenticular/Nuclear Sclerosis: All geriatric dogs (starting at about 6-7 years old) develop a hardening of the lens. However, it does not become noticeable until about 10. The lens is added onto throughout life, gaining layers of protein. As the new layers of protein are added, inner layers are compacted together and become harder. The hardening of the lens fibers makes it difficult for the lens to change shape – needed for focusing. Near vision is therefore reduces – just like in middle-ages people who need reading glasses. Pets become hesitant going down stairs and more difficulty when catching small treats or toys. You can help them with simple things like illuminating the stairs with tea lights or attaching scent trackers to vital places around the house.

Ears
Presbycusis, also known as age related hearing loss. Mid to high frequencies are affected first, followed by progressive loss at all frequencies. Onset is typically in the last third of a breed’s typical lifespan and will eventually progress to complete deafness.

Four types of presbycusis are described in humans and in dogs but the most common seen is the sensory presbycusis which is characterized by loss of hair cells and degeneration of the organ of Corti.

Although the loss is progressive, owners usually report an acute onset because of the ability of the animal to compensate for hearing loss until nearly complete deafness occurs. Age related hearing loss most often occurs in both ears, affecting them equally.

Skin:
An older animal’s skin and hair may look dull and lusterless due to the decreased production of natural oils by the sebaceous glands. This can also cause the skin to appear dry and flaky. Continued brushing will help stimulate the skin to produce the oily secretion and an excessively dry coat may benefit from implementing a fatty-acid supplement. The skin also loses elasticity as pet’s age and is more susceptible to infections. The worst side effect of a skin infection is that the pet smells and therefore is shunned out of the bedroom or living area.

Muscles – Can’t get up or down easily:
Sarcopenia is defined as the progressive loss of lean body mass in aging animals in the absence of disease. As muscle tissue mass decreases so does muscle strength which is why older people are less steady or have difficulty catching their balance. Our pets may exhibit similar signs such as changes in their movements reflected in difficulty getting up or reluctance to jump up. To help with this feed a high
protein diet and provide them with plenty of slow and steady exercise. Keep on moving! Several short walks per day can really help!

Lungs:

The elastic fibers in a dog’s lungs allow them to expand and contract with each breath. As a dog grows older, some of these fibers are replaced with fibrous scar tissue diminishing the ability to breathe as efficiently as possible. Pet owners should recognize that an older animal can’t exercise in extreme temperatures as well as they did when they were younger. Jogs or walks with your pet may need to become slower or shorter as they progress through their older years.

Trouble at Night – Panting and Pacing:

Some older dogs may become restless at night and stay awake pacing throughout the house or panting. There are many reasons an older dog may have difficulty sleeping at night including both medical and anxiety or behavioral related causes. Dogs do get cognitive dysfunction which is similar to dementia in people. Cognitive dysfunction is also referred to as sundowner syndrome and is categorized as a slow, degenerative and progressive disorder in our aging pets.

Sundowning is a syndrome in Alzheimer’s patients of recurring confusion and increased agitation in the late afternoon or early evening. The hallmarks of this syndrome in dogs are progressive confusion, reversal of day-night wake-sleep patterns and poor adaptability to new situations. The exact reason for this change in our geriatric pets is unknown.

This is just the tip of the iceberg when it comes to the ailments and common symptoms our pets face when they age. Telling an owner ‘Old age is not a disease’ is not the right thing to do during this time. Instead taking the time to listen to the problems the pet and care giver are facing, going over the causes and possible treatment options are key to helping manage the aging pet. Some of the simplest ideas that you can give pet owners may be the most helpful and they will appreciate you so much more. Caregivers go through quite a bit during this time and it can take a toll on them physically and emotionally. They may not be able to think of some of these simple ideas that will make such a difference in their daily lives along with their pets.

Providing in home evaluations can also provide you with insight to how the pet manages in their home and also how the owner is managing the pet. Both are very important. In-home evaluation: Provide suggestions for reorganizing the household for senior pet mobility/safety, such as barricading stairs, moving food bowls, using nonslip surfaces, improving traction by shaving hair between pads or using traction booties.

Sanitation:

Many pets have sanitation issues. Diapers or Chux pads (“puppy pads”), waterproof bedding (baby mattresses are an alternative to expensive dog beds as they are waterproof), baby powder, waterless shampoo, and shaving hair around the perianal area help keep pets clean and comfortable. Keeping the pet’s mind active and alert can make a huge difference in quality of life. Owners can simply change typical pet games: Instead of tossing the ball in the back yard, roll the ball to the dog while he is in bed. Long walks can be replaced with an inside activity, such as “hide and seek,” a game many dogs enjoy, or simply short frequent walks around the house to maintain core muscle. Pets with a high food-drive may love a Kong toy (kongcompany.com) filled with their favorite treats or unique bowls (aikiou.com) that encourage them to seek out food in compartments.

Summary:

Aging happens to all of us – and it carries with it many struggles. Recognizing the changes in the body and educating the client on how to care and manage them will provide a much better caregiver experience.

References:

Treatment and Care of the Geriatric Veterinary Patient, Gardner & McVety, Wiley Publishing
What you Need to Know About Cremation
Mary Gardner, DVM
Lap of Love Veterinary Hospice

What a pet owner elects to do with the remains of their pet is a personal decision. There is not ‘right or wrong’ and one option doesn’t mean a family loves their pet more or less. But if a family elects cremation, it is our duty to have done our due diligence to select a respected and trustworthy crematory.

The most important partner you will have with the end of life process is your pet crematory. They are not just handling the bodies of the pets – they are handling someone’s best friend and the physical remains of patients that we have grown to love. Regardless of whether a family would like the ashes back or not the pet should be treated with the utmost care and respect after the euthanasia procedure. That starts with your clinic and how they are handled. I have heard horror stories about crematories, but I have witnessed horrors IN clinics. Not only should we be respecting the pets and the families we serve but we should also be mindful of how our co-workers deal with their grief. We all deal with the euthanasia appointment differently and so we must be respectful to everyone around us. Less than 30% of the clinics I know have actually gone to the crematory they use. Personally tour the crematory I am going to send pets to as I want to ensure that they have the best processes for care and control of the pets. This is so important to me that I ended up opening my own crematory in South Florida! (monarchpetservices.com)

Some things to consider when evaluating a crematory:

1) How is their customer service – how they answer the phone and handle calls is important as many owners will call the crematory to find out where their pet is, where ashes were spread, or even ask them about the process. The need confident compassion when handling the types of questions that they will face.

2) What are their hours and do they offer body pick up. Often a pet may die at home and the family would like the crematory to pick up from the home. Does the crematory offer that service – if they do – how much and what days/hours. But more important – is how they appear, how the vehicle looks and are the drivers respectful. I have seen open bed trucks with garbage cans in the back and the crematory did not ‘see’ any problem.

3) Viewing Options – there will be on occasion when a family would like to view the pet before cremation. Is the crematory prepared to offer that service and for how much? Some families want to simply check out the retort to confirm their pet is the only one inside. (If you know a family wants a viewing – this may change the way you store the body)

4) Types of cremation and the process:
   a. Communal/Group cremation – this simply means that more than one pet is placed in the chamber at one time with no separation – typically piled on top of each other. Ashes are not returned to the owner and usually the crematory will dispose of the ashes. Some may spread them on a ‘garden’ or some other location, but it is important to get full details. Ashes can pile up – I have a hard time believing that ALL the ashes are spread where some crematories say they spread them. Maybe they only take a portion of communal ashes there. Often they are thrown away with the regular trash. No ‘right or wrong’ here – just make sure you know what your crematory does and that you can stand behind that process.
   b. Private or Individual – these are two separate processes. The end result is that the pet’s ashes should be returned to the owner. The difference is that one is when the pet is completely ALONE in the chamber with no possibility of cross contamination. The other is when multiple pets are placed in the retort but are separated by some means. It may be a tray, a small wall of bricks or simply placed in different corners. There is a confusion of which process has what title (Private or Individual) – some people say ‘True Private’ when they are alone in the retort. Again – just understand the process
c. Identification – for the private/individual cremation, know how well the crematory tracks the pet’s remains. Some use metal tags that are left behind, some have other very strict processes. When you visit the crematory – make sure you are comfortable with their identification process.

Memorial Package –
Crematories should all offer clay impressions (of paw or nose) – you will need to know if they put the name in the clay or not as families may ask that. At my crematory, we also do an ink impression and take a sample of fur (or feathers).

Cremation certificates are often offered which usually gives the family some comfort that their pet was privately cremated. Some crematories will add the ‘date of death’ on it so make sure you supply the accurate information.

There is usually a default urn package that your clinic will offer which is a good idea. Some families are not ready to make a decision on an urn. Crematories may supply you with a catalog of options which can be difficult to go over in the clinic. I personally direct families to a website with options so they can make the decision on their own time.

Aquamation
More and more clinics are adding crematories to their practice. There is much information about fire-based cremation but aquamation is another great option – particularly for smaller clinics as it takes up less space – and well – is not as hot (fire cremation requires a large area and can become very hot and dirty).

Aquamation is a relatively new type of cremation is gaining traction in the United States. It is a safe, natural and environmentally friendly alternative to flame-based cremation or burial. It produces no toxic air emissions and uses a fraction of the energy of traditional cremation. In fact, out of all the options available, Aquamation leaves the smallest carbon footprint on our planet.

Aquamation is also known by the following terms: Bio-Cremation, Alkaline Hydrolysis or Green Cremation.

How does Aquamation work?
Aquamation is a gentle, quiet, and eco-friendly process using a combination of flowing water and alkalinity to accelerate the natural course of tissue breakdown that occurs during burial in the earth. Because there is no burning in Aquamation, there are more remains left after the process has finished. This remaining compound is mostly made up of calcium from the body (bones, teeth, and other minerals). This “bone ash” is softer, whiter, and lighter than with traditional flame-based cremation.

What is the difference between aquamation and other choices?
Whether a family chooses burial, flame-based cremation, or Aquamation, the end result is the same; each body is eventually reduced to its basic elements of bone ash. The primary difference between burial, flame cremation, or Aquamation is the amount of time the process takes as well as the “catalyst” that supports the transition. Flame cremation creates smoke while the remaining by-product of Aquamation is made up of nutrients, amino acids and sugars.

What can a pet parent expect?
As described above, Aquamation allows for a slightly larger quantity of bone ash when completed. Therefore, two pets of similar weight and build cremated two different ways will have differing amounts of remains (Aquamation will be more).
Example installation at a pet crematory.

System operator removing the top screen, ready to collect the bone remains after a cycle. This video shows the process: https://youtu.be/7gl6b4zLMf4

Every Practice should:

- Partner with a reliable and trustworthy crematory
- Schedule annual visits to the crematory to ensure your highest standards are met
- Know exactly what happens with each type of cremation (private, individual and communal)
- Understand their abilities to provide additional service if needed (pickup from home, viewing, etc.)

Resources:

International Association of Pet Crematories: https://www.iaopc.com/
The Human Animal Bond: Is It Possible to be Over-Bonded?
Steve Dale, CABC
Stevedalepetworld.com
Chicago, IL

What is the human animal bond?
“The human-animal bond is a mutually beneficial and dynamic relationship between people and animals that is influenced by behaviors essential to the health and wellbeing of both. This includes, among other things, emotional, psychological, and physical interactions of people, animals, and the environment. The veterinarian’s role in the human-animal bond is to maximize the potentials of this relationship between people and animals.”
American Veterinary Medical Association:
https://www.avma.org/KB/Resources/Reference/human-animal-bond/Pages/Human-Animal-Bond-AVMA.aspx

Anthropomorphism: Encyclopedia Britannica
https://www.britannica.com/topic/anthropomorphism

The attribution of human characteristics or behavior to an animal, or object

Human Animal Bond Research Initiative  https://habri.org/
Committed to supporting scientific research to substantiate what many of us know to be true, that humans and pets share a special, mutually-beneficial connection.

People are happier and healthier in the presence of animals. Scientifically-documented benefits of the human-animal bond include decreased blood pressure, reduced anxiety, and enhanced feelings of well-being.

Human Animal Bond Certified
https://learningacademy.navc.com/

The Science Behind The Human-Animal Bond
https://habri.org/research/
Positive human-animal interaction appears to be related to changes in physiological variables both in humans and animals, particularly dogs. HAI has been shown to influence levels of blood pressure, heart rate, hormones correlated with well-being including cortisol, oxytocin, b-endorphin, prolactin, phenylacetic acid and dopamine.

Oxytocin
Oxytocin is a neuropeptide long known to promote maternal care in mammals. The oxytocinergic system has been linked directly to many of the observed psychological effects of human-animal interaction.

- Oxytocin is recognized for its role in bonding, socialization, and stress relief.
- Oxytocin causes many physiological changes, including slowing heart rate and breathing, quiet blood pressure, inhibiting stress hormones, and creating a sense of calm, comfort and focus.
- Studies have demonstrated that human-animal interaction increases oxytocin levels in the brain.[1][2][3]
- The reduction of subjective psychological stress (fear, anxiety) due to animal contact, as well as the dampening of physiological stress parameters in connection with activation of the oxytocinergic system represent a core mechanism in explaining many of the positive effects of HAI.[4]
- Studies have also demonstrated that oxytocin levels are increased in dogs interacting with their own owners versus strangers.[8]


Cortisol [https://www.webmd.com/a-to-z-guides/what-is-cortisol#1](https://www.webmd.com/a-to-z-guides/what-is-cortisol#1)

Cortisol is nature’s built-in alarm system. It’s your body’s main stress hormone. It works with certain parts of your brain to control your mood, motivation, and fear.

Your adrenal glands -- triangle-shaped organs at the top of your kidneys -- make cortisol.

It’s best known for helping fuel your body’s “fight-or-flight” instinct in a crisis, but cortisol plays an important role in a number of things your body does. For example, it:

- Manages how your body uses carbohydrates, fats, and proteins
- Keeps inflammation down
- Regulates your blood pressure
- Increases your blood sugar (glucose)
- Controls your sleep/wake cycle
- Boosts energy so you can handle stress and restores balance afterward

The beneficial effect of pet therapy has different possible explanations. According to the affective–emotional mechanism hypothesis, a relaxing human–animal bond acts on adrenal and other corticosteroid hormones inducing a reduction of arterial pressure and cardiorespiratory rates. The psychological stimulation induced by the presence of an animal and its need for care induces persons to take care of themselves. The game system theory suggests that playing with an animal can increase defense and augment recovery potentialities; furthermore, an effective, emotional, psychological stimulation is known to solve important psychosomatic problems.[7]

Millennials Taking Over Pet Ownership:
- Millennials are most likely to take their cat to a groomer
- Millennials more likely to buy cat toys, including automated toys; and scratching posts (less likely to declaw?)
- Millennial Pet Owners are online more than any other demo.
- Millennials most likely use social media to learn about pets and communicate with others about pets (dogs about equal to cats)
- Millennials have the youngest pets of any demo
- Millennials (and Gen X) FAR more likely to seek alternative veterinary treatment

### MILLENNIAL: BENEFITS OF PET OWNERSHIP

#### Dog Owners
- 50% Brings family closer
- 52% Good for health
- 69% Companionship
- 71% Like a child

#### Cat Owners
- 35% Brings family closer together
- 38% Good for health
- 75% Companionship
- 55% Like a child
Conclusions for Millennials

- Pets are their babies
- They mandate **Quality**
- Care about the pets’ perception (after all they are their babies)
- If they can afford it – **WILL** Justify the cost
- Influencers to Others (sometimes instantly)

The survey also examined views and reactions by generation and found millennials were particularly interested in learning about the human-animal bond benefits from their veterinarian:

- 77% of millennials would have a more favorable view of their veterinarian if they discussed the health benefits of the human-animal bond with them.
- 74% of millennials would be more likely to visit their veterinarian if they discussed the health benefits of the human-animal bond with them.
- 25% of millennials always talk to their veterinarians about the health benefits of pet ownership, more than other generations.

Certainly by sharing a bed with a pet, dressing up pets for Halloween or anytime (just to look fashionable) and spending money on “dog clothes”

**Rescue mentality:**
Adoptions of dogs from rescues/shelters more than doubled, 15 percent to 34 percent comparing 2006 to 2016
Adoptions of cats from rescued and shelters nearly doubled, 22% to 37%. 2006 to 2016

**Dogs sleeping outdoors:**
11 percent 2006 to five percent 2016 – today millennials have no idea what a dog house even is
Dogs sleep in beds with people in 2006, 42 percent and in 2016 53 percent

**Cats indoors only 63 percent in 2006 and 2016, 71 percent**

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### MILLENNIAL DISADVANTAGES OF PET OWNERSHIP

<table>
<thead>
<tr>
<th>Dog Owners</th>
<th>Cat Owners</th>
</tr>
</thead>
<tbody>
<tr>
<td>27% Cost of food and medicine</td>
<td>36% Cost of food and medicine</td>
</tr>
<tr>
<td>34% Shedding</td>
<td>29% Shedding</td>
</tr>
<tr>
<td>18% Damage to furniture</td>
<td>36% Damage to furniture</td>
</tr>
<tr>
<td>33% Finding care when away</td>
<td>23% Finding care when away</td>
</tr>
<tr>
<td>50% Sadness when they die</td>
<td>45% Sadness when they die</td>
</tr>
<tr>
<td>21% No drawback</td>
<td>15% No drawback</td>
</tr>
</tbody>
</table>
Homeopathic remedies dogs
Gen X 10 percent
Millennials 16 percent
 Boomers 5 percent
 Builders 2 percent
This in itself isn't the issue quite as much as getting the information from online to pet stores.

Homeopathic remedies cats
Millennials 10 percent
Gen X 6 percent
Baby boomers 5 percent
Builders 5 percent

18 percent of millennials have pet insurance for dogs, compared to about eight percent for other generations, and 12 percent of millennials for cats compared to under 7 percent for other generations.

Millennials take cats to the groomers at triple the rate of other generations, 19 percent.
Groomers cats
19 percent millennials
11 percent gen X
Baby boomers 5 percent
Builders 6 percent

Millennials are more likely to take their dogs to training classes, 16 percent.

Dog day care up from 3 percent to 17 percent, 2006 to 2016

Gluten free and grain free dog foods are up 7 percent in 20012 to 19 percent in 2016. These are not millennials but baby boomers leading the charge

Millennials:
Natural dog food rise, 13 percent in 2006 to 22 percent 2016
Natural cat food rise 11 percent in 2006 to 18 percent in 2016.

Premium food – more brands – but sales actually flat for dogs/cats.
Raw dog food 2016 at three percent up from less than one percent 2012. People in all demos.
Raw cat food 2016 at four percent up from less than one percent in 2012. People in all demos.

For two-night trips or longer, 23 percent take dog(s) with in car 2016, 19 percent in 2006 – the greatest percent are boomers and these least are millennials, who prefer pet sitters, boarding, leaving pet with a friend/relative

Millennials prefer designer collars, leashes, dog beds, etc. Nearly all the 12 percent of cat owners with leashes/collars millennials.

Increasingly, pet information from Internet now 42 percent – led by millennials

Millennials lead in buying birthday gifts or Valentine’s Day for dogs and cats or a gift for the pet “being good” For Christmas all demos.
Average cost of gift for millennials is $22 for dogs, $24 for cats

Millennials are purchasing more flea and tick products for cats

Scratching post numbers went from 52 percent in 2006 to 70 percent in 2016.

National Retail Federation:
More than 30 million people will spend an estimated $480 million pet to costumes 2018, more than double the $220 million spent on pet Halloween costumes in 2010.

Ameritrade “Millennials and their Fur Babies”
A 2015 Gallup Poll found that a growing number of Americans—almost one-third — felt animals should have the same rights as people.

The average annual cost of raising a child in a two-parent $12,800 and $14,970, and much more if you live in a major city. That’s a quarter of a million dollars before each child reaches adulthood, which doesn’t include college or post-adulthood basement-dwelling

Baby Replacement Syndrome is real

Anti Vaxxers/pets:

“Adopting shelter dogs: Owner experiences of the first month post-adoption”

“Love Is All You Need: The Revolutionary Bond-Based Approach to Educating Your Dog,” by Jennifer Arnold

Association of Pet Obesity

Fat Gap:

523
FIVE FREEDOMS

1. Freedom from Hunger and Thirst by ready access to fresh water and diet to maintain health and vigor.

2. Freedom from Discomfort by providing an appropriate environment including shelter and a comfortable resting area.

3. Freedom from Pain, Injury or Disease by prevention or rapid diagnosis and treatment.

4. Freedom to Express Normal Behavior by providing sufficient space, proper facilities and company of the animal’s own kind.

5. Freedom from Fear and Distress by ensuring conditions and treatment which avoid mental suffering.


Benefits/Needs of enriched environments:
https://www.stevedalepetworld.com/blog/category/enrichment/
Can You Implement Self-Care Practices For Your Team/Clinic?

Standards Of Self-Care (as outlined by the Green Cross Academy of Traumatology)

As with the standards of practice in any field, the practitioner is required to abide by standards of self-care. These Guidelines are utilized by all members of the Green Cross. The purpose of the Guidelines is twofold: First, do no harm to yourself in the line of duty when helping/treating others. Second, attend to your physical, social, emotional, and spiritual needs as a way of ensuring high quality services to those who look to you for support as a human being.

Ethical Principles of Self Care in Practice

These principles declare that it is unethical not to attend to your self care as a practitioner because sufficient self care prevents harming those we serve.

1. Respect for the dignity and worth of self: A violation lowers your integrity and trust.  
2. Responsibility of self care: Ultimately it is your responsibility to take care of yourself and no situation or person can justify neglecting it.  
3. Self care and duty to perform: There must be a recognition that the duty to perform as a helper can not be fulfilled if there is not, at the same time, a duty to self care.

Standards of Humane Practice of Self Care

1. Universal right to wellness: Every helper, regardless of her or his role or employer, has a right to wellness associated with self care.

2. Physical rest and nourishment: Every helper deserves restful sleep and physical separation from work that sustains them in their work role.

3. Emotional Rest and nourishment: Every helper deserves emotional and spiritual renewal both in and outside the work context.

4. Sustenance Modulation: Every helper must utilize self restraint with regard to what and how much they consume (e.g., food, drink, drugs, stimulation) since it can compromise their competence as a helper.

Standards for Expecting Appreciation and Compensation

1. Seek, find, and remember appreciation from supervisors and clients: These and other activities increase worker satisfactions that sustain them emotionally and spiritually in their helping.

2. Make it known that you wish to be recognized for your service: Recognition also increases worker satisfactions that sustain them.

3. Select one more advocates: They are colleagues who know you as a person and as a helper and are committed to monitoring your efforts at self care.

Standards for Establishing and Maintaining Wellness

Section A. Commitment to self care

1. Make a formal, tangible commitment: Written, public, specific, and measurable promise of self care.

2. Set deadlines and goals: the self care plan should set deadlines and goals connected to specific activities of self care.
3. Generate strategies that work and follow them: Such a plan must be attainable and followed with great commitment and monitored by advocates of your self care.

Section B: Strategies for letting go of work

1. Make a formal, tangible commitment: Written, public, specific, and measurable promise of letting go of work in off hours and embracing rejuvenation activities that are fun, stimulating, inspiring, and generate joy of life.

2. Set deadlines and goals: The letting go of work plan should set deadlines and goals connected to specific activities of self care.

3. Generate strategies that work and follow them: Such a plan must be attainable and followed with great commitment and monitored by advocates of your self care.

Section C. Strategies for gaining a sense of self care achievement

1. Strategies for acquiring adequate rest and relaxation: The strategies are tailored to your own interest and abilities which result in rest and relaxation most of the time.

2. Strategies for practicing effective daily stress reductions method(s): The strategies are tailored to your own interest and abilities in effectively managing your stress during working hours and off-hours with the recognition that they will probably be different strategies.

Inventory of Self Care Practice -- Personal

Section A: Physical

1. Body work: Effectively monitoring all parts of your body for tension and utilizing techniques that reduce or eliminate such tensions.

2. Effective sleep induction and maintenance: An array of healthy methods that induce sleep and a return to sleep under a wide variety of circumstances including stimulation of noise, smells, and light.

3. Effective methods for assuring proper nutrition: Effectively monitoring all food and drink intake and lack of intake with the awareness of their implications for health and functioning.

Section B: Psychological

1. Effective behaviors and practices to sustain balance between work and play

2. Effective relaxation time and methods

3. Frequent contact with nature or other calming stimuli

4. Effective methods of creative expression


6. Effective skill and competence in meditation or spiritual practice that is calming

7. Effective methods of self assessment and self-awareness

Section C: Social/interpersonal

1. Social supports: At least five people, including at least two at work who will be highly supportive when called upon.

2. Getting help: Knowing when and how to secure help – both informal and professional – and the help will be delivered quickly and effectively.
3. Social activism: Being involved in addressing or preventing social injustice that results in a better world and a sense of satisfaction for trying to make it so.

Inventory of Self Care Practice – Professional

1. Balance between work and home: Devoting sufficient time and attention to both without compromising either
2. Boundaries/limit setting: Making a commitment and sticking to regarding
   a. Time boundaries/overworking
   b. Therapeutic/professional boundaries
   c. Personal boundaries
   d. Dealing with multiple roles (both social and professional)
   e. Realism in differentiating between things one can change and accepting the others
3. Getting support/help at Work through
   a. Peer support
   b. Supervision/consultation/therapy
   c. Role models/mentors
4. Generating Work Satisfaction: By noticing and remembering the joys and achievements of the work.

Plan Development

1. Review current self-care and prevention functioning
2. Select one goal from each category
3. Analyze the resources for and resistances to achieving goal
4. Discuss goal and implementation plan with support person
5. Activate plan
6. Evaluate plan weekly, monthly, yearly with support person
7. Notice and appreciate the changes

SMART Goals For Maintenance or Growth

S-M-A-R-T Specific – Measurable – Attainable – Realistic – Time-Based

Are there obstacles or resistances to achieving your goals? What are they? List here and share with your accountability buddy.

What strategies might you use to overcome the obstacles/resistances? List here and share with your accountability buddy.
Secondary Trauma- The Great Pretender: Understanding Your Neurobiology Before It’s Too Late

Learning Objectives

- Differentiate between Primary and Secondary Traumatic Stress and how it manifests in the veterinary profession.
- Articulate current theories for the etiology and transmission of traumatic stress and how it relates to compassion fatigue.
- Recognize traumatic stress triggers and the effects of the physiological cascade on the nervous system.
- Identify and utilize resources. Make plans for resiliency and prevention for self.
- Create and maintain a self-care plan for self and others outlined by the Academy of Traumatology’s Standards of Self Care.
- Have some FUN, and not take ourselves too seriously.

Traumatic stress runs rampant throughout our profession and we aren’t actively practicing with awareness of this. How can we solve this problem without understanding it? We largely focus on the symptoms: Compassion Fatigue and Burnout, even suicide, which are actually the end stage of traumatic stress.

1. Primary traumatic stress; happens directly to you. If you can’t cope you develop post traumatic stress disorder (PTSD).
2. Secondary traumatic stress; is observed by you but is happening to someone else. Also known as vicarious trauma. If you can’t cope you develop secondary traumatic stress disorder (STSD a.k.a. Compassion fatigue).

We can experience both primary and secondary trauma in our work as veterinary professionals. Most people can recognize and understand primary trauma. However, it’s secondary trauma that often goes unnoticed or unresolved.

Trauma produces actual physiological changes, including recalibration of the brain’s alarm system, an increase in stress hormone activity, and alterations in the system that filters relevant information from irrelevant. We now know that trauma compromises the brain area that communicates the physical, embodied feeling of being alive. Behaviors that are a result of trauma are not the result of moral failings, lack of willpower, or bad character- they are caused by actual changes in the brain. Advances in brain imaging technology have helped us understand the effects of trauma in a more scientific way. One of the discoveries around trauma demonstrated that not everybody reacts the same exact way to trauma.

Some Common Symptoms Of Trauma:

- Speechless Horror (alexithymia): Broca’s area (a speech center in the frontal lobe is effected).
- Shifting to one side of the brain (Right brain vs. left brain).
- Faulty brain alarm systems.
- Inappropriate stress response/inability to control the stress response.
- Loss/Lack of filter (sensory overload) that leads to…Numbing out, depersonalization (loss of sense of self), feeling disconnected.
- Disassociation and Reliving trauma/the event.
- Loss of safety/feeling safe.
This session will cross examine how situations that are psychologically traumatic arise in the veterinary profession, and how it manifests as trauma. The good news is there has been an increase in research around how trauma effects the brain. This increase of research and knowledge around trauma has also opened up new possibilities to palliate or even reverse the damage. We can now develop methods and experiences that utilize the brains own natural neuroplasticity to help survivors feel fully alive in the present moment and move on with their lives. Part 2 of the session will focus on different healing modalities, overcoming the negativity bias and neuroplasticity.
Techniques to start rewiring your brain and overcoming the negativity bias

We will watch some videos on neuroplasticity and the negativity bias, discuss some healing methods and also practice some of these methods during the lecture.

Healing from trauma: We can’t change the past (yet).

We CAN deal with the imprints of trauma on the body, mind and soul. Imprints such as: the crushing sensations in your chest that you may label as anxiety or depression; the fear of losing control; always being on alert for danger or rejection; the self-loathing; the nightmares and flashbacks; the fog that keeps you from staying on task and from engaging fully in what you’re doing; being unable to fully open your heart to another human being.

Self Leadership: Finding a way to become calm and focused

Learning to maintain that calm in response to images, thoughts, sounds or physical sensations that remind you of the past. In order to regain control over yourself, you need to revisit the trauma: sooner or later you need to confront what happened to you, but only after you feel safe and will not be re-traumatized by it. Example: OVH surgery

The first order of business is to find ways to cope with feeling overwhelmed by sensations and emotions associated with the past.

1. Top down: by talking, (re-)connecting with others, and allowing ourselves to understand what is going on with us, while processing the memories of trauma.

2. By taking medicines that shut down inappropriate alarm reactions, or by utilizing other technologies that change the way the brain organizes information.

3. Bottom up: by allowing the body to have experiences that deeply and viscerally contradict the helplessness, rage, or collapse that result from trauma.

Balance is Possible and Real

Resolution lies in restoring balance between rational and emotional brains. It’s important to feel in charge of how you respond.

When we are “Triggered” we our pushed outside of our “window of tolerance”. We become reactive and disorganized. We are either hyper aroused or shut down. Triggers vary for different people.

Even if you can manage to stay in control but are hyper aroused or shut down you become inflexible, stubborn and depressed. (AA calls this “white knuckle sobriety”).

Recovery from trauma involves the restoration of executive functioning, and with it, self-confidence and the capacity for playfulness and creativity.

Limbic System Therapy

The only way we can consciously access the emotional brain is through self awareness (“interoception”- latin, looking inside).

Deep breaths and Breathing calmly : activates the Parasympathetic nervous system via Vagus nerve stimulation. Puts the breaks on arousal.
Emotional regulation techniques: our education system is lacking. Mindfulness, Yoga, Meditation, Tai chi and qigong, even rhythmic drumming.

Traumatized people are often afraid of feeling. Even if the perpetrator is long gone, it’s now their own physical feelings that are the enemy. This may lead to compulsive eating/drinking, fear of making love, avoiding social activities, drugs/alcohol use to numb out.

**Choosing a Professional Therapist**

There is no one “treatment of choice” for trauma, any therapist who believes that his/her method is the ONLY answer to your problem should probably not be your therapist, or take it with a grain of salt. They should make you feel comfortable and safe.

Patients who have been brutalized by their caregivers as children often don’t feel safe with anyone.

Is there anybody who made you feel safe while growing up? A teacher, neighbor, shopkeeper, coach or minister even? That’s the seed to be planted to help you learn to re-engage.

If there is absolutely nobody, then often animals are the answer. (LIGHT BULB!) This may be a clue as to why many animal care workers have gravitated to animals. Or why “we don’t like humans.” or think “people suck”. Hmmmm…

**Healing Power of Touch**

The most natural way humans calm down our distress is by being touched, hugged, and rocked.

Most euthanasia appointments often involves at least a touch on the arm/shoulder, or hugs all around.

Bodywork; such as massage, kickboxing, self defense classes, karate, yoga, sensorimotor psychotherapy, and somatic experiencing.

When physical tension is released, the feelings can be released.

**Cognitive Behavioural Therapy (CBT)**

First developed for phobias such as fear of spider, airplanes or heights, to help patients compare their irrational fears with harmless realities.

Repeatedly exposed to stimulus without bad things actually happening gradually results in them becoming less upset ; bad memories will become associated with “corrective” information of being safe.

CBT also helps patients deal with their tendency to want to avoid, as in “I don’t want to talk about it.”

Remains controversial in it’s effectiveness, therefore not used as a sole therapy but as an adjunct.

**Increase Your Support Network:** The single most powerful protection against becoming traumatized.

When we are terrified, nothing calms us down like the reassuring voice or the firm embrace of someone we trust. Frightened adults respond to the same comforts of terrified children: gentle holding and rocking and the assurance that somebody bigger and stronger is taking care of things, so you can safely go to sleep.

Recovery from trauma involves reconnection with our fellow human beings.

This is why interpersonal trauma (trauma within relationships) is generally more difficult to treat than trauma resulting from traffic accidents or natural disasters. For instance: work place bullying, toxic team members, abusive work place conditions, sexual assault.
If people whom you would naturally turn to for care and protection terrify and reject you, you learn to shut down and ignore what you feel.

You have to find someone you can trust enough to accompany you, someone who can safely hold your feelings and help you listen to painful messages from your emotional brain. A guide, an anchor, a feeling of safety.

Workplace support plays a huge role here! This will be discussed further in part 3.
The future of the veterinary profession lies in the hands of the Millennial generation. Much has changed over the past few decades, and what worked to attract new associates in 1980 simply doesn’t anymore. Bringing in a new associate involves a different approach to the meaning of work/life balance, and compensation methods, vacation time, benefits and professional fulfillment have all evolved. Millennials now are the largest pet-owning generation comprising over 35% of pet owners nationally according to a study by Idexx. Your practice may want to bring in a Millennial associate to better serve this generation of pet owners, you may want to mentor a recent grad or you might find that your area is highly attractive for millennial veterinarians. If your practice is looking for a younger veterinarian to hire, these factors are important to consider when formulating a compensation package, and the same values hold true when it comes to retaining Millennial associates.

**Write a Great Job Listing**

The first step to hiring a new associate is typically a job listing. The AVMA website is the most frequented for young veterinarians looking for a job. Other online job listing sites such as Indeed.com are also popular. When formulating your job listing, write an introduction to the area you are in and what makes it a great place to live. Then, briefly introduce your practice’s history before diving into the culture you want to create. Make sure to mention the aspects of veterinary medicine that your practice does best (surgery, alternative/holistic medicine, palliative care, dairy cattle, equine orthopedics, dentistry, rescue/shelter work, etc.) and the opportunities that are available for an associate to practice and learn.

**Go High Tech**

Millennials grew up with the internet, so investing in a cloud-based software with paperless records will greatly improve not only efficiency of the practice, but also your chances of hiring a millennial associate. Bringing your practice up to speed tech-wise takes time and money, but will pay off in the long run with efficiency and happy employees.

**Be Flexible**

Once you have staffed your practice with associates to cover the operating hours, providing flexibility can make a great impact on retaining associates. For weekend hours, consider if one associate prefers to work most Saturdays or Sundays, or if a rotating schedule works better. Be flexible with weekend hours in general, and if you are not profitable on weekends or are opening a new practice, consider closing on weekends (yes, it can be done!). If your associates make production only, allow them to choose what days they work and how often as long as your basic coverage is met. Some bigger practices allow their associates to request which days they want to work the month before. For smaller practices, this is not feasible, so flexible scheduling to accommodate associates with children is important.
Work/life balance

Work/Life is all the rage these days, and there’s a reason for it: Burnout. Creating and promoting a culture of self-care and balance is important for attracting millennial associates, but you have to walk the walk to retain them. Provide opportunities for associates to chart their medical records at home so they can do it on their couch instead of in the office after hours (invest in cloud based software!). Encourage associates to take their lunch outside the office and use their annual leave. The work never ends (especially for us practice owners), so remember that associates need breaks too. Make sure that your associates are 100% free of hospital duties on their weekends and days off. They should not have to come in for hospitalized treatment care on their patient when they can transfer the patient to another associate who is working that weekend or day.

Provide Mentorship and Feedback

Mentorship is one of the most important benefits for young associates. Nearly every recent graduate wants to learn and grow, and the first few years are critical for setting the tone of practice. Providing strong mentorship in a structured way will help you not only attract millennials, but retain the ones you have. As they say in business, people don’t leave jobs, they leave bosses.

Providing feedback and constructive criticism can be uncomfortable, but is necessary to provide any associate with a plan to improve performance. In the first few months, meet weekly to address any questions and concerns about medicine standards as well as guidance on handling communication with clients and staff. Create an open door policy for your associates and staff to come talk about goings on in the practice and voice any concerns or questions. Sit down with new hires to co-create a roadmap of what the next year is going to look like in terms of professional goals, and revisit that annually. Ask them to come up with their own reviews and what they can do to hold themselves accountable to staying on track career-wise. We like to be involved with our careers!

Compensation

Compensation methods have changed over the last few decades, but business principles have not: your millennial associate (or ANY associate) must generate enough revenue to cover their compensation. Once you have satisfied that, discussing compensation methods (salary vs. Pro-Sal vs. production only) with potential candidates will give you a better idea of what kind of associate they are. There are no right or wrong ways to approach compensation for millennials. Some doctors do better on salary, some do better on production. Involving the candidate and asking about their take on which they prefer can help you determine more about their style of medicine. Great associates can be found in both camps, and tailoring a compensation plan to an individual with a plan to check in every 60-90 days is the best way to figure out how your millennial candidate is motivated by compensation.

Insurance

Standard benefits packages include health insurance. Offering other benefits such as dental, vision, pet insurance, disability and life insurance can be helpful in attracting new associates, but only if they really want those benefits. Benefits companies such as PeopleKeep allow you to offer a set monthly benefit amount to your employees while allowing them to choose how they want to use that benefit. Because the needs of employees can vary widely, this is a great option to provide benefits that associates can custom tailor to their personal preference.

Time off
Millennials are a generation of explorers. We value experiences over things, and most of those experiences are attended away from work. Travel and self-care are major focal points for many millennials which means that time off can be a very important benefit to many younger veterinarians. We all know that the rest of the developed world gives 4-6 weeks paid annual leave, so the average 2 weeks in the USA makes it difficult to travel, spend time with friends and family and recharge. If you have an associate that values time off, negotiate an additional week or two of vacation time (paid or unpaid) to factor into the overall compensation package. I had a friend who left a job she loved because, despite offering to forego her 10% raise, her boss refused to give her an additional week off unpaid to see her family abroad. Don’t lose a great associate because you don’t want to give them the time off they need. You can adjust salary and/or benefits to offset the additional days of annual leave.

**Continuing Education**

Providing adequate opportunity for CE is extremely important to recent graduates. Covering travel expenses, hotels and conference registration is standard. Discuss with your associate what preference they have for CE (AAHA, AAEP, AVMA, Fetch, IVECCs, State VMA, Oquendo Center etc.) to negotiate time off and the total amount of expenses paid.

While we all have minimum CE hour requirements, remember that some associates want to develop their professional skills outside of traditional CE conferences. Online courses in acupuncture and herbal medicine, disaster preparedness, Fear Free certification, public health, business management etc. can all provide CE credit and may be more interesting and beneficial to new associates than traditional general practice focused conferences.

**Meaningful work**

Millennials know that we want our work to have meaning. So many corporate practices have a hard time retaining millennials because their associates find little meaning and purpose without great leadership and mentoring. Promoting a culture of purpose (stellar client services, gold standard medical care, work with local rescues/shelters, opportunity for community outreach, etc) can help drive opportunities for your associates to find a sense of purpose in their work. Consider these three veterinarians who answered the question “what do you do everyday?”: The first says “I take care of sick animals.” The second says, “I’m a veterinarian” and the third says, “I help people provide the best quality of life for their pets.” The first has a job, the second has a career and the third has a purpose.

**Professional fulfillment**

Employees who find purpose in what they do typically stick around no matter what. Everyone’s purpose and interests are different, so do some in-depth discussions with young associates and potential new hires to gauge what drives them: mastering new skills, teaching, giving back to the community, research, accomplishments in writing, practice ownership, travel, political animal welfare, etc. Individuals are motivated and driven in different ways, so this will take some creativity and effort to help a recent graduate identify what gives them purpose in life. Encouraging your associate to pursue that purpose as a part of their career leads to professional fulfillment.
“If You Build it They Will Come” and 10 other inspiring quotes for starting a new practice
Eva Evans, DVM

Starting a practice from the ground up is scary. There are a million things to do and know, and the risk of failure can be paralyzing. Still, all private practices that exist were once startups, though the times and processes may have changed. When I was toying with the idea of starting my own practice, I reached out to mentors and practice owners I admired for help and guidance. My former boss and one of the greatest people I have ever had the pleasure of working with encouraged me with this simple quote: “If you build it, they will come. I promise.” He assured me that if I started a new practice, clients would come. The money would come. The bills would be paid. That’s a lot of trust to have in yourself, and I still had anxiety every day for two years during the process of starting my practice that I would fail. Now that my practice is going strong, I want to pass on the motivation and inspiration to other veterinarians who may want to start a practice but are plagued with doubts and fear. The following quotes are meant to inspire those veterinarians who are seeking some support and self-confidence in their journey to open their own practice.

1. “If you don't like the road you're walking, start paving another one.” - Dolly Parton
   Practice ownership provides the ultimate flexibility to create the career path you want. Many veterinarians are fulfilled in an associate role, but for those who want more, practice ownership may hold the key. Ownership gives you flexibility of time, who you work with, what procedures you want to do and the type of medicine you want to focus on. You can fire toxic employees, hire the best nurses, give pay raises and set your own values for your practice. It also gives you the ability to change and tweak things that don't work well within the practice, increase efficiency and integrate new processes or services. Ownership, if done correctly, can give you financial freedom to live the lifestyle you want. There is nothing easy about anything great, but you can change the course of your life by paving a new path into ownership.

2. “Don't be afraid to give up the good to go for the great.” - John D. Rockefeller
   Many veterinarians are happy with their current associate position but feel like they may want more. It is normal to worry about what you may be giving up anytime you make a major life change. When I moved from ER to relief, I was worried that I wouldn’t have enough work (I was wrong). When I went from relief to practice ownership, I worried that I wouldn’t be able to travel as much and have flexibility in my schedule (I was wrong). I worried that if I opened a new practice, I wouldn’t make as much money as I used to (wrong again). I also worried that I might have to sell my house, move in with my mom and drive for Uber at night if the practice wasn’t successful (fortunately, I was very wrong about this as well). The process of opening a new practice takes a lot of work and involves so many unknowns at the start, but don't be afraid to give up the good for the great.

3. “Make the most of yourself by fanning the tiny, inner sparks of possibility into flames of achievement.” - Golda Meir
   When thinking about buying or opening a practice, the whole task seems daunting. It seems like such a HUGE project, which is overwhelming and leads to us to believe we cannot do it. Remember to fan the sparks of possibility into flames of achievement. Becoming a veterinarian is a HUGE project that requires years of hard work and panning, and each of us was able to navigate that process by taking it one step at a time. If you are overwhelmed by the process, just take the first step and talk to a practice sales company or contact a veterinary specific lender (Bank of America, Wells Fargo and Live Oak all have industry specific lending specialists who can walk you through the process). Better yet, contact a practice owner who has accomplished what you want to do and ask them to go to lunch to share their story and wisdom with you.

4. “If You Build it, They Will Come” –Field of Dreams
The pet population is exploding, and people are spending more money on their pets than they ever have in years past. In 2017, veterinary care spending was over $17 BILLION in the U.S. alone according to the American Pet Products Association (APPA) https://www.americanpetproducts.org/press_industrytrends.asp . There are nearly 150 million pet dogs and cats in this country, and nearly 1 in every 3 people owns a pet according to the AVMA. The AVMA publishes a Pet Ownership Calculator to estimate the number of pets in your community. (https://www.avma.org/KB/Resources/Statistics/Pages/US-pet-ownership-calculator.aspx). The demand is there for keeping these pets healthy, and veterinarians are in short supply these days. The Bureau of Labor Statistics estimated that demand for veterinarians will increase by 18% over the next 10 years.

If you are still wondering if you can “make it” with a startup practice, rest assured that the pet population of the United States is a thriving industry and that demand is currently outpacing supply. Growing communities NEED growth in the veterinary services industry. As long as you select a growing market area that is not already overcrowded, if you built it, they will come.

5. **“It Takes Money to Make Money”**

We all know that adding an ultrasound, dental x-ray or a cold laser to a practice increases potential revenue stream and is often still profitable even when a loan is taken out for the purchase. So how can you make your own potential revenue stream better? By opening a practice! Unless you happen to be independently wealthy, you will need a loan for your practice, but you have the potential to make a much higher income with a financed practice than working as an associate. According to a veterinary-specific lending bank, the average loan amount for a new practice in 2018 was $468,000. This includes the lease, working capital, construction to remodel or build out the space, inventory, equipment, etc. but does NOT include purchase of the land if you decide to buy the real estate too.

Know that you NEED to invest money in order to make money, and a business is a tax free investment strategy. Don’t be afraid of investing your own money and using a bank loan. The default rate on veterinary practice loans over the last 5 years has been hovering around 0.25% (that’s only 1 in every 400 practices that can’t make their monthly loan payment!). Most banks require 0% down for a practice loan, and are giving rates on average of about 5.5% currently (or even better if you have great credit), and remember: they would not loan you money if they thought you couldn’t pay it back! Veterinary specific lenders are a great resource to consult with about practice needs, average revenue and expenditures, loan payback amounts, etc.

6. **“Success is the Intersection Between Preparation and Opportunity” – Seneca**

Successful new practices are started by people who have done their homework. Preparation is critical if you want your practice to take off. Google will be your best friend during this phase of preparation. So much of what you want to learn is available online at no cost to you.

First of all, think about what your VISION is going to be for your practice. How will it look on the inside? What’s the “vibe” going to be like? What culture do you want to build? What will be your mission statement?

Research what practice ownership is like. Sit down with practice owners in your area and have lunch with them. Ask them about their schedules, what they struggled with, what to expect after opening. Ask them to refer you to a good lender, architect, contractor, distributor rep, etc. Ask them about their equipment, their budget during the startup phase and what they wish they had known before they started. Find out if this is truly what you want to do!

Next, Research the geographical area you want to be in, your target market (Millennials vs. Gen X vs. Baby Boomers? Urban vs. Suburban vs. Rural? Gold standard vs. low cost? Traditional medicine vs. alternative?), the area’s growth potential (available from your local city planning department) as well as population size, socioeconomic status, average income and education levels.

Prepare your finances for a bank loan. Talk to lenders to learn what they are looking for and what you can do to get the best rate offer on your loan. You will need GOOD credit (670 or higher according to a major veterinary practice lender) so if your credit is not above 700, make a plan to get it there. Pay down bad debt (credit cards) and make on time payments for good debt (student loans, mortgage, car loan).
Remember: CASH IS KING. Having cash in a savings account shows the bank that you have thought ahead, are able to save more than you spend and that you have liquidity. Do not spend all your cash paying down your good debt faster. You are better off saving $10,000-$20,000 in cash so that when you are ready for your loan, the bank will see you can manage cash flow.

Once you have your loan pre-approval and an idea of what kind of practice you want to create along with the area you want to be in, start looking for opportunities for space. Decide if leasing or buying real estate is better for your individual situation (*buying real estate is often less expensive monthly than leasing, BUT may take a bigger down payment and a higher level of commitment to the long term than leasing). Find a well reviewed commercial real estate agent to do the leg work to find your space (they make their money on the seller, so they work for you for free). Interview and meet with architects who have experience with healthcare practice design. Architects will usually be able to give you a list of recommend contractors as well. Get your architect and contractors on board before you lock down your space to make sure your build-out or renovation will be within budget. You do not want to be chained to a space that will cost more than you can afford to build out! Look for an attorney who specializes in commercial property leases that can review your contract if you decide to lease. If you purchase, your real estate agent will handle the contract review. Once you have your bank loan and your space leased or purchased, you are ready to build out your space and get started on the fun part!

7. “It’s not the absence of fear, it’s overcoming it. Sometimes you’ve got to blast through and have faith.” - Emma Watson

Becoming a practice owner is scary. The idea of having so much responsibility can be terrifying. What if you can’t make your loan payments? What if you lose your house? What if you fail? Ahh, but what if you succeed? All practice owners have gone through immense periods of fear of failure. We all have moments that are trying and test our will and commitment. We all have moments of doubt and anxiety. These are normal, and if you don’t feel anxious or scared at the idea of opening a practice, then you should probably do more research on the amount of work a practice takes to get off the ground.

“It’s not the absence of fear, it’s overcoming it. Sometimes you’ve got to blast through and have faith.” - Emma Watson

8. “Jump Off That Cliff and Grow Your Wings on the Way Down” – Robyn Benincasa

Every person who opens a new practice needs to prepare thoroughly before committing to the journey. However, you can never be 100% prepared when it comes to startups. There will always be unforeseen issues that arise, and you have to remember to ride the waves as they come. Opening a new practice is a lot like having a new baby: it’s expensive, time consuming, terrifying and exhilarating all at the same time. Just like being an expectant parent, you can read all the books, research all the expert opinions, have a team of experts on hand to consult with, but you will never be fully prepared for what’s coming. This is totally normal! Once you’ve done your research, you have to jump off the cliff and grow your wings on the way down. Nothing but experience will teach you what you need to know. You CAN do it and you WILL learn as you go. Tens of thousands of veterinarians before you embarked on this same journey; always look to your mentors for guidance and inspiration.

“Jump Off That Cliff and Grow Your Wings on the Way Down” – Robyn Benincasa


Successful startups focus on doing things uncommonly well. It’s not enough to just open a practice and be mediocre, because new practices rely heavily on word of mouth and online reviews to draw in new clients. Think about what you do uncommonly well. Are you great with clients? Do you have excellent surgical skills or dental skills? Are you certified in acupuncture or alternative therapies? Do you want to focus on physical therapy and rehab? Do want to have an awesome doggie daycare or grooming facility in your practice? Are you going to promote a practice that works hard to be ultra convenient for busy pet owners? Focusing on customer service and client experience will grow your new practice by leaps and bounds in an era of increasingly corporatized medicine that lacks that personal touch and continuity of care. Look for a niche or an unfilled need in your area that you can do uncommonly well and watch your new practice grow and become successful.

“The Secret To Success is to do the Common Thing Uncommonly Well” – John D. Rockefeller, Jr.
10. “Effort Counts Twice” – Angela Duckworth

As the author of the #1 New York Times Best Seller *Grit: The Power of Passion and Perseverance* shows us, the ability to consistently work hard toward a long-term goal is the single biggest predictor of success. Many of us are already familiar with Malcolm Gladwell’s “10,000 hour rule” to become an expert at anything from violin to chess to investment fund management. Through several decades of research and hundreds of psychological studies, Duckworth shows us that talent and ability do not predict success in the classroom or in our careers. Perseverance, passion and grit are what we need to overcome setbacks, challenges and the daily, boring minutia of accomplishing great things. Duckworth writes that talent plus effort equal skill. Skill plus effort equal achievement. Thus, in any equation for achieving your dreams, effort counts twice.

This should bring all aspiring practice owners a bit of relief to know that talent is not what will take you the distance. Effort, which anyone can grow no matter the person, is the difference between achieving the dream and only making it half way before giving up. Practice startups are hard! There will be daily struggles, big headaches, unexpected expenses and curve balls throughout the process. Having the grit to keep putting in the effort and never stop moving forward, no matter how slowly, is the defining characteristic for success. When things get difficult, which they will, remember that the more effort you put into it, the better it will pay off in the long run. Don’t give up!

11. “We do not need magic to change the world, we carry all the power we need inside ourselves already: we have the power to imagine better.” - J.K. Rowling

The most beautiful thing about practice ownership is the magic that comes with forming and shaping our business. You can make your practice your own. You can make it better. You can make it awesome. Nothing great comes without hard work and dedication, but the rewards of that effort are the ability to truly influence those we work with: employees, young associates, patients and clients. Practice ownership is one of many ways to achieve purpose, and purpose is the foundation stone of fulfillment.
7 Steps to Attract and Retain Millennial Clients

Eva Evans, DVM

Millennials are the fastest growing and largest pet-owning demographic, and many of them are first time pet owners. Millennials make up 35% of the pet-owning population which makes this group of 20 to 39-year-olds some serious pet lovers. This generation is waiting longer to have children, so their pets become their “kids” which means they want the very best for their family member. In an Idexx survey, 80% of Millennials say their pet is a member of the family, and 57% say their pet makes them happier than anything else! Because many Millennials are first time pet owners, most do not have negative preconceived notions about veterinary care; they are easy to educate and, as a result, make great clients if proper steps are taken at their first few visits after adopting their new pet. These seven steps will help you attract and retain this valuable client base.

1. Make your practice space hospitable
   If your practice hasn’t had a facelift in 15 years, it’s time to start updating the environment. Your practice needs to be hospitable to the younger generation. Make sure your practice is clean, organized, orderly and free from smells. Incorporate modern conveniences such as client wi-fi access, a Keurig machine and low-volume, pleasant music in the lobby. Consider a new coat of paint and replace any wall hangings that are older than 5 years. Strategically place fake (or real) plants in the lobby and exam rooms to make the atmosphere more welcoming. Upgrade your waiting room chairs to something comfortable, and greet pets by their name as they come in. Remember, these pets are family members!

2. Get with the times on technology and convenience
   Technology is critical for progressing into the future. If your practice is not paperless, transitioning to cloud-based software is the easiest, cheapest way to become paperless and will increase your practice’s efficiency and value. Being able to email records, Rabies certificates, etc. saves time and decreases medical errors from lost or missing records. You will also save time and money using email or text reminders for upcoming vaccines while eliminating outdated mailer cards. Millennials want easy appointment reminders via email or text message. Online appointment booking, email appointment booking and updates via email are convenient for clients and free up time for your front office staff. Save doctors time by using email to check in on patients instead of calling, and if a client has a question about a lesion, you can have them email a photo or video to see if they need to make an appointment or not. Not only is it convenient, but you can often triage via email or text which is a valuable perceived asset to worried clients. Clients don’t want to rush into the vet if their pet isn’t really sick, and veterinarians don’t enjoy seeing a room for reverse sneezing or a new lump that turns out to be a rib, nipple or fat pad. Letting your client know via email that the photo of the “black stuff” on their dog’s skin is normal pigment and doesn’t need to be seen will help them gain trust in you as credible, ethical and caring professional. Clients want to know that we care about their time, money and experience in addition to their pet.

3. Gain trust by keeping pets in the exam room
   Taking pets to “the back” is a thing of the past. Invest in skilled nurses and Fear Free training to do a majority of your exam, diagnostics and treatment in the exam room. Pheromones, treats, kitty-burrito wraps and calming music in the room go a long way
toward making most visits a breeze. If the pet truly needs to be away from the owner, let
the owner see that you’ve tried to do everything in front of them first and then ask if they
think it would be easiest on the pet to perform treatments away from the owner. They
always want what’s best for their pet, and if they see you show concern for the pet’s
anxiety and comfort, they will feel more at ease being away from their pet. Experiencing a
calm, fear-free visit from start to finish will make a huge impact on retaining millennial
clients as well as building trust through competency and compassion.

4. Empower Millennials with Accurate Scientific Information

Millennials grew up with the internet and are well versed in online research. Your
Millennial clients will certainly use the internet to help them understand symptoms or a
new diagnosis. Get one step ahead and offer printouts or emailed links to online journal
articles, helpful websites and educational material from the AVMA, DVM360, American
Heartworm Society, etc. Millennials want to feel like they are involved with their pet’s
healthcare; providing good resources will not only get them on board from a compliance
standpoint, but will greatly minimize the risk that they will believe some of the bogus
information that may turn up in a Google search if they aren’t steered toward reputable
sources.

Encouraging your millennial clients to do their own online research (while stressing that
they can’t believe everything on the internet) promotes building trust in you as a doctor –
just make sure you are relaying accurate and up-to-date medical information.

www.DVM360.com has a wealth of information and helpful articles on all types of
medical, behavioral and nutritional issues. Use online resources to back up your depth of
knowledge and the most up to date treatment protocols; their confidence in you as a
competent doctor will soar.

5. Promote Pet Insurance, Wellness Plans and Health Savings Accounts

According to an Idexx survey, 61% of Millennials are willing to make financial
trade-offs to afford vet care for their pet. This means that subscription based health plans
such as pet insurance, wellness plans and pet health savings accounts are well received
by this demographic. Remember, because they view pets as family, they want to make
sure they are taken care of. Many Millennials working for large corporations are receiving
pet insurance as a benefit already, so be sure to ask all new clients if they have pet
insurance. DVM360 has a great handout comparing major providers here:

http://veterinarybusiness.dvm360.com/pet-insurance-comparison-chart . Talking about
pet insurance prompts clients to think about the future and reminds them that they should
be prepared for emergency vet care. Recommending pet insurance (especially on pure
bred puppies) is a great way to prepare clients for future health problems (atopy, hip
dysplasia, GDV, IVDD, etc.). Many Millennial clients purchase purebred puppies and
have no experience with the breed or their related health concerns. Encourage them to
sign up when their pet is young to avoid pre-existing condition exemptions! Be sure to
remind them that payment is due at time of service and most pet insurances pay a
reimbursement. This trend is changing though, and we predict that within 5 years, most
insurance companies will make payments directly to the veterinary provider.

Wellness plans and health savings accounts are another great way to help
Millennials budget and plan for their pet’s care. There are many providers of wellness
plans, or you can easily set up your own Pet Health Savings Accounts for your clients to contribute to monthly as a pre-payment option.

6. **Offer the Best Medicine and the Most Current Treatments**

Because Millennial clients want the best for their pets, they are more likely than older generations to seek out **affordable** newer diagnostics and treatments. READ: Millennials may want the best, but their generation is plagued with student debt load, graduating into the recession, fewer years in the workforce and lower inflation-adjusted income which means that many cannot afford things like MRI, TPLO, full chemotherapy or radiation treatments easily. However, diagnostics such as dental x-ray and SDMA tests are income accessible for most Millennials. Millennials are also looking for alternative treatments for pain such as acupuncture, cold laser, physical therapy and CBD oil. Many have known a relative who was in hospice care, and want access to end-of-life pain management for their beloved fur babies. Incorporating scalpel-free cryosurgery, nutraceutical anxiolytics and steroid-free allergy medications show that your practice is progressive and compassionate. Invest in affordable, progressive diagnostics and treatments to keep millennial clients coming back to your practice.

7. **Engage your clients by asking them to write reviews, then thank them**

Your millennial clients are online all the time. Why not ask them to write you a Google or Facebook review? Select clients who you know appreciate you and have had a good experience, then send them a personalized email with their name and their pets’ names asking them to write an online review. Provide links to your Google, Facebook and Yelp pages to make the process easy on them, and instruct them to write a single review, then copy and paste it into the linked websites. After we receive a great online review, we reward our clients with a surprise $5 gift card to our local neighborhood coffee shop along with a hand written note that says “Thanks a Latte!” and a few sentences about how much we appreciate them and how they are helping us grow. Millennials want to feel connected to the products and services they use, and asking someone for a small favor psychologically makes them like you more. Translation: Don’t be afraid to ask your clients to help you!

Lastly, never underestimate the power of a hand written card. For all new clients, we mail out a cute, logo-branded Thank You card (available from Vistaprint and many other online printers). We write a few sentences welcoming them to our practice family and thanking them for coming in. We also send logo-branded Christmas cards to our top clients with all the staff’s signatures and a few little tidbits of personalization about each family or pet. Hand written cards sent via snail mail (get the cute animal stamps from the post office!) are a magical way to brighten up your client’s day and bond them to your practice.
New practice startups are risky. Many professional consultants will tell you not to pursue this route, and to purchase an existing practice. What if there are no existing practices for sale? What if you can’t find one with the location, clientele, business model, etc. that fits you personally? What if you want to be creative and build your dream instead of buying someone else’s dream? Practice startups may be daunting to think about, but every practice in existence today was once a brand-new practice. These steps can help guide you through the process from start to finish to create your own vision and let you define your career by your own standards. While this session will focus more on small animal general practice, many of these considerations still apply to large animal, mobile practice and specialty hospitals.

Create Your Vision
All new practices must be envisioned. Starting a new practice without a vision of what it will look like, how it will function, the purposes it will serve, etc. is like driving your car blind. You won’t get very far before you crash. Focus on what you want your practice to be. What city? What neighborhood? Urban, Suburban or Rural? Large, Small or Mixed Animal? How many associates do you want to have one day? What type of clientele will you serve? What will your practice focus on (excellent customer service, physical therapy and rehabilitation, alternative therapies, high volume/low cost, specialty services). What will your practice look and feel like? Ultra modern, cozy, historically preserved, colonial, strip mall or farm house style? What types of services will you offer? Boarding, grooming, underwater treadmill, CT, ICU/ER, comfort rooms, mixed animal hospitalization, separate waiting areas for cats and dogs? What will your hours be? Will you be open on Saturdays? Will you see emergencies? Once you have a vision of what you want your hospital to be, then the work starts.

Get a Mentor
This process is long and difficult. Find someone who has already made this journey with a successful startup practice similar to what you want yours to be like and ask them to lunch. Reach out and find mentors in the same city or across the country, to help guide you along the way. Despite tales of greedy, conniving veterinarians, most practice owners are happy to share their wisdom about what how they built their practice. If you know of other business owners (not necessarily veterinarians) in your area, pick their brains about good references for professionals such as attorneys, architects and accountants as well as contractors and construction crews, sign companies, etc.

Do Your Homework
Starting a new practice will set you back anywhere from $100,000 for a small mobile vet to $1 million + for a general practice small animal clinic. The average bank loan for a small animal startup practice is around $487,000 (practice only - not including real estate). It’s a lot of money to borrow if you haven’t done your homework to know what all is required to get the job done. Read books on business (Small Business for Dummies for example), use the internet to find articles about practice ownership, how to get a business license, tax requirements, etc. Spend enough time doing your research so that you have a decent grasp on what all is needed legally for your state and county as well as federally for the IRS. Take time to learn how much lease costs and buildout costs are in your location as well as time frames for architects, building permits and contractors to build out your space. These will all vary on your area as well as the current state of the economy.

Once you are ready to start, file your paperwork with the IRS and your state to get a business license, FEIN and to register as an LLC or Corporation (consult with a professional to determine which structure is better for your state and your personal financial situation). LLCs are the most common, but do your research and figure out which will work best for you.

Enlist Professional Help
Enlist a group of professionals to help you start your practice. For most, this will include a bank lender representative, commercial realtor, insurance brokers (AVMA is a great resource for this), architect, contractors, distributor reps, pharmaceutical reps, equipment reps, attorneys, and accountants. These people make their living helping others build businesses, so reach out early to find good contacts who can help you navigate through the process. Other business professionals are knowledgeable about their own
industries to a level of detail we veterinarians will never be able to know, and simple economics makes it possible for veterinarians to utilize these professionals to help us move quickly through the process of practice startup. For example, it is not worth your time to find your own real estate to lease or purchase, research fair market value and negotiate terms – let a commercial realtor due the leg work for you. You don’t need to know how to lay tile or hang drywall – hire it out to a professional contractor.

**Secure Financing**
Financing is a major part of any practice purchase or startup. **Do not sign any legally binding agreements such as leases, real estate purchases, equipment purchases, etc. without securing your financing first.** You may have found the perfect location but signing a lease before you know if you will even be approved for the practice loan is a great way to go bankrupt.

Contact veterinary specific lenders first to get an idea of what is needed for pre-approval. Local banks, credit unions and larger lenders may also be able to provide funding with a SBA loan, though this is a much more time consuming process and carries additional fees and a higher rate. Once you have found the best offer in regards to rate and terms, you can move forward with opening the loan. At this point, you may start drawing out of the loan to pay for your lease, equipment, professional fees (architect, contractors, attorneys, etc.). Be aware that the money drawn out of your loan prior to opening is subject to a higher APR than your loan will be once the loan is closed, so try to plan large purchases as close to the opening date as possible (i.e. do not purchase a $50,000 x-ray 12 months before you plan to open – you will owe a lot of interest on that over those 12 months which will roll into your final amount owed!).

**Find and Lock Down Your Location**
Using a commercial realtor, figure out what area you want to be in and what type of property you want. Do you want to lease versus buy? Buying is a larger long-term commitment up front, but can actually be less expensive monthly than leasing depending on your area. Price out real estate purchases vs. lease options to see what works best for your situation. Be advised that all leases have a built in annual increase in monthly rent (usually 3-5%), so your starting monthly lease amount will go up every year. Do not sign a lease for less than 5 years if you plan to remodel and build out a space. When your lease is up, you may not have the option to re-sign and you may be forced to find a new location for your future busy practice. Your realtor can walk you through the details, pros and cons of each. Be sure to budget for your rent or mortgage. A well managed practice should spend 8-10% of gross revenue on lease or mortgage.

Once you’ve found a realtor, consider how much space you will need based on your budget, type of practice, services offered and how many doctors you eventually want to accommodate. Avoid buildings with second floors as much of this space is unusable due to the Americans with Disabilities Act unless you have an elevator. How many parking spaces will you need? Is the property already zoned for commercial use? How easy will renovations be to turn the building into useable veterinary space? Do you need a special layout or space for certain equipment such as a CT or isolation room? Is boarding allowed in the zoned area? What will traffic and noise be like? Will your clients be able to easily find your building from the main road? Ask a friend to examine your potential space from a client’s perspective. If you are building a brand new building on an empty lot, assess the soil and drainage, flood risk and property easements that may become future issues.

Engage an architect in the process BEFORE you sign a lease or purchase agreement. They can give you an idea of how much build out will cost, bid out the work and also point out potential problem areas that may prevent you from using the space as you wish such as a load bearing wall that cannot be removed.

**Construction Buildout**
Your architect is instrumental in helping you find good contractors. The architect will also oversee much of the project to make sure the contractors are building to code and the blue prints are being followed. There is no hard, fast rule on how to decide on a contractor simply based off bid amounts. Ask for references from other projects and call those business owners to get an idea of how the construction company performed previously. Your contractors can be your best friends or your worst nightmare. Select a company with a long history of a great reputation. Be prepared for mistakes to happen in the buildout phase (wrong paint color, light switches on the wrong side of the door, etc.) and visit the site frequently to catch these problems early. Be prepared for building inspections and permits to take longer than expected as well. A good rule of thumb for timeline is taking the time you think it will take and multiplying...
that by 3. You may think your buildout should only take 8 weeks, but chances are it will take closer to 6 months.

**Purchase Equipment**
Each major distributor that services your area will have a representative available to call on your clinic. Major distributors include Covetrus (formerly Henry Schein), Patterson and MWI. Other smaller distributors may also service your area. Your distributor rep should be your ultimate ally in the otherwise confusing and overwhelming task of ordering equipment and inventory. Choose which company to use based on the rep’s effort to gain your business. A good distributor rep will set up meetings with equipment sales rep, pharmaceutical sales reps, provide you with samples of products to try, answer all your questions about which equipment to purchase for your individual needs and should be able to give you a good idea of the total cost for all the equipment you want in your practice. They make their money not only on your initial purchase order, but on all ongoing inventory and equipment purchases in the future, so they are incentivized to build long lasting, trustworthy relationships with practice owners.

Once you have settled on a distribution company with a rep you have a good relationship with, start thinking about what pieces of equipment are “must haves” and what you would like if there is extra room in your budget. Check with your state board to find out what is required by law for your type of practice. Your lender can help you decide how much to spend on equipment vs. inventory etc. Some equipment takes 6+ weeks to arrive so be sure to have it picked out ahead of time in order to place the order at the correct time. Do not order equipment (especially legally binding agreements) before you have a locked down a place to put it all! You do not want your $6,000 autoclave showing up while your building is under construction and suddenly disappearing. You may also find that the extra long surgery table you ordered won’t actually fit in your new surgical suite.

Deciding on a veterinary practice management software is an important decision as well. There is no reason for a startup practice to use paper records. Technology is moving forward and cloud based software is the future. Investing in server based software that will have to be replaced in 5-7 years is not a good investment given how great cloud based software providers are now.

**Order Inventory**
Contact your distributor rep and your Pharma reps a few weeks before you need to order your inventory. They can walk you through what products are available. You need a pharmacy, but you do NOT need to stock everything! You can always order products that are not often used as needed for clients. Avoid expensive products with quick expiration dates such as Convenia and Proheart until you are busy enough to use the entire bottle before it expires. Carry only one or two options for heartworm and flea/tick prevention. Stacking a “lean” pharmacy can easily cost you $30,000 and you don’t want those meds and vaccines expiring on your shelf! Most companies offer a buy 1 get 1 offer on the initial stocking order for new hospitals. This is great, but don’t be tempted to over-purchase just to get more free product. If it expires before you can use it, that’s money in the trash. Also, don’t forget you are paying loan interest on every pill you purchase with your practice loan. Money spent on your pharmacy cannot be used for other things such as payroll, lease payments and other carrying costs until someone buys that product from your shelf.

**Hire Staff**
This session is not meant to dive into the HR aspects of practice ownership. However, keep in mind that because timelines for opening are rarely ever met, do not start your hiring process until you know exactly when you will open (usually about 3 weeks out from opening). Chances are with a brand new practice you will have very few clients at first. Don’t overstaff at the beginning or you will quickly find yourself unable to meet payroll.

**Finishing Touches**
You’ve done all the hard work, and now it’s time for the finishing touches! Spend time thinking about what furniture and décor you want for your new office. Landscape if needed. Order business cards, get your website, Facebook Business Page, Google Business Page, Yelp! Business Page, Twitter and Instagram accounts. Order scrubs or other uniforms, stock and organize your pharmacy, exam rooms, reception desk etc. Secure your agreements for outside services such as trash, lawn care, cremation companies, teleradiology consults, etc. Have your sign installed. Find a payroll company (many easy and low cost
options are available) to process your direct deposits and handle employee FICA taxes. Make sure your insurance (general liability, disability, PLIT, etc.) is adequate and current. Call your state board to schedule your inspection for a facility permit and fill out all necessary paperwork. Finishing touches are fun, but will take more time than you expect given the nature of all the tiniest details that we don’t plan for.

Open For Business!
Today is the big day! Spread the word in your neighborhood via online social media marketing that you are open for business! Consider having a “Grand Opening Party” about 2 weeks after your original opening to give you time to work out the inevitable kinks that come with any new business. The work doesn’t stop here, but more of the fun starts. Creating your own practice as a startup is challenging but rewarding in the most personal of ways. Difficulties in life and business always arise, but the amount of time and effort spent getting to this point makes the victory of your grand opening that much sweeter. Relish in the moment, and remember: now the hard work of building your client base starts!
Why you should sell to a millennial associate, why millennials should buy and how to get it done

Eva Evans, DVM

Transitioning into retirement for practice owners can be daunting when it comes to selling your practice. Many practice owners don’t have an exit strategy, so corporate buy out is the fastest way to retire for many and often includes a large cash offer up front free from any funding stipulations. Long term benefits to clients, existing staff, associates and the community are lost with many corporate buy outs as “practice flipping” becomes more and more prominent throughout the country. The following sections detail the benefits to selling to a millennial, why millennials should buy and the process to finalize the transaction.

**Why Sell to a Millennial?**

Every practice owner needs to sell eventually, so why choose a millennial over corporate? Practice owners who transition their practice to the next generation of veterinary ownership can reap great fulfillment personally and professionally. While changes will happen with any ownership transfer, they can be progressive and positive with careful planning and vision on the owner’s part. With the heavy debt load on today’s young veterinarians, ownership is a great way to build long term wealth. If protecting your staff and associates, helping the next generation of veterinarians, continuing your practice’s good will and protecting the legacy you built are important aspects of your career fulfillment, consider making a 2-5 year plan for finding the right young veterinarian to buy your practice.

**Find an Interested Associate and Mentor**

Not all veterinarians are cut out to be owners. Focus on associates or potential buys who have the curiosity and drive to know more about ownership. If you have associates who may be interested, focus on long term training and exposure to what practice ownership looks like. Don't hide your financials from your associate. Many young veterinarians are averse to owning because they don’t have the business training from school. Develop young associates by encouraging them to ask questions and take part in the aspects of ownership such as hiring, participating in staff annual reviews, learning about employment law, understanding how depreciation and estimated tax payments work, etc. You can develop a formal training program for a mentee who is interested in learning a lot, or you can make these casual learning experiences as they come up in day to day practice. Practice getting comfortable sharing your wealth of ownership and management knowledge and wisdom.

**Get a Practice Manager**

If you don’t already have a phenomenal practice manager, get one. Your practice manager can help young associates with questions and guidance as well as support and confidence that they can take on the ownership role without feeling overwhelmed. Practice managers who know the business side can serve as valuable mentors to potential new owners, and if you plan to sell to a non-associate, having a practice manager will increase the value of your practice by providing a smoother transition to the new owner.

**Market Your Practice Toward Millennial Buyers**

If you don’t have a current associate who may want to buy your practice, market your practice toward millennial buyers. Minor cosmetic updates such as paint, furniture and décor will make your practice more inviting to younger veterinarians who can “visualize” owning the place and taking pride in it. When advertising, include your desire to mentor the new owner and help with the transition after purchase. Get creative with the affordability including owner financing and partnership buy in opportunities. If the idea of helping the next generation of veterinarians succeed and build wealth is important to you, brainstorm ideas of how to help them overcome the challenges of the process. Starting early and having a flexible plan is key to making this a successful business transaction.

**Think Long-term**
The onus of transitioning a practice’s ownership does not fall solely on the current owner. Millennial veterinarians need to think long term about the future of their career over the next 35-45 years as well as the future of our great profession. Where do you want to be in 10 or 20 years? How much do you want to be making by then? What are you doing to fund your retirement NOW? Know that humans are extremely capable of growing, evolving and learning to fill the ever-changing roles of our lives. I liken practice ownership to having a baby: it’s expensive, challenging, frustrating, tiring but oh so rewarding. If practice ownership excites you in the tiniest way, explore what that looks like and know that there are people, both professionals and mentors, to help along the way.

Visualize the Benefits
Though practice ownership comes with its own share of headaches, it comes with many more freedoms and benefits. In addition to the obvious benefit of making more money, as practice owner you have much more control over your working environment and schedule. Though it requires a lot of hard work, owners enjoy the flexibility to change the schedule and take more time off than an associate. As long as you have trained your staff properly, your clinic can and will function without you for two weeks! Relief coverage and associates allow owners the time off to be able to take vacations, pursue hobbies and activities outside of their career and also give back to the community. This doesn’t insulate you from the occasional staffing crisis that may arise during your time away, but developing your support staff and empowering them to problem solve and make sound decisions in your absence can provide you with much more time off than you would ever have as an associate.

Another perk as owner is deciding who you work with. You have complete control over what personality types fill your practice. You also have the (often difficult) privilege of being able to terminate employees who underperform or undermine your practice’s values and ethics. You also have the ability to train your staff to perform medical and client services in the ways that align with your values. You get to practice medicine at the level you want! You also hold the final decision on new equipment purchases, protocols and processes to implement in your practice.

Though retirement is rarely on the minds of young workers, it is important to make a long term plan for savings and investments. While your practice is an investment itself and will likely be worth a lot of money when you sell it down the road, don’t forget the benefits of business ownership when it comes to retirement accounts. IRAs cap annual contributions at $5,500 ($6,500 if you are 50 or older) and those filing single with over $122,000 in taxable income are phased out from contributing completely at $137,000 ($193,000 to $203,000 if filing married). Even those contributing the full $5,500 annually, starting at age 26 and retiring at 67 would save just over $1,000,000 by 2060 assuming a 6% return rate. Unfortunately, $1,000,000 in the year 2060 will be about $363,000 in today’s dollars (assuming 2.5% annual inflation). As a practice owner, you have access to not only tax-free investment into your practice, but also retirement savings plans. These include SIMPLE IRAs, SEP IRAs and Self-Employed 401(k).

Let’s Talk Taxes
Taxes – the most dreaded word in the English language? Taxes can be daunting, but as I like to say, “For every tax action, there is an equal (or even better?) and opposite tax reaction.” The money you pay for your practice, your staff, your equipment, your repairs, and even your business taxes are all tax deductible. Every dollar you invest in your practice is tax-free. At the current 2019 Federal Income Tax brackets, those making more than $84,200 unmarried or $168,400 married will save 24% of every dollar spent on their practice in the form of income tax reduction (and the savings get better the more money you make). Purchasing a $500,000 practice will save you $100,000 or more over several years in income taxes through depreciation.

With the 2018 tax law revisions, veterinary practice owners are also allowed to deduct 20% of their business income (IRS: “Section 199A: Deduction for Qualified Business Income” [https://www.irs.gov/newsroom/irs-issues-proposed-regulations-on-new-20-percent-deduction-for-passthrough-businesses] ) as long as your taxable income does not exceed $315,000 filing married or $157,500 filing single. If you will go over these limits, invest in new equipment or updates to your practice to reduce your taxable income. Consider that every dollar spent on your practice equals $0.24-$0.37 you don’t have to pay the IRS.
CLOSING THE DEAL

Practice Valuation
Owners should have a valuation done and consult with a professional to figure out what their practice is worth. Involving an associate who is interested in buying the practice is a great way to introduce them to the "behind the scenes" aspect of ownership. Once a fair market value has been established, owners can work with associates or non-associate potential buyers to formulate a plan for transition.

Financing
Bank lending is the most common way to finance a practice purchase. Banks such as Bank of America, Live Oak Bank and Wells Fargo have veterinary specific lending groups that can walk potential buyers through the process. Important things to plan for if ownership is in your future include a good credit history and score (MINIMUM 680, but average veterinary borrowers have credit scores around 730) and a savings account (there is no set minimum, but $10,000-$20,000 is a good starting point). Savings in cash or other liquid assets will show the bank you are capable of planning ahead and saving for unexpected emergencies. Banks want to see a solid history of on-time payments to creditors, wise use of credit, low amounts of "bad debt" (credit card debt) and a solid credit score. Student loans are considered "good debt" and veterinary-specific lenders say that student loan debt rarely prevents them from lending for a practice purchase assuming that the borrower has a good credit history and has demonstrated the ability to practice smart financial decision making. In addition, your production numbers as an associate can help you score a bigger loan (especially if you have other debt besides student loans) because it shows you are capable of bringing in an income for the practice if something were to happen to any associates and you were the only veterinarian on staff. Contact a veterinary specific lender or a lending group to discuss what type of loan you can get, what down payment is needed (if any), rate, terms, etc.

In the event that a traditional lender is unwilling to provide financing, some practice owners may be willing to serve as the “bank” to finance the deal. The buyer pays the seller directly every month plus interest based on the agreed upon terms. If the buyer defaults on the loan, the practice would then belong to the seller again and not a bank. This is a great option for practice owners who don’t need the full cash payout up front, and thus can enjoy the benefits of retirement with a monthly check almost like an annuity long term. Plus, a seller who is willing to act as lender in the sale of their own practice will collect a large amount of compounded interest over time. For example, a seller who sells her practice for $500,000 at 5.5% APR with a 20 year term will collect not only the $500,000 but an ADDITIONAL $325,464.09 in interest! Plus, if the buyer defaults, the seller regains the asset (the practice) and can resell the asset. It should be noted that current default rates on veterinary practices is currently <0.25%, so the risk for default is extremely low.

Another option for sellers wanting to retain some ownership is a partnership buy-in. The terms of partnerships can be anything you want them to be, so be sure both parties consult with professionals and attorneys and spend several months at minimum exploring what that looks like for both parties. Graduated buy-in structure (purchasing a certain percentage at a predetermined interval of time) is also an option, especially for young veterinarians who have a large student loan debt or want to experience ownership at a slower rate with less up-front exposure to risk.

The Transition
Because the entire purpose of selling to a younger veterinarian is to continue the legacy of your hospital as well as protect the community, your clients, staff and associates, a transition period should be expected with any sale. Spending 6 months to a year helping your buyer transition after purchase will ease the strain on the staff and clients as well as the new owner. This transition period is common and most sellers are willing to stay on to help smooth out the changes. Staying too long can cause a rift if the new owner wants to change protocols, standard of care, etc. that the seller doesn’t agree with, so avoid adding into your terms of sale that the seller will agree to stay on for more than 1 year. You don’t want to find yourself (as the seller or buyer) unhappy that things are changing (or not changing) in a way you wouldn’t agree with. As the seller it is important to remember that this beautiful practice that you built with your blood sweat and tears is an ever-evolving business and it is ok that the new owner may do things differently.
Practice ownership is not for everyone, but proper mentorship, encouragement and professional development of interested associates can help the profession grow while keeping wealth accumulation in the hands of our fellow veterinarians and not in the pockets of corporate shareholders. Young veterinarians must also make an effort to learn about ownership, business skills and make it known to potential sellers of their interest to pursue ownership.
BUDGETING DOESN'T HAVE TO BE BORING OR HARD

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INTRODUCTION

Everyone talks about the importance of budgeting but actually setting one up can seem like a daunting task. However, doing so is not as hard as it sounds and is well worth the effort. A budget is essential to:

- Improving the medical and surgical services offered by a practice
- Increasing revenue
- Setting fees
- Analyzing expenses
- Monitoring cash flows
- Specifying operational changes

A budget, done well, forces a practice to plan and planning makes both medical/surgical and financial success possible.

WHAT ARE THE STEPS NECESSARY TO CREATING A USEFUL BUDGET?

Gather the basic information

Start the budgeting process for the next year three to four months before the end of the current year. The first thing needed is the profit and loss statements (P&L) or tax returns from the past two full years as well as a year-to-date statement from the current year. If the practice accountant makes a lot of changes to the P&L statement generated by the practice before filing the tax return, the tax return data may be more accurate than the P&L and it would be best to use the tax return data for the years it is available.

Input historical data into a spreadsheet program

While budgets can be done on paper, they are much easier to do using a basic spreadsheet program such as Excel. An electronic spreadsheet program allows practice owners or managers to do “what if” scenarios easily. What happens to the cash flow if the practice buys a new ultrasound? What happens to revenue if the practice buys a new ultrasound? What happens to cash flow if the practice gives the staff a raise?

It is only necessary to know the basics of the spreadsheet program—how to input data, how to sum data and how to do simple arithmetic formulas. Don’t worry about having to learn macros, complicated financial functions or other sophisticated techniques—they won’t be needed for this. If no one in the practice knows how to use a spreadsheet program, there are multiple courses available on the internet, at local community colleges or from other sources that will teach the basics.

Input the data from the last two year’s financial statements into a spreadsheet program as well as the year-to-date information from the current year. Each year should have a column for the dollar amounts and the dollars calculated as a percentage of gross income. The partial year data should be annualized using the practice’s best estimate of what will happen during the rest of the year. The revenue and expense categories shown in the spreadsheet are listed down the side and the dollar amounts and percentage of gross revenues for each year across the top. The net income or taxable income line (titles will vary depending on the source reports used) should be a formula subtracting all the expenses from the revenue. The net income calculated after all the revenue and expenses are entered should equal the net income or taxable income shown on the P&L statement or tax return—if not, one of the numbers have been entered wrong and it will be necessary to go back and check the input.
It is important to include the percentage data because some expenses fluctuate with revenue instead of being a flat amount each year. For example, if a practice sees more patients and has more revenue, the laboratory costs will be higher than if it saw fewer patients. These kinds of cost are called variable costs because they vary with revenue. Other costs such as rent are called fixed costs because they are the same no matter how many patients the practice sees. The percentage column also allows for analysis of the consistency of revenues and expenses even when the dollar amounts are changing—changes in the percentage amount may indicate areas in which the practice is doing well or in which it needs to improve.

The calculation for the percentage category is as follows:

\[
\frac{\text{Revenue or expense line item}}{\text{Total gross revenues}}
\]

As noted above, the revenue shown on the income statement will usually consist of just one line called medical revenue or fee income or something similar. Use the revenue category report to break out this amount into the different areas of practice income. There will usually be a difference (either positive or negative) between the total on the revenue by category report and the financial statements—if this difference represents more than 5% of the total revenue, the practice’s financial advisor should be asked to help reconcile the differences. Otherwise simply put it in a line item called “unidentified revenue” or something similar.

Add a column to the right of the historical data and title it next year’s budget. Also add a column for the percentage calculations.

The data just entered generally results in practice profit or taxable income; however, the most useful budget for most individuals is one that shows cash flow. In order to convert profits to cash flow, make adjustments for principle payments and depreciation at the bottom of the spreadsheet. The depreciation added back to the net income is the same amount shown as an expense above this line and the principle payments should include the principle amount paid on all loans and capital leases during the year. The practice’s financial advisor may need to help obtain these and other non-cash numbers.

Identify the financial changes expected in the practice next year

For example, will the rent increase? Have a tech just been hired whose salary isn’t included in the prior year financial statements? How much of a raise is planned for the staff and associates? These items will need to be included in the budget. Make a list of all the expenses that will definitely increase next year and the expected dollar change.

Identify the changes the practice would LIKE to see in the practice next year

Think about the practice and owner goals, both personal and professional, for the next year. This shouldn’t be limited to goals thought of as purely financial. Everyone has goals. They may be as specific as “I want to take home $10,000 more next year.” or “I want to purchase an ultrasound during the next year.” Or they may be a little fuzzier, such as “I sure wish I made more money” or “How can the practice down the street afford an ultrasound and I can’t?” Everyone has heard the clichés—“If you don’t know where you’re going, how will you know when you get there?” or “If you can’t measure it, you can’t manage it.” The reason these phrases are clichés is because they are true! Sit down and think about what the practice wants to accomplish next year and put it on paper. Some common goals to help start the thought process include:

- Purchase of new equipment—ultrasound, new x-ray machine, laser surgery unit, etc
- Attend a particular CE conference
- Hire more staff
- Hire a new veterinarian
- Give the staff a raise and more benefits
• Provide more training for the staff
• Take an exotic vacation
• Send your kids to private school
• Spend less time at work and more time at home
• Renovate your building
• Build a new building

At first glance, some of these may not seem to have anything to do with budgeting, but they all can be achieved with careful financial planning. Some are obvious; if the practice is going to buy a new ultrasound, it’s going to cost $35,000. Where is the financial tie-in to wanting to spend more time at home and less at work? Think through what it will take. A relief doctor? A part-time associate? More support staff? Estimate the financial aspect of each goal.

Most of the goals people have involve spending money from the practice—whether the goal is to purchase equipment or for an owner to have more personal earnings. To achieve these new goals, the practice needs to make more money, spend less money or change the areas in which it is spending money. Generally a practice will do more to achieve its goals by focusing on revenue increases than on expense cuts. Expenses must be reasonable for the practice, but at some point cutting expenses will hurt the business. For example, cutting support staff below a certain level will harm client service. While focusing on revenues is generally more productive, it is also harder for most veterinarians, because it means focusing on areas they have less comfort in changing such as client service, marketing or staff training. Focusing on expenses comes more naturally—it’s easy to call around and price shop long distance plans or the cost of health insurance. This will be discussed further in the next section.

Determine the budget for the next year.

First of all, estimate next year’s revenue by multiplying the current year’s revenue by any planned fee increase. For example, if the planned fee increase is 5%, the amount in the revenue cell of the budget column would be last year’s revenue multiplied by 1.05.

Next, look at each expense item and determine if it will be the same as last year or does it need to be changed? Most of the “Cost of medical services expenses” fluctuate with revenue so it is necessary to estimate them using a formula in the appropriate cell of the budget column—the percentage of revenue in the prior year multiplied by the estimated current year’s revenue. Fixed expenses are generally entered as dollar amounts. At this point, enter all the expected expense changes identified above. For example, if rent is expected to increase by $3000, the rent in the budget column should be the dollar value of last year’s rent plus $3000. If the practice plans to raise staff salaries by 5%, then the amount in the budget column staff salary cell should be last year’s staff salaries multiplied by 1.05.

After inputting the expected changes in expenses, next enter the costs of the goal items identified. After inputting the expense side, analyze what can be done to increase revenue to result in the cash flow desired by the practice. Will revenue increase more next year? How can the amount be determined? Looking at history is certainly helpful—if the practice revenue has grown at a steady 5% for the last 10 years (not counting the fee increase factored in above), it is reasonable to assume it will do so again, assuming there are no significant practice or client changes expected. But much more effective information can be gotten from the budget by using it as a planning tool, not just a measurement tool. What else can the practice do to increase revenue? Focusing on specific programs is more successful than just hoping revenue increases will occur. What specifically is an area the practice would like to focus on?

What is the effect on net cash flow of both the revenue and expense changes expected to occur in the budget year? Is cash flow at the desired level? If not, what other things can be done to increase revenue? Will increased marketing help? Or increased efficiency in the use of support staff? Or
additional focus on services in addition to the pre-anesthetic blood work? Or can the planned expense changes be spread out into future years?

**TIPS FOR SUCCESS**

A budget is an iterative process—the practice will likely change revenue and expenses and the corresponding operational plans several times before deciding on a final choice of goals for the practice in the next year. The final decision will depend on the net cash flow desired next year as well as the importance of individual goals. Remember to be liberal with expense estimates and conservative with revenue estimates and to leave room for surprises. No budget will ever correspond 100% to reality and it is critical to have some cushion built in for unanticipated expenses or revenue programs that don’t meet their goals.

Start with a simple budget and then use it to calculate the effect of more complicated plans. A simple budget is better than no budget at all and all everyone will become more adept at using the budget and will find it more and more helpful, the more it is used. While this information should help start the process, don’t forget the practice’s financial advisor is always available for additional help.
IF YOUR INVENTORY ACCOUNTING ISN’T RIGHT, YOU’RE FLYING BLIND

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INTRODUCTION

Drugs, medical supplies and food costs are some of the biggest expenses a practice incurs and these costs are increasing in most practices. What should the practice do if these costs are too high? First of all, it is important to drill deeper into the accounting and PIMS records to truly understand whether the costs are too high and WHY this is so. Secondly (and most importantly), changes need to be made in the practice to improve inventory efficiency and costs. Effective inventory management is key to keeping these costs under control. Inventory control is sometimes seen as a boring and tedious task, but it can have a huge impact on the practice’s profitability and is actually one of the easier things to do well in a practice.

HOW TO KNOW IF INVENTORY COSTS ARE TOO HIGH?

Before delving into inventory accounting, let’s review the two financial statements most commonly seen and used by a practice. The first is the income statement. This is an accounting statement reflecting the financial performance of an entity between two points in time; i.e. it shows what the revenue and expenses of the practice are for a particular period. This statement can be called many things but still represents the equation: Revenue-Expenses = Net Income. Some of the names used are:

- Profit and loss statement (P&L)
- Statement of operations
- Statement of revenues and expenses

The balance sheet is less useful usually for management analysis but still an important statement. The balance sheet summarizes the financial position of an entity at a point in time; i.e. it lists all of the assets and liabilities of the practice.

Most practices review their drugs, medical supplies and food expense numbers to help determine if their costs are too high. There are published benchmarks readily available for comparison for primary care companion animal practices. And while every practice is a little different, these benchmarks can be very effective in determining if a problem may exist. For this comparison to be effective, however, the expense figures in the practice’s financial statements and tax returns must be accurate and often these figures are not correct.

When analyzing inventory expense, the figure should represent what drugs, medical supplies and food items are USED or SOLD TO CLIENTS during the period in question, not what items are PURCHASED. This is a critical distinction. When a cash basis of accounting is used, the expense included in the profit and loss statement (P&L) is often what is purchased, not what is used. In accrual basis financial statements, the expense should be what is used or sold to clients not what is purchased. In both cases, poor inventory control or random adjustments may mean that the figure is inaccurate so even when accrual accounting is used, the figure may not be accurate.

Most practices use a cash basis of accounting for internal purposes. When using a cash basis of accounting, fluctuations in when bills are paid (whether deliberate or accidental) can have a significant impact on analysis. For example, let’s assume a practice normally spends about $30,000/month on drugs & medical supplies expense. When comparing monthly expenditures for the last month, the owner notices:

<table>
<thead>
<tr>
<th>Apr</th>
<th>May</th>
</tr>
</thead>
<tbody>
<tr>
<td>555</td>
<td>555</td>
</tr>
</tbody>
</table>
Drugs & medical supplies expense $30,000 $25,000

At first, it looks like the practice’s efforts to better control inventory are paying off. However, it turns out that the decrease occurred because the bookkeeper went on vacation during the last week of May and didn’t pay the rest of that month’s bills until June. June’s expense was $35,000. It is important that the people doing the bookkeeping and the financial analysis have enough real accounting knowledge to understand the implications of cash vs. accrual accounting and even though the practice likely doesn’t want to entirely switch over to accrual accounting, some off the books analysis of the inventory costs can be very helpful.

There are two critical inventory figures in the practice’s financial statements:

- Inventory expense (P&L statement)
- Inventory asset (Balance sheet)

Ideally, when inventory is initially purchased and stored on the shelves of the practice, it is recorded in the balance sheet inventory asset account and when it is used or sold to a client, it is treated as an expense on the income statement. The inventory asset (the amount on the balance sheet) should represent the amount of product sitting on the practice’s shelves at a point in time. This figure is often used to calculate the accrual based figure for the amount of product sold or used and if the balance sheet figure isn’t right than the figure representing used or sold product isn’t right.

The balance sheet number is often not accurate because:

- The inventory isn’t counted at the end of the year and so no one really knows how much is on the shelves
- The amount of inventory on hand from the Practice Information Management System (PIMS) inventory module is used for the balance sheet figure but the PIMS report isn’t correct
- The balance sheet figure is manipulated to show a more favorable tax position

If the balance sheet figure isn’t right, the income statement figure isn’t right and if the expense isn’t right, the practice team doesn’t know if the costs are within normal limits.

So what numbers does the practice need to really know if the inventory expense is reasonable? The practice needs an accrual basis revenue number and an accrual basis inventory expense figure. The accrual basis revenue figure is easily found in the PIMS. The accrual basis inventory expense figure can come from the financial statements if they are accrual basis and if they are accurate or the figure can be calculated each year using the following formula:

\[
\begin{align*}
\text{Inventory on shelves at the beginning of the year:} & \quad \$75,000 \\
\text{Inventory purchases during the year:} & \quad \$150,000 \\
\text{Inventory on shelves at the end of the year:} & \quad (\$65,000) \\
\text{Inventory usage during the year:} & \quad $162,000
\end{align*}
\]

The “inventory on the shelves” figures can come from the PIMS report or an actual physical count. If using PIMS report figures, the practice needs to be sure that the figures equal what is actually on the shelves. Inventory purchase figures come from the accounting software.

The most common categories used to record purchases in the accounting system are:

- Drugs & medical supplies expense
- Laboratory expense
- Medical waste disposal
- OTC product expense
- Dietary product expense
A relatively simple way to make the adjustment needed to accurately record an accrual basis inventory expense figure in the financial statements is as follows:

- Record purchases in the above accounts during the period desired (month, quarter, year)
- At the end of the period, count the inventory or retrieve the actual inventory on hand figure from the PIMS (assuming you are sure that number is right)
- Record the inventory on hand figure in the balance sheet inventory asset account with an offsetting credit in the appropriate inventory expense account (if there is already some amount in the inventory asset account, this amount should be adjusted to the current correct value with either an offsetting debit or credit to inventory expense)

**WHAT CAUSES INVENTORY COSTS TO BE TOO HIGH?**

Of course getting the right figures is just the starting point. If you determine the costs are too high, you must then determine why. Inventory costs can be high for a number of reasons including:

- Too much is paid for particular products
- Too much inventory sits on the shelves without being used—this can occur because the practice carries too many products in a certain category, carries too much of a particular product given the short time period it takes to order and receive the product or keeps products on the shelves that are almost never used
- Product is stolen by either clients or employees
- Product is accidentally given away

**GOOD INVENTORY CONTROL**

Correcting these issues is not that difficult but it takes a system and the right people administering that system for this to happen. Key controls are necessary at various point; one of the most important inventory management procedures is regular counting of the products on the shelves with comparison to the PIMS records. Most practices do not count their inventory on a regular basis. At best, they do it once a year for tax purposes as discussed above. The count done for tax purposes is not sufficient to make sure that the inventory system is working effectively. All items need to be counted on a more regular basis.

If the practice has not been using its PIMS inventory module effectively, there may initially be many discrepancies between the PIMS and what is on the shelves. Before implementing regular counting of certain products, it may be necessary to first count everything in the hospital and update the PIMS records. This project should generally be done when the practice is closed and it is essential that all inventory is counted. Going through every room of the hospital and making a list of all storage places (shelves, drawers, etc.) will help.

Once the actual inventory in the clinic equals the PIMS, a regular counting system can be initiated. The items most susceptible to theft are food, heartworm preventative and flea/tick products; these should be counted at least monthly to make sure they are not being given to clients without being charged for or stolen. In the beginning, it may be necessary to count them more frequently if the practice is having problems keeping track of the inventory. Make a list of all of these items (list each size individually) and then divide it by four so that each item is counted once a month.

Count the product on hand and immediately check the balance indicated in the computer for this product. It is critical to do these two steps right after each other so that the comparisons are between “apples and apples.” If the product is counted and the computer balance checked later, product could be sold or received and added or deducted from the computer balance which would then not agree with the amount counted.

The counts and computer work should initially be done by a practice owner and should be “visible”; i.e. done during business hours so that the staff is aware that this procedure is taking place. The counts should not be done before or after hours and they should be done when several staff members are around. The counts shouldn’t be treated as an unusual procedure nor should it be suggested that they are being
implemented due to the possibility of staff theft, but do let it be known that this is a new procedure that will be done regularly. If asked why the counts are being implemented it should be said that the cost of inventory is one of the biggest expenses in the hospital and with the growth of the practice, the owners want to control this cost a little better by improving the inventory system.

If there are discrepancies in the counts, ask the appropriate questions of the staff people:

- Are there any product purchase invoices that haven’t been entered into the inventory module?
- Was any product used in-house that hasn’t been recorded in the inventory module? (i.e. through a dummy client account?)
- Was any product sent home with either clients or employees that hasn’t yet been recorded on an invoice? This is more often a problem with hospitalized or boarding patients than with out-patients.
- Was any product returned to the manufacturer that hasn’t been deleted from the inventory module?
- Was product used for any other reason and not deleted from the inventory module?
- Is product stored in some other location which may not have been counted?
- Does the staff have any other ideas as to why the discrepancies exist?

Depending on the level of the discrepancies and whether or not reasonable explanations can be found for the discrepancies, it may be necessary to institute more stringent inventory control procedures until the problem can be found.

Once this part of the system is in place for food, heartworm preventative and flea and tick products, expand the counts to include other products. Unless the practice is experiencing a problem, the counts on the other products usually do not need to be done as frequently. Frequency will be determined by the $ value of the item, its likelihood of being stolen or given away and your experience with this product in your clinic. Don’t forget that controlled substances should be counted much more frequently.
LESSONS FROM VIDEO: SUCCESSFULLY FORWARD BOOKING CLIENTS IN YOUR PRACTICE

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INTRODUCTION

Forward booking simply means booking the pet’s next appointment before he/she leaves the practice after the current visit. At my dentist’s office, I can’t get out of the exam room, much less the practice without booking my next preventive care appointment, but we’ve been slower to adopt this concept in veterinary medicine. Forward booking has advantages for everyone—the client is more likely to bring Fluffy in on a timely basis if the appointment is already on their calendar, the pet will stay healthier if they’re seen regularly and the practice gets the satisfaction of providing pets with better care as well as enjoying the financial return.

The Veterinary Hospital Management Association (VHMA) has collected data on the usage of forward booking in practices. The first question asked: Is your practice “forward booking” (i.e. making an appointment for the next visit before the client leaves the practice) for RECHECKS/MEDICAL PROGRESS EXAMS?

![Graph showing percentage of practices forward booking recheck appointments]

As can be seen above, in 2018, about 75% of the respondents answered “always” or “most of the time”; this is the same % as in 2014. No surprise, although a little disappointing, that there wasn’t much change because forward booking for medical appointments has been going on for many years in veterinary medicine.

The second question in the VHMA surveys asked: Is your practice “forward booking” (i.e. making an appointment for the next visit before the client leaves the practice) for ANNUAL/SEMI-ANNUAL WELLNESS OR PREVENTIVE HEALTHCARE EXAMS? 2018 responses showed a small increase over 2014.

As noted above, only a small number of practices are actively forward booking preventive care appointments. Like any cultural change in a practice, it will take time until forward booking becomes “the way we do things here.” There are specific things you can do, however, to make implementation of this change easier and faster; some of these are discussed below:

Discuss the benefits with everyone in the practice

Don’t assume everyone in the practice already understands this. It’s important to talk through the advantages of forward booking as a part of your team training. Particularly focus on the benefits to the pet and the client and help the team see how those advantages outweigh the hassle of implementing the change. If the team doesn’t genuinely believe this is a good thing for the client and pet, it will be hard to get them to have forward booking discussions with clients. Doctors and practice owners must be on board; no change will work will without that leadership involvement. The entire practice team has to
agree on the philosophy of forward booking for ALL pets. The mantra should be: “no client will leave the practice without his or her pet’s next appointment booked.”

The first and most important benefit of forward booking is seeing the pet and pet owner more regularly; the pet will stay healthier if the practice has a more frequent opportunity to provide the necessary care. And of course, the practice team gets the satisfaction of providing pets with better care; that’s why they come to work every day.

Client convenience is another significant benefit. Most pet owners are busy and silly though it may sound, having to call and make an appointment is a hassle especially if the pet owner doesn’t think about it until 10 at night. Research done for the Partners for Healthy Pets suggests that many pet owners have every intention of getting regular check-ups for their pets, but with the many day to day demands of a typical household, calling to schedule an appointment just doesn’t get done. With an appointment already scheduled, pet owners are more likely to either keep the appointment or reschedule for a different time. One of the respondents above posted this link—it’s a great read from the client’s perspective: https://www.linkedin.com/pulse/forward-booking-your-veterinary-practice-seriouslywhy-judy-gillespie

And, of course, forward booking can help the practice financially as well and financially successful practices are those that can continue to invest in better pet care and their teams.

Shift your thinking about what clients will or won’t do

A few pet owners won’t like the change and won’t make their appointments early but this isn’t a reason not to implement forward booking. There isn’t a single thing you recommend in your practice that EVERY client accepts. For the majority of clients, forward booking shifts the responsibility for scheduling the visit away from them and this means it’s much more likely the pet will be seen on a timely basis. Most people love making their dental appointment in advance because it’s one less thing they have to remember to do and they will feel the same about their pet’s appointment. It doesn’t make sense that there would be something unique about forward booking veterinary appointments that clients wouldn’t like when they like doing it for the dentist, the hair stylist or their human doctor.

Words matter

Forward booking preventive care exams works best when the exam room team and the front desk team work in tandem with each other. Most practices have found that including the doctor in these conversations makes a difference in client willingness to forward book the first time. It doesn’t have to be hard or time-consuming; the doctor can say something like this as they are wrapping up the appointment:

“Max is all up to date on his vaccinations and parasite protection and, if everything goes well, we won’t need to see him again until this time next year. Chelsea will set that appointment up for you before you leave so you don’t have to worry about it.”

The front desk team should acknowledge to the client that most of us don’t know our exact schedule a year in advance, but can suggest an appointment day/time similar to the current one:

“We’ve booked Fluffy’s next appointment for one year from now, again on a Wednesday afternoon. I know many of us have no idea what we are doing in a year but as the date gets closer, you’ll receive plenty of reminders from us. If Fluffy’s appointment time doesn’t work for you, we can always reschedule it to a different day.”

There will likely be some client pushback, particularly when the practice first starts forward booking. Check out the videos at the Partners for Healthy Pets website for ways to deal with this: http://www.partnersforhealthypets.org/communications.aspx
Reminders are critical!

Set up reminders several weeks and then several days in advance of the next year's appointment so the client can change or cancel if needed. Don't forget to consider generational differences and use the type of reminder the client prefers; some clients may want a text, email or phone reminder while others prefer a traditional reminder card. Invest in appointment cards to send home with the clients so they can put the appointment on their calendar if they aren't doing it electronically right then.

You may need to talk with your practice management information software system help desk about how reminders work with forward booked appointments and make sure all clients are getting the appropriate ones.

Don't worry about some clients forgetting their appointment; that is what the reminders are for. Some clients will have to call and change the date, but that's ok too—it still means their pet will likely be seen much sooner than if the practice had to wait for the client to reach out to book the appointment.

Resources

Visit www.PartnersForHealthyPets.org website for a number of resources to make implementing forward booking easier. These include: buttons, posters, a training guide and a number of videos demonstrating how to deal with client pushback and the role of each team member in talking to the clients about forward booking.
INTRODUCTION

It’s hard to know what’s really happening with fees in the profession. If we count each fee, there are well over 100,000,000 data points and any particular study only looks at a fraction of these. So is the data representative of the profession? We also have so many different kinds of practices—for companion animals alone, there are general (high end, main stream, affordable), specialty practices, emergency practices and those associated with animal welfare organizations. And of course, there are mixed animal, equine and food animal practices too.

There have been many studies on the impact of pricing in the last 20 years with comments such as the following coming out of them:

- ~50% of dog/cat owners said: “I would take my dog/cat to the vet more often if each visit was less expensive.” (BVCUS 2011)
- 39% of veterinary team members think cost of care is primary reason clients don’t return to practice (VHMA Insiders’ Insights Nov 2016)
- 64% of pet owners would purchase more pet meds from veterinarians if they offered more competitive prices (Packaged Facts Jan 2018)

A 2015 pilot study between the AVMA Veterinary Economics Division and the National Center for Food and Agricultural Policy showed that the cost of a “routine check-up” ranged from $0-$500 and practice revenue was maximized at $120. Up to $200, demand was elastic, meaning that if the practice charged less, more clients would elect to have the check-up done at that price. There were some clients to whom price didn’t matter, however, and demand was inelastic from $200-$500.

Although there may be concerns about the quality of some of the study methodology, it is very clear that the majority of the pet owner studies indicate price is an issue for many pet owners. Only a very few studies said pricing didn’t matter. Most practice team members say pricing is an issue and that the most common reason pet owners leave a practice is cost. There is no question that a more thoughtful focus on pricing makes sense.

FEE CHANGES IN 2018

There is limited information about price increases in 2018. However, each year the Veterinary Hospital Manager Association (VHMA) asks practices about fee increases for the year; either those planned or already implemented. Over 200 people responded to the survey and the vast majority of those practices said yes to the question: “Have you or will you raise your professional service fees in 2018?” A larger % in 2018 said they would be increasing fees on both shopped and non-shopped services compared to 2017.

Most of the hospitals said the average increase on shopped services would be 3% and the average increase on non-shopped services would be between 4-6%.

In two surveys done in early 2018, the VHMA also explored the factors or strategies used by practices in setting fees. When asked about general factors or strategies considered in the decision about whether to increase the fees on both shopped and non-shopped services, responses were similar for both types of fees and the main factors taken into account for each were:

- Inflation
• Overall cost of doing business
• Increases in practice costs
• What other practices are charging
• Fee references such as AAHA, WMP

Compared to 2017, practices appear to be feeling even more comfortable with the economy and their clients’ willingness to spend money on veterinary care as evidenced by their level of fee increases and the types of fee increases the practice made or is planning to make. But are these types of increases sustainable? Should practices be concerned about the long-term impact? What factors should practices be thinking about when making decisions about fee increases?

DOES YOUR PRACTICE HAVE A PRICING PROBLEM?

Should you re-think your current fee strategy? Not if:

• Your practice is truly profitable
• Your practice has real growth in:
  o Transactions
  o Visits
  o New clients

If the above is true, it is likely that your current fee strategy works for your practice and your client base, at least at the current time. It is important to re-evaluate your profitability and transaction/visit/new client growth at least annually.

If your practice isn’t as profitable as you’d like or isn’t growing, price may not be the issue, but you need to at least consider that possibility. In addition to better pricing strategies, don’t forget that profitability can be increased through other means such as increasing marketing programs and bringing in more clients, reducing expenses and improving productivity.

PRICING STRATEGY

In many practices the biggest component of the hospital’s pricing strategy is by what percentage should fees be raised? Pricing, however, is much more than that. Pricing is really a marketing issue and, as can be seen in figure 1, is just one component of the marketing mix. Pricing can’t be determined without

looking at the other components as well and the have to correlate. For example, if a business offers a low quality product in a bad location but tries to charge a high price for it, consumers won’t buy it. Figure 2 demonstrates the pillars of pricing strategy that link with the marketing strategy; again, prices can’t be set in a vacuum.

**VALUE BASED PRICING**

As noted above very little veterinary price strategy research has been done, although some organizations are currently in the process of looking at the price sensitivity of consumers in the market for veterinary services and at more sophisticated pricing methodologies. A first step in exploring additional strategies occurred on August 6th, 2018, when the Veterinary Hospital Managers Association held its Critical Issues Summit on Pricing. Leading the discussion was Utpal Dholakia, Ph.D., a Rice University professor of marketing and noted author and authority on motivational psychology, consumer behavior and marketing.

Dr. Dholakia’s focus was on value based pricing. Interestingly, one of the VHMA survey questions asked “What is the primary approach your practice uses in setting SERVICE prices?” Answers varied widely; the approach selected by the biggest group of respondents was cost-based pricing with 40% of the respondents choosing this answer. Costs are certainly an important factor in setting prices; in the long-run, if a practice charges fees that don’t cover its costs, it will go bankrupt. However, cost can’t be the only factor in setting prices because, to a large extent, customers don’t care what it costs to provide a service. They want to pay a price that correlates with the value they receive. Only about 5% of the respondents to this survey selected value based pricing as their primary approach. We don’t know why so few practices chose that method but it is probably because it can be difficult to correlate value to price and we don’t have strong data in veterinary medicine to indicate what kind of value pet owners are willing to pay more for. This is clearly a strategy practices can use more effectively.

A product or a service offered by a business “is a bundle of features that provides quantifiable functional and hedonic benefits to the customer.” A functional benefit of a vaccination would be the protection the pet gets against rabies or another disease. Hedonic benefits are intangible and emotion producing. An example here would be the satisfaction the pet owner gets from taking care of their pet and the contribution the pet owner makes to a long and happy life by a family member. An example of the types of benefits related to the services pet owners receive in a typical surgical situation is shown below:

<table>
<thead>
<tr>
<th>Bundle of features</th>
<th>Functional &amp; hedonic benefits</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Exam &amp; consultation</td>
<td>• Problem properly diagnosed</td>
</tr>
<tr>
<td>• Diagnostics</td>
<td>• Owner feels they are getting the best advice &amp; recommendations</td>
</tr>
<tr>
<td>• Surgery</td>
<td>• Potential underlying issues identified</td>
</tr>
<tr>
<td>• Hospitalization &amp; nursing care</td>
<td>• Owner’s anxiety about anesthesia is reduced</td>
</tr>
<tr>
<td>• Medications</td>
<td>• Rusty won’t limp any more</td>
</tr>
<tr>
<td>• Rehab</td>
<td>• Gold standard treatment</td>
</tr>
<tr>
<td>• Follow-up appointments</td>
<td>• Surgery performed by an experienced doctor</td>
</tr>
<tr>
<td></td>
<td>• Owner feels like they are doing the right thing for their dog</td>
</tr>
<tr>
<td></td>
<td>• Rusty gets appropriate post-op care</td>
</tr>
<tr>
<td></td>
<td>• Owner feels good that Rusty is being watched over by the experts</td>
</tr>
<tr>
<td></td>
<td>• Potential infection is properly managed</td>
</tr>
<tr>
<td></td>
<td>• Pain is kept to a minimum</td>
</tr>
<tr>
<td></td>
<td>• Owner’s concern about pain is reduced</td>
</tr>
<tr>
<td></td>
<td>• Rusty’s recovery is faster and better</td>
</tr>
<tr>
<td></td>
<td>• Owner doesn’t have to do the PT work</td>
</tr>
<tr>
<td></td>
<td>• Any potential problems with recovery are caught sooner rather than later</td>
</tr>
<tr>
<td></td>
<td>• Owner knows they have someone to turn to for continued advice</td>
</tr>
</tbody>
</table>
Breaking down a product or service into its functional and hedonic benefits is difficult and time-consuming, especially for a small business. However, this is definitely an area that is worthy of profession wide research and can be done to some extent by individual practices through client surveys, focus groups and conversations with clients.

Hedonic benefits may cost very little in dollars to the practice and customers are often very willing to pay a lot for these benefits. Because the management team doesn’t really understand what they are and how important they are to pet owners, they often get ignored; yet emphasizing these benefits in marketing materials and in conversations with pet owners helps support the practice’s prices in the consumer’s eye.

WHAT DO OTHERS CHARGE?

According to Dr. Dholakia, consumers almost never make judgements about prices in isolation. The judge a product or service’s price in relationship to other prices for the same or similar products or services. These other prices are called “reference prices.”

Knowing what other practices are charging is important so your practice has an idea of where it fits into the community, both from a price perspective and from a value perspective. However, this doesn’t mean you need to match those prices, either the higher ones or the lower ones. Any evaluation of another business’ prices must include an evaluation of the value that business provides as well. For example, if the practice 2 miles away from you has prices that are 20% higher than your practice’s, should you raise your fees? Maybe or maybe not. If they are located in a nicer facility, have more convenient hours and a better client service experience than your practice offers, a price increase will probably not be well received. However, if your practice is equivalent or greater in value, then increasing prices may be worth considering.

Fee references such as the AAHA Veterinary Fee Reference and information found in the Benchmarks 2017: A Study of Well-Managed Practices are essentially a large conglomeration of data regarding what other practices are charging. They are useful books to understand where your practice falls in the mix and to see how certain types of your fees are priced compared to others but they shouldn’t be an absolute mandate for what your prices should be because they are not local in nature. As much local research as possible should be done to understand both the prices charged by other practices and the value those practices give to the pet owner in comparison with your practice.

INFLATION

Many practices say that they use inflation as a guide to set fees. And while this is no doubt true for a small number of practices, in reality, the average fee increases made by most practices are well above inflation and have been for many years as shown in this chart prepared by the AVMA:

This analysis ends in 2014 but similar results can be seen in the years following. Inflation from 2014 to 2018 ranged from 0%-3% and yet most practices are raising their fees by greater percentages. Note that
the bottom two lines in this chart are the number of cats and dogs who don’t get any veterinary care at all; these figures increase as prices go up. In the long-run, regular fee increases over the rate of inflation may be damaging to a practice and to our profession, particularly if no increased value is seen by the pet owner and the pet owner is not in a high income bracket.

A practice certainly doesn’t have to limit its fee increases to the rate of inflation but if the increases will be higher, it is important to think through the long term ramifications of those higher increases and whether the practice is offering increased value desired by the pet owner in exchange. The inflation figures used above are those from the Consumer Price Index. It has been suggested that “veterinary inflation” is higher but this isn’t always true and understanding the actual price increases for the goods and services your practice buys is an important part of the fee-setting process. Remember, too, that inflation is a change in price for the goods and services the practice currently purchases. Buying a more expensive piece of equipment with more functionality is not inflation; that is an investment that the practice ultimately hopes to reap additional rewards from.

ACCURATELY UNDERSTAND THE OVERALL COST OF DOING BUSINESS AND INCREASES IN PRACTICE COSTS

In the long-run, if a practice charges fees that don’t cover its costs, it will go bankrupt. However, cost can’t be the only factor in setting prices because, to a large extent, customers don’t care what it costs to provide a service. They want to pay a price that correlates with the value they receive. A practice therefore needs to operate its business in a manner that allows it to price goods and services in a way that correlates with value and also covers costs.

Practices often overestimate the amount by which their costs have increased in any given year and by what amount they need to increase fees to cover those costs. Many practice owners or managers would tell you that the average cost of their medications has gone up 5% or 8% or more and yet information from Animalytix indicates this figure was closer to 1.8% in 2018. (Animalytix LLC is a large animal health industry clearinghouse for sales and market share data related to the products sold to veterinary hospitals.) The 1.8% figure quoted above is an average; certainly some products increased more than that but others increased less. The fee increase to pet owners to cover this 1.8% increase in costs is about 1/3%. Of course, this isn’t the only cost increase seen in a practice and a practice’s cost increase for medications may be more than 1.8% but the fee increases practices pass on to consumers often are significantly higher than that needed to cover increased operating costs.

Again, a practice certainly doesn’t have to limit its fee increases to the level of cost increases seen in the practice but if the increases will be higher, it is important to understand the long term ramifications of those higher increases.

The above are just some of the factors practices need to consider in setting or changing prices. In addition to the absolute price, practices also need to look at price execution which means does the practice actually charge the client what the stated price is or are there discounts applied. And what is the impact of payment options on acceptance of price by the pet owner? If the practice does a better job of educating pet owners about payment options, are more pet owners accepting of the price?
Get Real About Raises
Bash Halow, CVPM, LVT

In this article we’ll explore
- How to know if you deserve a raise
- What to do in preparation for asking for a raise
- How to ask for a raise
- What to do when you don’t get the increase that you want

How To Know If You Deserve A Raise
There are a couple of ways that you can find out if you deserve a raise. Here are just a few, but we’ll cover more of this in the lecture.

Know the ‘Going Rate’
Though employees have the legal right to talk openly about their wages, the question can make others uncomfortable. For information on what the average veterinary professional earns, look for resources published through the VHMA, NAVTA or any state or local veterinary management or technician organization.

The Reality: A 3% Unemployment Rate
In general, unemployment is at historic lows and specific to our industry, there are severe doctor and licensed technician shortages nationwide. If you are a doctor or licensed technician, asking for a raise is really just part of a marketplace tide that’s flowing in your direction. Still, it’s best to be tactful. Your employer might be forced to choose between a raise that he or she will struggle to support or the loss of an employee that he or she can’t afford to lose. It’s not a great position to be in, so be respectful.

You Earn Enough For the Practice To Support It
This is the best way to come to the table: knowing that the practice is more profitable because of your specific efforts. Client care reps, are you capturing those phone shoppers? Assistants, are you effective at communicating the value of annual wellness bloods? These sorts of sales can be tracked through the software, so it should be easy to demonstrate the kind of revenue that your efforts are stimulating. Bringing in one more new client, stopping an upset client from leaving the practice, rallying your team to be more supportive of practice-wide initiatives…these actions are worth tens of thousands of dollars. It’s not unreasonable for you to share in that wealth; you were responsible for generating it!

What To Do In Preparation For A Raise
If you have just started at a veterinary practice, ask what you will be evaluated upon during your annual review. Ask things like, “As my employer, what are the top five things that you would like to see me demonstrate over the next year? Would it be all right if I checked with you to see how I’m doing in this area before my next review?” You might also say something like, “I’m glad to be working here and I want to make you proud that you hired me. What do I have to do from now to my next review to demonstrate that I’m a really valuable member of the team?”

Managers, could you answer the above questions? Are you telling new hires what their reviews will be predicated upon?

How To Ask For A Raise

Be Respectful
I have looked at dozens of financial statements from veterinary practices. Most don’t show a lot of profit. Owning a veterinary practice, working as a veterinarian, and shouldering the lion’s share of responsibility, risk, and decision-making is stressful to say the least.Swaggering into a practice manager or owner’s office and demanding more money may be justified in today’s
economic climate, but it’s rude and terribly inconsiderate given what your practice leaders are
doing on a day-to-day basis to provide you with employment.

You should also know that most practice owners would love to give everyone a raise, but worry
they won’t have the cash flow to do it. Payroll at a practice grossing 1 million a year is probably
between 5 and 8 thousand dollars per week. Imagine floating that kind of responsibility 52 weeks
a year, year-after-year. Remember too that your practice owner has that responsibility when you
are sunbathing on Labor Day, crying ‘Happy New Year’ on December 31st, and toasting weenies
on July 4th. It’s a never-ending responsibility and a source of constant low grade anxiety. Be
nice and be respectful!

Determining What’s Reasonable
Most practices can afford to spend 16% of their gross revenue on doctors, about 13% on their
technicians, and about 9% on their receptionists and technicians. Let’s say that new clients are
worth about 400-600 dollars in gross sales per year (avg. transaction times average number of
visits). As a receptionist, you commit to working hard and converting 20% more phone shoppers
a month. At your practice, that turns out to be 4 more new clients that the practice otherwise
would not have had without your efforts. Four new clients times 500 dollars is 20 thousand
dollars a year. If we multiply that by 9%, that’s 1,800 dollars, or 35 dollars a week, or roughly an
increase of one dollar per hour. Ask for a dollar increase.

Your Practice Owner Needs Help, So Offer It.
Don’t just come to the table with demands, come to the table with offers of how you can help
grow the business, or take some of the responsibility off your practice owner or manager’s
shoulders. Let them know that you get it: this is a hard business and you want to help…and you
would be very appreciative if they could provide you with remuneration to do it.

Be Willing to Walk
If a wage increase is that important to you, you can probably find it in today’s market, but it will
mean that you have to leave your current job. That’s often a big deal and walking away from
friends, support, clients that you know, and a schedule that you love may not be worth that paltry
35 dollars a week.

Working in a veterinary practice, with the right people, can be very fulfilling and lucrative. I invite
everyone to think how we can leverage great wages and benefits to attract talented, fun, engaged
people to work with us, to create an outstanding team and practice culture, and to grow our
business to support the wage increases.
Consolidation
Consolidation in the veterinary space, a normal phenomenon that occurs in all industries, continues to heat up. According to experts, we are in stage 2-3 of a four part process in which smaller companies merge to create larger, more efficient enterprises. These companies then form strategic alliances with others to create industry giants whose products, sales, and influence can span the globe.

Consolidation is driving historic sales prices. In the past, veterinary practices were often sold for a 4-6 multiple of their adjusted net earnings (EBITDA). Today, some companies are paying 8-11 times EBITDA for single or small groups of practices with large groups of practices being sold for as much as 13-19 times their EBITDA.

Consolidation puts a significant amount of pressure on older veterinarians to sell and tends to keep private practitioners shut out of a bidding war that they cannot afford. Additionally, consolidation is having these effects on our industry:

- It is driving up the wages of doctors and credentialed technicians.
- It is improving the work life balance of veterinarians and some lay staff.
- It is providing more upward mobility to support staff members, former practice owners, and managers as opportunities inside groups that own 300-1000 veterinary practices grow.
- It is drawing some pharma funding and support away from private practices.
- It is putting pressure on smaller suppliers as wholesale deals lower the profit margin on products.
- It is making website visibility more difficult for private, smaller practices as larger corporations flex their expertise and wealth to optimally advertise online.
- It forces veterinary associations, at every level, to reevaluate the needs of their membership and to decide whether to emphasize the needs of the consolidated groups or the private practices that they have traditionally served.
- It reduces the need for independent continuing education events as corporations provide more internal learning opportunities.
- It draws outside investors into the marketplace whose sole interest is to capitalize on the consolidation trend by buying practices, packaging them, and then reselling them at a higher multiple to larger entities.

Independent Veterinary Practitioners Association
In response to what some felt was a lack of advocacy for their needs, some private practitioners decided to create an association whose goal was to specifically address the inequality in buying power and the unfair influence that they believed corporations were having on the industry. The Independent Veterinary Practitioners Association (IVPA) is currently looking for ways to leverage its size and influence to help private practitioners everywhere.

Data Mining and Ownership
There is a growing concern that many of the companies that have access to client data through veterinarians’ POS software are selling that data to third parties who can then turn around and use that same data to compete with the private practices from where it was obtained. The Association of Veterinary Informatics (AVI) is a not-for-profit group of veterinary professionals that explores, encourages, and assists private practices with understanding and leveraging data. As concern about data mining and ownership mounts, interest in the AVI grows. Their annual conference is held in conjunction with all three Fetch conferences, so be sure to attend.
Integration
Companies like Idexx and Henry Schein are working hard to integrate their software with all other aspects of their business. Veterinary practices that partner with these two industry giants will have access to more detailed reportage and more client follow-through capability. The ability of Avimark’s Rapport to now allow clients to schedule their own appointments will undoubtedly be leveraged by the software’s corporate accounts to streamline new client acquisition.

Payment Options
Companies like Vetbilling.com now offer Pet Health Savings Accounts and Wellness Plans. They are also beta testing an app that can efficiently assist the practice with writing payment plans for clients. The app:

- Allows practices to quickly gather client data;
- Runs a soft (doesn’t affect the client’s credit score) credit check (with the client’s permission of course);
- Allows the client to provide verified approval for the credit check via their cell phone;
- Provides immediate recommendations to the practice for the terms of the payment plan;
- Is free to the practice, provided that the client agrees to the plan.

The company has seen dramatic success with its product at some veterinary hospitals. In one case, a practice increased revenue by 400K in one year!

The Explosion of CBD Products for Pets
The decriminalization of CBD in many states has waves of pet owners looking for sources of CBD for their pets. Veterinarycannabis.org, led by Dr. Casara Andre, aims to teach veterinary professionals how to safely and legally educate pet owners about CBD in pets.

Erosion of Food, Pharmacy and OTC Sales
Online pharmacies and giants like Amazon, CVS, Wal-Mart, and Chewies continue to flex their marketing prowess and online ubiquity to capture new clients and lock them into to sales. Pharmacy sales at many practices have dropped by 5% and it’s likely that both food and pharmacy sales will rapidly decline as the above-mentioned companies and others use online tracking to understand the buying habits of pet owners and to market to them with increased effectiveness.

The Pet Experience
Petco is following the lead of companies like Whole Foods and Apple Computer and creating retail experiences for pet owners. The model continues to evolve. To date, PetCoach, as the venture is called has experimented with pet food cooking stations, on demand veterinary professional advice through a monthly subscription service, telemedicine, grooming, and pet training. These stores are tracking the movement of clients through the store, taking note of what clients are finding interest in and what they are not, and making adjustments accordingly.

Higher Wages and A Shortage of Veterinary Professionals
It was mentioned above, but it merits reemphasizing: the current growth in our industry, the changing demographics, and competition by veterinary corporations is creating a shortage of veterinarians and veterinary technicians. Some veterinary practices have been searching for associate veterinarians for more than a year without a single applicant.

As ownership opportunities decrease, it is likely that fewer students will opt for a career in veterinary medicine that comes with an MD-sized debt without the MD-sized salary. Already many hospitals are searching for foreign trained veterinarians to close their ranks.

Telemedicine
Despite the smoke, telemedicine has yet to catch fire. Still, some practices are exploring free telephone consults with veterinarians as a way to introduce new clients to the practice and to try
to drive sales. Practices that have wellness plans are using telemedicine to connect licensed veterinary technicians with plan holders that have had a recent visit to the vet. Instead of coming into the practice, the plan holder can call a veterinary professional and consult with him or her over the phone. Under the direction of the veterinarian, advice or medicine can be dispensed, provided that the client patient relationship is still in good standing. The practice can then take advantage of the appointment slot that would have otherwise been filled to provide service to a new client or one that is not on the plan. Since the plan holders are paying a monthly fee for the service, there is no drop in revenue.

At the time of this writing, there are other new trends and products, but we’ll discuss these at the meetings in Kansas and San Diego.
Wanted Octopus To Work Busy Reception Desk
Bash Halow, CVPM, LVT

A well-trained client service representative (CSR) can increase a practice’s revenue by tens of thousands of dollars a year. He or she can build invaluable loyalty with clients and bring extra value to every patient interaction. Unfortunately, CSRs are given an excessively long list of tasks that take them away from their primary role as client service facilitator, or are demoralized as low ranking members of the veterinary practice team. In this lecture, we’ll discuss a business model that demands excellent client service from a CSR team that’s not given the training, the time, or the respect to do the job properly.

A Task List That Never Stops Growing
CSRs are turned to on a regular basis to get additional tasks completed in their ‘free time’. Here’s a list of some that are especially time consuming, that are not directly involved in the business of connecting with clients, and suggestions on what to do to improve.

Chart Management
Practices that don’t have electronic charts force their CSR team to waste dozens of hours of time every week looking for, and organizing, paper charts. This mire also makes for a less-than-stellar client experience, as CSRs have to hunt down a patient chart before they can even talk to a client. Make the leap, go paperless or paper-lite. You’ll free up valuable time in which your client service representative can focus on socializing not searching.

Coupon and Discount Management
Having team members organize discount coupons and arranging for client rebates is a nice thought, but it’s work that the vendors, pharma companies, or clients themselves should be doing, not the CSR. If the rebate/discount means that your team members have to do the ‘paperwork’ in order for the client to get the rebate, go with another vendor.

Babysitting Computers and Equipment
Printers that regularly jam, fax machines that won’t fax, slow computers, and inefficiently laid out work areas make an already stressful job nearly unbearable and most likely undoable. Shell out the money. Upgrade the equipment and hire someone to optimize your computers and software so that they work efficiently.

Everyone’s Secretary or Executive Assistant
I recently visited a practice where one client care representative had to research veterinary license requirements in other states for one of the vets. At another a veterinary assistant asked the receptionist, “Did you order lunch yet?” Am I employing customer reps or diner waitresses? The CSR team is a concierge of service for our clients, not for the staff. Stop turning them into secretaries.

Callbacks
Callback and follow up calls make all the difference in the world to compliance, to service, and to great care, but why do the receptionists have to make all of them? Everyone on the veterinary team should be trained and expected to follow up by phone or email with clients. Following up isn’t an added service; it’s a continuation of the one that we started when the client was first seen at the practice.

Reminder Maintenance
CSRs typically don’t have the experience or time to ensure that all patient reminders are right. While they should always have their eye on reminders, the task of ensuring their accuracy should be shared with doctors and technicians.

Looking for AWOL and MIA Employees
Unlike any other job at the practice, CSRs can almost never leave their post. Doctors get to withdraw into their office; vet techs can take a moment to cool off in the pharmacy; assistants can slip a leash onto a patient’s neck and step outside for a walk and a vape. Not so for the CSRs, who almost always have someone in the lobby to entertain or serve, or have someone on the phone that needs help. The last thing they need is to call on the intercom for an employee or doctor and get radio silence in return. It forces them to leave their post and to briskly walk through the whole practice searching for that one rogue employee that won’t answer a page. If someone calls for an employee and they’re not there, say as much, and then offer to help in their place.

Invoicing
Having clients gather up their things in the exam room only to plop it all back down again in front of a CSR so they can pay their bill is an inconvenient mess for all involved. The CSR doesn’t know anything about what happened in the room; he doesn’t know the follow up plan, or even if the invoice is complete. Do the invoicing and scheduling in the room where it’s quiet and where the client can pay attention to the discharge and medicine instructions.

Essential Job Responsibilities of CSRs

Assist The Practice Team With The Orderly Care of Clients and Patients
Someone should have a game plan for how this day will unfold. I nominate a smart, experienced CSR to work in conjunction with a head tech to review the planned case load of the day and create a list of things that all practice team members must do to ensure a welcoming, orderly, professional, and efficient service experience for the day’s clients and patients.

Welcome Clients By Name As They Arrive
As clients enter the practice, even as they enter the parking lot, CSRs should lift their heads up from the papers on their desk (where their work is not) and focus on the client (the real job at hand). CSRs should remember to serve the client, not the computer, not the form, not the clipboard, and certainly not the latest technological error (“I’m so sorry, my computer is acting up this morning”). Do what it takes to know who is coming in, what she needs, and then see to it that she is served when she arrives. Once that’s done, you can attend to the clipboard, slow computer, etc.

Ensure A Timely Visit
Pay attention that team members are alerted that the client has arrived and are mobilized to serve.

Pay Attention To The Client and Pet’s Body Language
Be responsive to clients who look like they are in hurry or who appear worried. Look at the pet. Is he or she elderly and arthritic? Offer a comfy cushion for the pet to rest on instead of the hard floor. Don’t wait to be asked. There’s a saying at the Ritz, “We are ready to serve even the unexpressed wishes of our guests”. Be ready to do the same at your practice.

The Phones
Though we live in an Internet age, phones still rule as the number one tool to convert a new client or to secure the sale of one of the practice’s services. When the phone rings, take a second to focus, and then pick it up with the intention of making a connection to a client. The steps to a great call are:

- Listen to the client’s needs.
- Demonstrate that you understand the need and that it is meaningful.
- If necessary, triage the call so that you understand whether or not this is a now or later visit.
- Do not attempt to diagnose the problem on the phone or to collaborate with the client on home remedies or alternatives to coming to the office.
• Make a case for the value of an appointment and book it.

Phone Shoppers
Phone shoppers/new clients are worth a conservative 14K dollars over the lifetime of a pet. A pleasant, non-rushed greeting, no hold time, and helpful interaction with the client can set your practice apart from the competition and capture the sale. Here are the mileposts of how to handle a phone shopper/new client on the phone.

• Celebrate the call: “I’m so glad you called us! We love meeting new clients. My name is Bash and I’ll be happy to help you.”
• Look for opportunities to get to the know the client: “What’s your pet’s name? What kind of a dog is Bingo? A beagle! I had a beagle when I was kid. They’re the best. Does he enjoy hunting?” There’s no rulebook for this. Just allow yourself to enjoy the experience of meeting a fellow pet lover; everything else will fall into place.
• Don’t be coy about sharing your pricing: Clients are busy. They don’t want to hear you tap dance around providing pricing on the phone. Either give it to them if you can or if you have no idea how much the visit will cost, offer to have another team member call them back with more information. The trick is to regain control of the call so that you’re not left stating prices and waiting for a response from the client. Try, “A visit is 80 dollars and the vaccines range in price from 20-40 dollars. One of the benefits of our practice is that we have lots of financing options and ways to manage the cost of veterinary care. Would you like me to go over those with you? I’ve enjoyed talking with you and would love to find a way for us to care for you and your pet.” This tactic allows you to drive the questions, steer the conversation, and close the sale.
• Find a way to follow up: Lots of phone shoppers move on to the next practice, but before they go, ask for their permission to follow up with them by phone later in the day or the following day. Clients will be impressed that you took the time to call. Even if you don’t capture the client this time, they’ll remember your service and turn to you when the other veterinarian they choose has a service falter (which they inevitably will). Just make sure that you believe in the value of the call and that you aren’t firstly approaching it as a way to capture a sale. “Hi, it’s Bash. We spoke yesterday about Bingo. I just wanted to make sure that everything worked out for you.”

Follow Through
As many as 40% of clients (sometimes more!) do not respond to reminders. Call these clients. “Hi Mrs. Wilson, this is Bash at Downtown Veterinary. How are you? I’m calling because we’ve sent you three emails reminding you that Bingo is due for his annual exam, but we never heard from you. Is everything okay?”

Call back more than once. Clients are busy. There’s a chance that calling them a second or third time will be annoying, but it’s more likely that they haven’t brought Bingo in because they were swamped with other responsibilities. Think of your call as a well-intended reminder and as interest on your part to help the client.

Focus On Connecting And Serving Clients
Converting phone shoppers and underlining the value of your practice to existing clients is worth hundreds of thousands of dollars in annual revenue. If you love to meet and interact with other pet owners; if you love to talk to pet owners about pets; and if you love your practice and think everyone should know how valuable its service is, then I invite you to do what you love to do at work.
Millennials in the Mist: The Good News/Bad News for your practice’s future
Mark Olcott, DVM

Overview:
This talk will center around 3 simple, digestible, and actionable topics.
1) Who are Millennials and why are they important?
2) What are their beliefs about pet care and how they are different from their parents?
3) How should my practice evolve to deal with these realities?

During this interactive discussion, the speaker will present the following innovative ideas:
- Attendees will come away from this talk with a better understanding of not just what Millennials believe with respect to technology but also WHY they believe this. Additionally, we will discuss some "pearls of wisdom" attendees can take back to their practice and implement almost immediately: this doesn't have to be intimidating!

Who are Millennials?
Millennials are individuals born between 1981-1996. As of 2019, millennials are aged between 22-38. They are also more diverse than prior generations, with only 56% of those identifying as white. Accounting for future immigration into the United States, this generation is projected to reach 73 million.

Generational Changes Occurring?
Millennials are the largest and fastest-growing segment of pet owners. Unlike Baby Boomers, who prefer personal communication (talk on phone, face-to-face), they prefer more impersonal and immediate forms of communication, such as texting, email, and social media. Millennials are also living in more multi-generational households, showing an increase in living with their parents, and a decrease in living with their spouse or partner. Additionally, they tend to be delaying having children and becoming pet owners instead.

How are they as Pet Owners?
Millennials are more inclined to become pet owners before they become parents, as they feel that this responsibility better prepares them for the next step. In some cases, millennials view pets as replacements for children. Millennials look for convenience when it comes to buying anything, including pet products. As a result, they are both spending more on pet products and buying more of those products online compared to prior generations.

How to do business with them?
Millennials are used to getting answers immediately, with over 90% accessing the internet on a mobile device and nearly 80% identifying as online shoppers. They want to interact on their own terms, directly, with the majority preferring text messaging and online scheduling rather than the more traditional phone call. Instead of feeling intimidated, practices should adapt to these trends and make changes to how they do business. To attract more millennials, practices should focus more on the user experience, rather than just dollars and discounts.

References:
1) Brakke Consulting
2) VMX
3) WVC
4) Packaged Facts
5) American Veterinary Medical Association (AVMA)
7) Millennial generation is bigger, more diverse than boomers, CNN Business
Paying for care: A summary of the existing and new ways for clients to say “Yes”
Mark Olcott, DVM

Overview:
In virtually every survey of pet owners, cost is cited as a major factor in not just whether they seek care but also how they seek it. This talk will discuss the latest AVMA data on the rising cost of pet care, which is real. At the same time, we will review new findings around trends in consumers’ “willingness to pay” for veterinary care, which are at the same time both very interesting and somewhat alarming. During this interactive discussion, attendees will come away with a better understanding of the evolution in existing solutions (like pet insurance) and some of the newer tools (like wellness plans) that are gaining in popularity.

Pet Insurance:
As advancements are made in veterinary medicine, the cost for pet owners to take care of their pet has increased drastically. An unplanned trip to the vet can cost pet owners between $800-$1,500. Despite the growing costs, under 2% of pets in the United States are insured. Pet insurance helps offset the costs of diagnosing, treating, and managing pet’s illnesses or injuries. Additionally, pet insurance has been shown to change the types of conversations that pet owners have with their pets; when a client has their pet insured, they tend to think about the quality of care, while when their pet is uninsured, they more often consider the cost of care.

Point of sale (POS) credit:
A growing trend in veterinary practices is Point of Sale (POS) credit. Popular among millennials, POS credit allows consumers to spread out the cost of expensive purchases over multiple months. Services like CareCredit, VetBilling.com, and Scratchpay allow for pet owners to pay for their vet services the same way they would for an expensive purchase, by letting them pay for their vet visits in affordable, monthly installments without the need to open up a credit card.

Wellness plans:
Pet wellness plans, pet owners typically pay a discounted monthly fee to receive a “bucket of services” for their pet. Wellness plans help pet owners afford the care that their pet needs, showing an increase in compliance for vaccine services, and for both In Plan and Out of Plan service recommendations. They have also been shown to help attract new clients, and more importantly, grow client loyalty. Wellness plans help increase the amount a pet owner spends by 3x, while meeting the client’s changing purchasing behavior and desire for monthly spread-out payments.

References:
Brakke Consulting
VMX
WVC
Packaged Facts
AVMA
June 20, 2019, CNBC
Telemedicine: Medical and legal realities for the not-so-distant future
Mark Olcott, DVM

Overview:
The “Dr. Google” phenomenon is very real and based in human nature: we expect instant answers to our questions. In a relatively free market like veterinary medicine (as compared to human medicine) consumers usually get what they want. (Uber, anyone?) This means that telemedicine IS coming to our profession, whether we like it or not. Telemedicine can enhance the “triangle of care” between veterinarian, client, and patient, and we will discuss how to do this ethically and with a focus on patient needs.

During this interactive discussion, the speaker will present the following innovative ideas:
1) Consumers want increased access and “instant gratification” and the internet explosion has only further empowered these expectations.
2) Offering “real-time, virtual consults” to existing clients can be a way to increase client loyalty, expand a practice’s digital storefront, and generate more revenue.
3) The rules for veterinary medicine are entirely different than for human medicine, and we will spend a lot of time talking about what practitioners can (and cannot) do from both a medical and legal standpoint.

Practitioners and managers need to understand the legal and medical implications of this consumer driven trend. Telemedicine will come to veterinary medicine because the consumer wants it. We have the opportunity to shape that future, and we should start talking about it now.

What is telemedicine?
Telemedicine is the use of electronic communications to host virtual consultations. These offerings enhance the “triangle of care” between veterinarian, client, and patient. It’s an emerging technology that can help increase accessibility, revenue, and communications while supporting patient care. Telemedicine can include phone calls, video calls, email, texting, and online or mobile chats.

Why do pet owners want it?
Consulting a veterinarian can involve stressing out a pet, taking time off of work, or visiting an emergency room after hours. As more informative content is published online, pet owners are seeking advice by turning to “Dr. Google,” and as a result, they are purchasing their pet’s medications through other marketplaces rather than their pet’s vet. Instead of leaning on Google, telemedicine in veterinary practices can give owners the ability to speak to their vet in real-time about their pet’s specific situation and get instant guidance on how to move forward. It can provide peace of mind for a pet owner who has been concerned about their pet’s symptoms and wants to get their treatment going as soon as possible. It can also provide reassurance that everything regarding their pet’s health is perfectly fine and no further treatment is needed.

Why should you be learning about it?
Veterinary practices are struggling to keep up with consumer expectations. It’s become quite easy for consumers to get their medicine from places outside of their vet, and practices are losing money as a result. In an increasingly competitive field, veterinarians cannot afford to just “keep up”. You understand the value of using technology as a competitive advantage. By adopting telemedicine technologies, practices can increase client loyalty, expand their digital storefront, and generate more revenue.

How should your practice respond?
Practices should examine their current offerings and evaluate the needs of their existing client base. They should also look at the competition and see what other practices are doing to increase their client retention. By asking the right questions, practices will learn what they can and cannot do from medical and legal standpoints and be able to determine if telemedicine is something they’d like to offer to their clients.
References:
AVMA Practice Advisory Panel’s report on telemedicine, January 2017
Virtual Consultation Services, CVMA
The Technology Adoption Lifecycle-MaRS Discovery District, 2009
Millennials lead on some technology adoption measures, but Boomers and Gen Xers are also heavy adopters, Pew Research Center, May 1, 2018
Balancing high-tech medicine with warmth and compassion
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The James Herriot books have done a lot to promote the beauty of our profession, though some may argue that they, in a way, hurt us a little. There’s no doubt that he often oversimplified our medicine and surgery and led many to believe that we should be able to diagnose a problem just by looking at the patient, and that our recommended treatments should be just as uncomplicated. I’m afraid, however, that many of us and our colleagues have moved to the opposite extreme, becoming almost too technical, too advanced, have too many toys, and have lost sight of why we entered this great profession in the first place. Think about it for a second. What made you choose to become a veterinarian? The money? Hardly! The great hours? Not! I hope most of us would agree that a love for our animal friends was a prime motivating factor. Do you truly love animals? I would hope so, but I know from years of experience and talking with pet companions and parents (owners), that many of us forget to show how much we love our four-legged, scaly and winged creatures. We are actually pretty lucky, because if pediatricians were caught doing to their patients what we are allowed and should be doing to ours, they’d be arrested! Why is it that when I walk into an exam room with a new client and patient, and get down on the floor to smooch with and play with my new friend, the client inevitably says that she’s never seen a veterinarian do that? They usually go on to say that they’d expect to see it—they just haven’t. What happened to all the “animal loving” veterinarians of the world? This is the part of James Herriot that we shouldn’t forget. We shouldn’t forget the compassion, the warmth, and the true appreciation that we should have and display towards our animal friends.

Here’s one; “I became a veterinarian because I enjoy working with animals, but dislike dealing with people.” Oh, that will get you far in this business!! (I actually had an associate who came to me with that revelation just 6 months out of an internship, and is now doing emergency work exclusively.) When our patients become talented enough to bring themselves in to our offices and write their own checks or sign their own credit card slips, then you might do okay with this attitude. Don’t hold your breath! We need to spend more time bonding to our clients as well as to our patients. We are in a terrific position in that when we do our jobs well (and there is no reason why we shouldn’t) we actually receive double the appreciation. Our patients love us (at least most of them) and can’t wait to come and see us again, and our clients adore us for helping their pet companions feel better and stay healthy. It is such a high to get the strokes that we get all day long—something that most people rarely get from their jobs on a day to day basis.

These relationships are not easy to develop. It can take years of work to achieve that level of trust and respect from a client, but once you have, it takes a lot to destroy it. I find that too many veterinarians today spend way too much time on some of the less important issues and not enough trying to develop these lasting bonds. I’ve recognized the importance of cultivating these bonds years ago, and in my practice I’ve become the most popular veterinarian. Why? Am I the least expensive? Not even close—I have the highest per patient average and I generate the highest revenues. Do I spend more time with my clients than my associates? Not necessarily—though I try! My schedule is the most solidly booked, so this is often difficult. I wish I could spend more time with them. Am I the best, most talented, or most accomplished veterinarian in my practice? No way! I have a boarded surgeon in the practice and two other associates who are much sharper than I am. So what is it? It’s simple. I truly love what I do, I adore my patients and my clients (at least most of them) and work hard to develop those lasting bonds, and I remain very committed to all of them. My clients know that I am always there for them and can be reached 24 hours a day. I try to never allow a client to leave my office dissatisfied. Have I made mistakes? Of course I have, but when clients know how sincere you are and how much you really care, they tend to be much more forgiving.

The true elements which lead to success in practice have considerably less to do with medical, surgical, and technical skills than they do with “people” skills! Ouch!! Just how good are we? How good do our clients think we are? The reality is, our clients don’t really know, but their perception is more important than the true reality!!
As long as you don’t really mess up (consistently), you deliver results, and your clients and patients adore you, you are going to do very well as a practitioner. As long as you continually show them that you truly care and that you love their precious pet companions, you will be a winner.

One problem I continually see in practice is that many new graduates and interns really lack confidence! It’s one thing to be new and a little green, but displaying any sense of indecision or a lack of confidence can mean the “kiss of death” for that developing client-doctor relationship. The new graduate or the DVM who has completed an internship program is definitely ready to start meeting the challenges of practice, he or she simply needs to know what they don’t yet know. When you exude confidence, there is no detriment in telling a client that you are not sure about something, as long as you get the correct information to them in a very timely fashion. You definitely need a game plan though, so when you walk into that exam room, you can present your plan to the pet guardian. Depending on the case, it may be totally appropriate to offer more than one plan, (especially if finances are an issue), but even the alternative treatment or diagnostic options should be offered with confidence. The problem I often notice is the veterinarian having very little clue about something, so they basically ask the pet’s parent(s) what they would like to do. “Well, gee,” the client would say, “I thought YOU were the doc!”

Communication is key! I’m sure this is no surprise!! As with any successful relationship, trust and open communication are paramount. Without a doubt, 95% of problems in a hospital setting, be they staff issues, patient care issues, or client issues, result from breakdowns in communication. To prevent these problems, make sure your clients truly understand your game plan, your post-visit instructions, estimated fees, etc. Make sure all your support staff is also very aware and familiar with your hospital cases and that they understand all of the doctor’s orders. And, almost more important than trying to avoid these problems and communication breakdowns, is how you deal with them when they arise. How good of a listener are you? How HUMBLE are you??? How badly do you want to keep the client/patient? Putting these client “fires” out is a true art—one that is certainly worth developing. Mistakes will happen, so the better you are at this skill, the less permanent client relationship damage you will experience. Never challenge a client, especially in the reception area. The last place you want an angry or upset client to be is in the reception area. Once alone with the client, find out from him/her what the problem is, and what, if anything, has been done to rectify it. One valuable (and financially sound) lesson I’ve learned over the years is to let the client do the talking first, and you simply do the listening. Try to practice active listening where you repeat what you’ve just heard, or at least what you think you’ve just heard: “Let me make sure I understand, you’re upset because…….” This allows the client to actually hear what they’ve just said. After listening, sympathize/empathize with your client, let them know how you understand why they are so upset or disappointed (this does not admit guilt or necessary wrongdoing on you or your staff’s part), then let them know how much you value their relationship and how important it is to you that they leave happy or satisfied, and ask what you can do for them to ensure that happens. Let them tell you what will make them happy, don’t start offering them the world. I’ve found that most people are fairly reasonable and will ask for less than you would have been willing to give. Try to resolve all issues! Evaluate the trade-off and determine both the financial and goodwill “worth” to each client’s relationship to your practice. Remember, a dissatisfied client will tell at least 7 people of their negative experience!!

Unless this particular client has a history of being difficult or rude to your staff, it is generally not worth allowing them to leave with unresolved issues. Conversely, when a client sees how much you’ve gone out of your way to make sure they leave happy, the bond they have with your practice actually strengthens.

It’s been a while since I’ve been in school, but seeing many new graduates over the years, I’m a bit concerned over what is been stressed as “important” to our new colleagues. It seems that many graduates are led to believe that unless they join multi-doctor “specialty” group hospitals, their practical skills will suffer. They need this type of practice environment or they won’t become good doctors! Where did this nonsense come from? I guess that means that most of us here today must not be very good doctors!! I think the opposite is more true—that you become a better general practitioner when you work in an environment where you don’t have access to all the high tech “toys,” an environment which forces you to think, reason, get a thorough history, and, using your hands, ears, and head, perform a better physical exam.
This approach also keeps things more personal with your client and patient, which, once again, enhances the bond! I'm not saying that having the great ultrasound machines, lasers, endoscopes, video otoscopes, tonometers, etc. are bad, I just don't think we should rely so heavily on them. After all, not all of our clients can afford to allow us to use them. Besides, I'm not so sure that all of us can afford to buy them in the first place.

Lately, I've begun to wonder whether our schools in their admission processes, or we for that matter in our interview and hiring practices, are actually using the wrong criteria to select the best candidates to enter our wonderful profession. Does a stellar academic record ensure success as a practitioner? Do outstanding clinical skills also guarantee the same? In my opinion, they don't. Furthermore, how important are these for a new graduate? How much should we expect our new grads to know just entering practice for the first time? Even if they had great clinical and academic records, are you going to let them loose with your patients and clients unsupervised? Well I sure wouldn't! And I'm sure most of you wouldn't either. Do I really care about these qualities? Not really. Why? Because these skills can be taught! Some catch on more quickly than others, but basically, anyone able to get through the very challenging academic and clinical programs which our U.S. schools offer, can learn and develop the skills required to become a competent veterinarian. Competent? Most definitely. Successful? Not necessarily!! As I'm sure most of you have learned over the years, success in practice is much more strongly dependent on communication skills, compassion, empathy, confidence, organizational and management skills, and in developing strong relationships with your clients and patients—and, unfortunately, these qualities cannot always be taught!! If someone has the basic personality, these qualities can be perfected and fine-tuned, but personality cannot be taught. It's time our Veterinary colleges and universities take a closer look at who they want representing them in the future!

In conclusion, I encourage all of you to take a good look at yourselves in the mirror and try to become a bit more introspective about why you chose to become veterinarians, then get out some of those old James Herriot books to help you realize what's really important for us as healers and as protectors of that wonderful bond which exists between us and our animal companions. Who knows, you might become even more successful!!
Client (and team member) conflict resolution in the veterinary hospital
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Not many things can ruin our perfect day as fast and as completely as an unhappy client. It's not enough that they sit there and complain and argue, often leaving one of our loyal and usually happy team members in tears, but they insist on doing so smack in the middle of the reception area, during the busiest time of the day, right in front of our other clients! We've all had to deal with these gems, and for me it is one of the most unenjoyable parts of practice. Additionally, Murphy's Law often dictates that these vocal complainers will not be satisfied speaking to anyone but the practice manager or, as often happens in my case, the BOSS (which would be me)!

I've learned many things over the years, and one major lesson I've learned is how important communication is to the smooth, effective, running of a hospital. Without a doubt, most of the problems we have to deal with in practice, be they staff issues, patient care issues, or client issues, result from breakdowns in communication. Good communication is key! I'm sure this is no surprise!! As with any successful relationship, trust and open communication are paramount. To prevent these problems, make sure your clients truly understand your game plan, your post-visit instructions, estimated fees, etc. Make sure all your support staff is also very aware and familiar with your hospital cases and that they understand all of the doctor’s orders. And, almost more important than trying to avoid these problems and communication breakdowns, is how you deal with them when they arise.

This brings up another important lesson I've learned—that conflict resolution, as we like to call it, is definitely an art—one that really needs to be mastered in order to restore peace and harmony into the practice. There are many aspects to effective conflict resolution that need to be learned, yet as with any skill, practice makes perfect. This becomes our proverbial “Catch 22,” in that in order to practice this skill to get good at, means that we have to have a lot of fires to put out—in other words, having more unhappy clients than we’d ever dream of wanting to have. But, in reality, the longer you practice and the more clients you see, the more problems you will have, and, unfortunately, the more opportunity to master this “art.” The ultimate goal is that by the time you finally master this art, you and your staff will be so well trained and in tune with each other that there will be no more communication breakdowns, and therefore no more unhappy clients to practice your mastered art on! (Yeah right—don’t hold your breath!)

Okay, down to business! How good of a listener are you? How HUMBLE are you??? How badly do you want to keep the client/patient? As I mentioned, the art of putting these client “fires” out is certainly worth developing. Let’s face it, no matter how hard we try or how good we are, mistakes will happen, so the better you are at this skill, the less permanent client relationship damage you will experience. One of the basics of conflict resolution is to never challenge a client—especially in the reception area. As I’m sure you can imagine, the last place you want an angry or upset client to be is in your crowded reception room. Instruct your front or technical staff that at the first hint of a client becoming upset or nasty, they should quickly escort that client into an examination room or an office to “better assist and serve them.” Once alone with the client, find out from him/her what the problem is, and what, if anything, has been done to rectify it. One valuable (and financially sound) lesson I’ve learned is to let the client do the talking first, and you simply do the listening. Try to practice active listening where you repeat what you’ve just heard, or at least what you think you’ve just heard: “Let me make sure I understand, Mrs. Smith, you’re upset because……” This allows the client to actually hear what they’ve just said.

After listening, sympathize/empathize with your client, let them know how you understand why they are so upset or disappointed (this does not admit guilt or necessary wrongdoing on you or your staff’s part), then let them know how sorry you are that they are so upset, how much you value their relationship, and how important it is to you that they leave happy or satisfied. At this point it is actually okay to ask what you can do for them to ensure that they do leave happy or, at least, satisfied. Let them tell you what they think would be fair and will make them happy, don’t start offering them the world. I’ve found that most people are fairly reasonable and will ask for less than you would have been willing to give. Try the best you can to resolve all
issues! It's very important to evaluate the trade-off, and determine both the financial and goodwill “worth” to each client’s relationship to your practice. Remember, a dissatisfied client will tell at least 7 people of their negative experience!!

Unless this particular client has a history of being difficult or rude to your staff, it is generally not worth allowing them to leave with unresolved issues. Conversely, when a client sees how much you’ve gone out of your way to make sure they leave happy, the bond they have with your practice actually strengthens.

What about firing a client? This is something that many of us have had to do—though it’s usually not pleasant. When necessary, the first thing I recommend is to prepare a complete copy of the client’s file, including all notes, laboratory work, and radiographs, reports, etc. I then also prepare a list of hospitals in the area to recommend to the client, something which is actually required in some states, and then, after some discussion, present these to the client. What I like to do is to partly take the blame for the problems so the client will leave without any ill feelings. I might say to a client how important it is for me in practice to try and make my clients and my patients happy, and how personally I take it when I fail. I tell the client that when they are unhappy or dissatisfied, nobody wins, and that there is no reason that we should all be feeling as badly as we do. I then tell the client that I truly feel that he or she deserves to be happy, and since it is obvious that we have not been able to achieve that, he or she would probably be better off at another hospital which might be better able to meet their needs. I then hand them the list and let them know that I would make myself completely available to speak with the new doctor if there were any questions about the patient in the future. Two of my clients were so shocked when I did this, they refused to take the records and promised that they would be better with my staff, and not complain so much anymore. To this day, they’ve kept their promise!

Now, what happens when the “conflict” is internal, and involves your own staff members?

I remember when I started my practice over 25 years ago that I was surprised and intrigued by certain employment policies that I heard some of the newer “corporate” practices, as well as many smaller practices had implemented. One policy in particular that, at the time, I found amusing was that employees were not allowed to become romantically “involved” with one another, and it was even discouraged for employees to socialize with each other outside of the hospital. Personally, I found that to be too restrictive, and even wondered about the legal ramifications. Also, since so many of my own employees were already friends who came to me from each other’s recommendations, it would have been very hard for me to enforce a policy stating they couldn’t fraternize with each other outside of the hospital. Plus, I liked the fact that they all got along so well as it kept the spirit around the practice upbeat.

Unfortunately, it didn’t take long for the employee’s inter-personal “like-affairs” to become challenged, and with that the advent of even more issues than I bargained for as a new practice owner. I was amazed just how petty people could be, and how sensitive their fragile egos were. I knew from my first job as an employee veterinarian how I needed to sharpen my skills in the art of conflict resolution to deal with the many client issues and problems which often occur in practice, but I honestly didn’t think that I’d be using those same skills to deal with my employee issues as well.

My practice has now grown to a 4-doctor practice, which is great, but sadly, having 3 occasionally quarreling associates, who have sometimes given new meaning to the term “cat-fight,” I’ve found myself dealing with petty issues between them as well. The truth is, the larger the practice, the larger the staff, hopefully, the larger the revenues, but definitely the larger the headaches!

I’m sure the issues I encounter are nothing new, but for humor’s sake, I’d like to go over some of our frontrunners. As far employees, problems arise around issues of scheduling, responsibilities, advancement, and, of course, raises. Don’t kid yourselves—your employees all know (or have a pretty good idea) of what their co-workers make. I’ve seen many a “friendship” turn sour because of these issues. How often have we taken a kennel person who has shown promise and started to train them to become a technician assistant, and then, depending on how talented they are and how quickly they learn, try them as a technician. Now, all of a sudden, his or her former co-workers become resentful—especially if they think they could do the job even better. Or, simply taking one of the kennel or technical staff and promoting him or her to a manager can create animosity and problems. What’s even worse is having to deal with cultural issues—specifically Latin men suddenly having to answer to a Latin woman!! Don’t ask why, but I’ve seen this create problems many times! When employees are friends outside of work, there are always the personal issues which arise which
can affect the chemistry and teamwork at the practice.

With employee veterinarians, the problems can be just as petty. Probably the most significant issues revolve around cases, schedules, and, of course, salary. How is seniority in a practice determined? Does one consider length of time at the current practice, or the number of years spent as a practicing veterinarian? What criteria do employers use to determine success and advancement potential? Do we use total length of employment as our main gauge, or production and other intangibles such client and employee feedback? The key is, regardless of how these are evaluated, there is a good chance that someone will be unhappy! I am a firm believer in production-based salary, and offer my associates a minimal base, basically to give them some piece of mind and a little sense of security, then offer a production-based bonus. Though this seems to have become a popular compensation program—actually many hospitals have done away with base salaries, and only offer production-based compensation—there seem to be some inherent problems associated with this. What I’ve witnessed are doctors selecting cases based on ultimate earning potential, instead of priority. So if two clients are put into exam rooms at the same time, one, a first time client with a healthy puppy, who came in first and who should be seen first, and the other an older dog with a 3 day history of vomiting and diarrhea, which will obviously need a significant work up, and the next available doctor looks at both files and rushes in to take the second client before the first, leaving the easy new puppy exam to the other associate. I HATE that!! But, it does, so I understand, happen everywhere.

Whether your problems are identical, or similar, they all really disrupt the smooth and efficient functioning of our practices—which often leads to much frustration with other team members, and, of course, with our clients. First and foremost, our team members, including our doctors, need to grow up. Our patients, our clients, and our hospitals need to be the number one priority. Any baggage and petty, nonsensical differences or arguments between employees need to be left at home—and this needs to be a firm policy and understood by all! Obviously, resolution is the ultimate goal, and the principles of resolving these conflicts are the same as any others, and start with—you got it—communication.

When faced these issues, try to attack them before they explode, and probably best to do so in the staff meeting forum keeping the situation very general. Try to divert attention away from the involved parties and handle the situation in more broad terms. Trust me, the involved culprits know who they are, and this subtle approach is often all that is needed to get things back on track. If, or when, this doesn’t seem to take care of the problem, a more private meeting with the involved individuals is in order.

The involved individuals need to be confronted and need to be aware of the consequences of not cooperating and behaving in a manner consistent with the professionalism required of hospital team members. They must work out their issues and get back to productive work, or need to learn to keep their differences at home and behave as adults while in the office. We’ve all had our days when things aren’t going right and we feel emotionally miserable, yet we can’t share those emotions with our staff or our clients. Our employees need to develop those same skills for the benefit of the practice, and we need to help them whenever possible.

So, what do I now think of those crazy policies prohibiting team members from dating each other or socializing outside of the practice? Well, they may not be so crazy after all! But, they are too restrictive for my liking. I have a great staff with a lot of energy, and who, for the most part, get along great with each other, and do enjoy each other’s company after hours as well. Though, over the years, some have had issues to deal with, which could have been disruptive, they’ve always seemed to work things out without causing any friction within the practice. For me, the positive effects of having a crew that truly like and respect one another, and work very well together as a team, outweigh the negatives which may occur from an occasional discord.

What will work best for your practice? The answer has a lot to do with your current staff, the types of problems you are currently experiencing, their existing inter-personal relationships, and their willingness to behave in such a manner which puts the interests of the hospital first and foremost.
Own the exam room: The basics of a great appointment
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What is the most profitable room in a veterinary hospital? The surgical suite—as surgeries are probably the most expensive single services? What about the special procedures room? Echocardiograms, advanced ultrasound studies, and endoscopies are not cheap! I would argue, however, the real winner is the examination room. Most of the services, and thus dollars, you bill out are generated in the examination rooms. So, one’s ability to “sell” in the exam room ultimately determines the success of a practice.

There are a number of “givens” when considering potential for practice success, but also, of course, a number of “intangibles!” Having graduated from our programs, all of us here today are extremely intelligent and talented. We all began our careers on a somewhat level “playing field.” So, why is it, years later, a number of us are extremely successful and loving what we do, while others are experiencing feelings of disenchantment? I believe that those who have mastered the exam room are able to sell the services that will, of course, help their patients, and will bring them practice satisfaction and success! Currently, the industry standard is that only 60% of our clients will act on our recommendations. Our goal is to increase this to 90%!

Firstly, confidence is key! Walking into that examination room without it will often spell disaster! Our clients need to see that we are there for THEM and for the welfare of their precious PETS. They need to see that we truly CARE. In fact, clients don’t’ really care how much we know, until they know how much we care! When a procedure or test needs to be performed, we must clearly send that message to our clients. Be careful with the language we choose to use. Don’t say “this is what I’d like to do,” or “this is what I think we should do,” but rather, “this is what we need to do for Buffy.” Our focus should always be what is best for the pet.

As with anything we’ve learned either in school or in practice, mastering certain elements of practice is an art. Mastering the examination room is no different. There are many elements to this mastery—and it will require a multi-faceted approach.

Firstly, you need to truly identify your own personality. Shy? Introverted? A natural “people person?” Do you typically speak clearly? Are you a good listener, and do you know how to “actively” listen? How well do you know your clients? Realize that your “style” for one client may be totally different for another. Female clients are very different than male clients. Women expect more sensitivity and professionalism. Try to exceed their expectations. Men won’t ask as many questions, are more apt to want to talk about themselves and their pets, and seem to bury their feelings. How formal is your practice—the tone is set by you, and sometimes even by your clients. Personally, I’m the casual type while I’m at work—I’m most comfortable wearing Khakis and a golf shirt, which for me is perfect since I spend most of my time on the floor with my patients crawling all over me. The nice slacks, button down shirt and tie, or the white coat don’t work for me.

When you walk into an exam room, especially with a first-time client, walk in with purpose. Make eye contact with your client, and make sure to introduce yourself to a new client the way you would like to be addressed. To my clients I am “Dr. Jeff” or just “Jeff” as I like to keep it rather informal. Next, address the pet affectionately! If it’s a small dog or cat, pick it up and pet it (unless, of course, it’s aggressive and hissing or growling at you). If it’s a larger dog, bend down and pet it. I will usually get down on the floor and start playing with the dog. Clients LOVE to see this, and quite sadly, too many of our colleagues DON’T do this!!! Say something nice about the pet - “oh, she’s so gorgeous,” or “what a sweetie,” or “what a beautiful coat,” etc. If the pet is nervous, try spending a little time calming it first before starting your exam. Do NOT rush through this—the client should feel that she and her pet are the MOST important to you at this moment!

Next—a thorough history. Let the client talk, and listen with interest and intent. When appropriate, ask questions for clarification. This also confirms to the client that you’ve been listening. When ready, proceed to your examination. Don’t rush straight to the presenting problem! Even if the problem is a limp of a hind leg, still start your exam at the head and work back. I actually like to save the problem area for last. You’d be amazed how many OTHER problems you’ll pick up when doing a thorough exam—some even more serious or concerning than the one the client came in for.
As you are proceeding through the different parts of the exam, comment on what you are finding. If you spend a little extra time ausculting the heart without comment, clients will naturally fear a problem. If everything sounds good, let them know. If you are examining a painful area and you expect a reaction from the pet, say something to both the pet and the client - “sorry cutie, this might hurt for a second,” or “Mrs. Smith, I’m going to be very gentle, but Buffy might feel this.” This shows you care and will eliminate the surprise factor for the client. Also, keep in mind that the “tone” and “style” of your examination may vary depending on the case—is this a new puppy or kitten exam, a basic wellness exam, a sick senior, or a possible euthanasia?

Now it’s time to present your diagnostic and/or treatment plan. What makes us truly great (and loved by our clients) is the ability to offer viable alternatives—always with our patient’s best interest in mind. Not all can afford the “best of the best!” It is, of course, recommended to discuss, and offer the best approach, in fact, you’d be surprised that even the clients that you may not have expected to grant permission to proceed, will do so. Make sure your clients understand what they should expect from treatment. Clear expectations prior to performing a test or a treatment can make the difference between a client who is satisfied and one who is not. Never promise success!! Ask your client if he or she has any questions. Answer them now—this will eliminate problems later. Also, provide estimates and have them approved. Clients dislike financial surprises as much as they do treatment ones. Offer options when appropriate—something clients appreciate. Stress, is that your only motivation in your recommendations is your patient’s (then client’s) best interest—never your own. If a client ever suspects that your motivation is financial, you’ve lost that client forever! I’ve noticed that when given a choice to opt for “plan B,” a less expensive option, clients are much more inclined to allow you to proceed to the “A” plan if “B” fails. If you don’t even offer a “plan B,” and they find out later that a less expensive alternate plan was available, again, you’re toast! I am not impressed by the doctor who can run every test in the book and gives me the right answer—I am impressed by the one who can’t run every test, but still gives me the right answer! Use your heads! Take the information from your history and physical, then reason, think, and analyze, and come up with a fair, reasonable “game plan.”

Make sure your exam room is clean, presentable, well equipped, inviting, and comfortable. The tone of your office and staff should match the appropriate sentiment of the case at hand. You, of course, want to have that cheery staff when a client is coming in with that new puppy or kitten, or that new rescued pet. But if your client is bringing a pet in for a probable euthanasia, you don’t want that same happy tone.

Remember, follow-ups! When I follow up with clients, be it post-surgically, after a hospital stay, or even after a new pet exam, I am amazed at how impressed they are much. Many have told me they wish their own physicians exhibited that much care and concern.

You now have the tools to “Master” the exam room. Practice what you need to, make any necessary changes in your style or approach, and start having fun! This will allow you to promote and sell the services you’ve been trained to offer, will improve your patients’ health should endear your clients to you. This will all bring you both practice and financial success.
The art of case management: How to work within clients’ budgets
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Many of you have mastered certain techniques aimed at attracting clients into your offices. However, due to current economic trends and an extremely competitive marketplace, new clients are unfortunately not tearing our doors down. During lean times clients seem to be even more demanding. Therefore, we must develop strategies geared at nurturing all of our new and existing client base. What is the point of spending time, effort, and energy trying to attract new clients if you haven't developed methods to keep them there? We need to realize that though our professors and mentors would like us to think otherwise, Veterinary Medicine is NOT a "one size fits all" profession.

What makes us “good” is utilizing information from our histories and physical examinations, deciding on a sound diagnostics plan, and selecting an appropriate treatment plan to successfully help our patients. What makes us “great” is the ability to select those plans even when we don't have the luxury of performing all of the appropriate diagnostics our profession dictates. I've often said that I am not impressed by the doctor who can run every test in the book and give me the right answer—I am, however, impressed by the doctor who can’t run every test, but still gives me the right answer! With economic hardships and client financial constraints seemingly the rule, and not the exception, an individual approach to case management is the key to our success. We need to customize our approach to our cases to meet our clients' financial limitations.

We feel that the 3 most important components to attracting and maintaining a client base is "service, service, and service." And, giving your clients the opportunity to provide the best possible care for their precious pets is the ultimate! Certainly, we don't mean to underplay the import of quality medical care, but the truth is that most clients don't really know how to measure or recognize quality care as long as they see results, but service and fairness is something they can definitely measure and seem to respond to.

Here are some of the sure-fire, successful methods to keep clients loyal and happy, and many of these have little or nothing to do with the actual veterinary care you provide!

1) Ask your clients what they want! It's often difficult to provide clients with a service if you're not sure exactly what service they'd like to be offered. They may want, early drop-offs, late pick-ups, a system for hassle free prescription refills, a particular product, etc. You may never find this out if you don't ask. Provide questionnaires for comments, suggestions, criticisms and recommendations. Often a client will make a passing comment to one of your staff members, so make sure your staff members report back to you!

Oh yes, one more thing--it doesn't help to collect these comments if you don't act upon them. Clients want to know that their voices are being heard.

2) Show your clients that you care. Don't be afraid to display affection to your patients. It really shouldn’t be that difficult for an animal lover to pick up a small dog or cat and hold it or give it a hug, or to kneel down and play or rough-house with a large dog. Clients love to see their veterinarian hug, kiss, or play with their pets. News of this type of behavior definitely travels quickly through your client's circle of friends.

3) Call backs: There is little more appreciated by a client to show that you truly care than a text message or phone call from you or a staff member checking up on their pet. If you call, and your client is not home, leave a message, or better yet, leave it for their pet. Our clients really appreciate these texts and phone calls. Try it--you'll be amazed at the feedback you get.

4) Correspondence: Effective communication is of utmost importance! Communicating with your clients means educating them. Once educated, your clients will be more apt to respond to the special programs you may design in the future. Correspondence may be in the form of an e-mail, monthly online newsletters, postcard mailers, over the counter fliers, or give-aways. Whichever you choose, the goal is to keep your clients informed about trends in veterinary medicine, and changes or additions to your practice and staff.
5) **Be available.** If you offer emergency services, great, but if you don't, at least be available to your clients in their time of need. Make arrangements with a local emergency facility that will work with you. Availability also means promptly returning your clients’ messages or phone calls. Any delays may send the wrong signal that you are either too busy for them or that their pet is not a high priority. This will send your clients the message that you are on the ball and that you truly are concerned about them and their pets.

6) Recent economic fears have definitely taken their toll on veterinary care. Even the most affluent client seems to be more discerning with regards to their pet’s care. More clients are now reviewing estimates with extreme precision, and any are electing to split up procedures rather than taking care of everything “today!” We need to be very careful about how we “sell” our services, and what we promote as essential care. Let’s not send mixed messages to our clients! We shouldn’t promote a test or procedure as essential, if we are too quick to skip it once we find out a client has financial limitations. We need to understand that we all don’t drive Bentleys or Porsches because we can’t afford them. But, we do drive—we’ve actually found our “Chevrolets,” cars that we can afford, and will get us to work and around town. Our clients are no different! They all can’t afford the “Rolls Royce” or the “Bentley” of veterinary care that we would love to offer. Our jobs and responsibilities, if we are really good and want our clients to love us and stay loyal, is to find out what the best care possible we can provide that can still fit our client’s budgets. Not all of our clients can afford the “best,” but should NEVER leave your offices in tears, because they couldn’t afford to take care of their pets. Our jobs are simply to “make it work!” Doing so will bring tremendous satisfaction to your clients and create amazing loyalty. Word will get around.

7) **Custom-tailored target marketing:** As we mentioned earlier, try to stay aware of your clients' demands for various services. This information will prove invaluable when creating a strong detailed internal marketing plan. Based on your clients’ feedback, programs such as dental, grooming and bathing, or deworming specials can be implemented. For example, in September we created the B.B, & B, or Bath, Brush and Back to School special. We offered a free bath with every dental prophy and polish. The response to our special was overwhelming, as we averaged 7 prophy and polish procedures per day during the month of September. The results of our special left us with a remarkable revenue increase through pre-anesthetic lab tests, dental prophys, dental radiographs, extractions, and other ancillary procedures such as ear cleanings, growth removals, vaccinations, etc. Everybody "won" with this promotion--our patients, our clients, and our hospital.. Try to tie-in some sort of special or promotion to celebrated events and holidays. Some examples might be a grooming special on Valentine's Day to keep your patients "Lovingly Cute", or a boarding special over the Christmas Holiday.

Lastly, everyone loves receiving birthday messages! Not only should you be sending your patients messages, but you can impress the heck out of your clients by sending them a message or a card on their birthdays--from their pet(s)!

We are sure that for some of you these case management, marketing and client/patient care strategies may sound great, but are rather overwhelming. These marketing and possible necessary adjustments in care may not be cheap, but will be very effective. Actual marketing projects need not be very elaborate, expensive, or time consuming, but they should always remain focused and look professional. You may want to enlist the help of a marketing specialist to help you develop and implement you marketing plan. These professionals are usually available to consult on a per project basis, and need not be hired full-time. You might have some extremely talented and creative people under your employ who can write well, draw well, develop catchy slogans, and come up with phenomenal marketing and promotional ideas. With regards to fees, service/treatment plans, and payments, be flexible. Turning potential business away may not be to your best interest. There are a number of solutions available to keep your clients, and your practice managers and accountants, satisfied. Remain thoughtful and caring, and be creative if needed!
Declassified: The secrets to building unstoppable self confidence

Sarah J. Wooten, DVM, CVJ

Mind your company

According to Jim Rohn, you are the average of the 5 people you spend the most time with. This equates to the law of averages, in which the result of a situation is the average of all outcomes. Whether we like it or not, we are greatly influenced for good and for bad by our relationships. Our closest relationships affect our motivation, our self esteem, our decisions, and our ability to reach our goals. It is necessary to have both positive encouragers and constructive critics in our inner circles - people who will cheer for us and push us to become better. Try to develop a tribe of 3-4 people that you can share failures and successes with, choose some people who challenge you, who are ahead of where you are - it will inspire you to grow. If your current circle is filled with toxic, demotivated confidence killers, then it is time to think about an upgrade in your social circle.

Mind your media

Consuming the 24 hour fear-based news cycle can sabotage confidence and breed fear and anxiety. In fact, this condition is called “Headline Stress Disorder”, and the American Psychological Association, released a study in 2017 showing that ⅔ of Americans are stressed out about the future of the United States. Screening attention grabbing headlines about terrible things going on around the globe are horrifying and overwhelming and leave one with a sense of powerlessness, yet people have a hard time looking away. How are you supposed to be able to build confidence when you are afraid that the sky is going to fall all the time?

Studies also show that scrolling on social media can lead to imposter syndrome, excessive comparison, and depression. Imposter syndrome is a mental and emotional pattern where a person constantly doubts their own achievements and is marked by a persistent, irrational fear of being found out as a „fraud“. When it comes to sabotaging confidence building efforts, imposter syndrome is a hot mess of harm. Perfectionism and comparison are symptoms of imposter syndrome, and spending excessive time on social media comparing yourself to other people is a
sure fire way to undermine your confidence and depress your mood. Take Facebook off your phone, install a screen time minder, and show love to yourself by getting off the internets.

**Mind your self talk/internal dialogue**

The greatest self confidence saboteur is usually our own inner voice. According to the Four Agreements by Don Miguel Ruiz, inside every person resides several voices, including a victim and a judge. The inner judge judges and harshly ourselves when we fail, and the inner victim feels that failure. We don’t do this to ourselves consciously - usually we have developed a subconscious habit (usually from our childhood) that creates the mental flogging we engage in when we fail. In order to build confidence, you must silence your inner chatter, and screen it before you believe the confabulations that your mind is making up. Is your self talk negative or irrational? Is it true? Does it run through a trauma lens? Would the situation look the same from somebody else’s point of view? What kind of words do you use to describe your reality?

**Mind your meat skeleton**

You’ve got only one body to drive around, and how you treat it affects your confidence and happiness levels. Your confidence will be the highest if your meat skeleton is rested, loved, and well cared for. We are conditioned to look at our bodies and magnify the flaws and ignore the things that are good about our bodies, which translates to a hateful relationship with our meat skeleton. Even something as seemingly innocuous as „I hate the shape of my nose” can undermine confidence. You not only want to feed and care for your meat skeleton like you would care for a friend, you also want to be mindful of how you feel about your meat skeleton and how you talk about your meat skeleton. If you find yourself making derogatory comments about your body, pause and be mindful of what you are saying to your meat skeleton, and change the conversation. If you are about to commence on a new diet or exercise regimen, make sure that your underlying intention comes from a place of love and caring for your body, not punishing or depriving. These may seem like silly differences, but they will make all the difference in the long run for your confidence levels, your stick-to-it-ness with the diet or exercise, and your satisfaction with your progress and success.
According to Amy Cuddy’s research made famous by her Ted Talk, good posture and power poses boost testosterone and lower cortisol levels, the hormones associated with confidence and stress, respectively. Holding a confident pose for 2 minutes can boost mood and confidence, and is a great way to prepare yourself for a stressful or confidence-busting situation.

Mind your mindset

What if you could break all the limiting beliefs that you hold about yourself and who you could be? Veterinary medicine tends to exacerbate self-confidence sabotaging perfectionistic tendencies in highly intelligent, sensitive people. Developing a growth mindset and reframing failure are critical to reaching our potential, and goes hand in hand with higher self confidence. Our profession tends to take a very critical view of failure, which is eroding confidence levels of veterinary professionals. We need to understand that working as a veterinary professional, even performing surgery, is called practice for a reason. Your job skills aren’t something that you have to prove to everyone over and over again, they are changeable abilities that will continue to improve with practice, and that practice includes failure. Develop a view that failure is as valuable a teacher as success, sometimes moreso. Expect that you will fail, and when you do, fail forward. Learn everything you can from the experience, be very gracious and loving to yourself, and move forward.
Mindful Management 101 and 201

Sarah J. Wooten, DVM, CVJ

Just like our patients, humans have evolved a „fight or flight“ response to threats and potential danger. While this response helped our ancestors survive, today, many people are walking around in chronic low grade fight, flight, or freeze and consequently, are behaving very badly.

To clients, vet hospitals can be scary and stressful, and many clients are „fight or flight” triggered even before they walk in our door. By identifying the human fear response, we can successfully navigate the situation in order to reduce client communication barriers in difficult situations.

In a stressful situation, the amygdala, the part of the brain that is involved in the fight or flight response, emotionally hijacks the cortex and sends an emergency signal to the hypothalamus. The hypothalamus activates the sympathetic nervous system and the fight or flight response, oftentimes even before we know what is going on - our neural wiring is so quick and efficient that the fight or flight response can kick in even before the visual cortex is done decoding what it just saw.

Common Fight or Flight Triggers:

- Unpredictability or sudden change
- Transition from one setting/activity to another
- Loss of control
- Feelings of vulnerability or rejection
- Confrontation, authority, or limit setting
- Shame or embarrassment

If your client is behaving badly, then there is a high likelihood that he or she is (unknowingly) in a fight or flight reaction, and will need support and care. We must remember that we serve people in crisis.

Signs of Fight Reactions in Humans:

- Aggressively argues, attacked, and debates
- Raised voice
- Tries to silence others
- Tries to win at any cost
- Interrupts
- Arrogant or condescending behavior
- Dismisses comments by others
- Explodes and directs feelings onto others
- Intimidating behavior
- Sarcastic or belittling comments
- Criticizes or accuses others
• Bullies others into submission
• Turns words against you

Signs of Flight Reactions in Humans:

• Shuts down
• Disengages
• Uses humor or jokes
• Quickly changes subject
• Pretends to agree to avoid conflict
• Uses crying to distract and not engage
• Gets defensive
• Becomes overly guarded or withdraws
• Ignores, minimizes, downplays, or avoids issues
• Tries to smooth over conflict
• Placates to keep things under control

Signs of Freeze Reactions in Humans:

• Blanks out, forgets what wanted to say or do
• Zones out
• Feels frozen
• Doesn’t respond
• Overly anxious and scared
• Stares at phone

Best Practices in Dealing with Reactive Humans:

1. Acknowledge the difficulty of the situation right away. This establishes empathy and instills loyalty. Show you care. Asking “how are you?” can go a long way. Smiling, using humor when appropriate, and meeting your client where they are will all break down barriers to care and establish trust and rapport. Let the client know that you understand their pet is nervous and that it is natural. I draw parallels to how I look when I visit the dentist, which usually gets the client to laugh.

2. Don’t use big doctory words.

3. When explaining, intersperse „does this make sense?” as you go along to make sure the client maintains the highest understanding and clarity possible.
4. Avoid in depth explanations for some clients, or in emergency situations. Learn to read your client. Some clients want to know everything about their pet’s condition – they will geek out on everything you give them. They thrive on explanations like „your pet has gastroenteritis secondary to dietary indiscretion. We are going to give an injection of maripotant to reduce vomiting and gut inflammation, and then this is an antibiotic called metronidazole that is designed to…on and on.“ Other clients are what I call „going to the mechanic client“ – they don’t care about the disease process, they just want it fixed and not to happen again. They get overwhelmed easily with information. They are happy with „Your pet has diarrhea. Give this and if it doesn’t clear up in 24-28 hours, call me.” Start with basic explanations, and then offer more information if the client seems interested.

5. Maintain eye contact, smile when appropriate, give the client your undivided attention, and use open, relaxed body language.

6. Avoid using adjectives like „kinda” when describing your recommendations, response to therapy, etc. „after surgery, your pet will be kinda groggy” or „I kinda recommend a urinalysis” etc. Using „kinda” can be like „um” – a space filler when you aren’t feeling confident in your communication. Think before you speak, so you can be intentional, clear, and what you say can have the greatest impact.

7. Never assume that the client is not doing his/her best.

8. Avoid convincing a reticent client. They are either going to listen, or they are not. You lay out the facts to the best of your ability – that is your job. If you hit communication roadblocks or „no’s“, if you have a good relationship with the client, you can try this: “Ok. I definitely hear you. I think it would help me to help you and your pet the best if you explain to me your reasons behind your decision.” What the client does with it is their decision, not yours, and if you are too pushy, you can piss them off. When they don’t do what you say, document it. Sometimes, the answer is initially no, but if you show respect to the client, respect their decision, and maintain the relationship, sometimes the answer changes over time to „yes“.

In any challenging situation or conflict, you have a choice on how you respond. Your choice impacts your relationships and quality of life. In this session, attendees will learn about 5 ways to respond to difficult situations with clients and/or staff, make choices about their own responses in conflict situations, and apply their knowledge to real life scenarios. Responses include:

- Reactions that externalize blame. This tends to include reactivity, judgment of others or blaming, complaining, anger, resentment, and/or gossip.
- Reactions that internalize blame. This tends to include doubting and judging yourself, and is based in fear of rejection or failure.
- Waiting before choosing a reaction. This includes objective observation of yourself and others in a situation, curiously, vigilance, and mindfulness about a situation.
- Working from a framework of healthy boundaries. This includes being assertive without aggression, self-awareness, growth, and freedom.
Miriam-Webster defines “capacity” as a) the potential or suitability for holding, storing, or accommodating or b) the maximum amount or number that can be contained or accommodated. This discussion will mostly involve the latter, since most shelters I have ever visited are overcrowded and feeling it. However, when shelters practice good management, they may discover that their capacity for helping their community is actually not being met, and new programs or more animals may reap the benefits. The goal of this lecture is to help shelter veterinarians identify those maximum numbers, and give some strategies for reducing the number of resources needed to help the same number or even a larger number of animals just as effectively.

Every shelter I visit insists that they really are “very different” from other shelters, and in some respects, that is true. Every community is unique, and every shelter has different needs, excesses, scope, reach, and mission, just as every pet owner has different constraints. The main constraints for shelters with missions that involve pet intake and rehoming include: number of animals coming in, housing for those animals, the provision of care, the resources to provide that care, and the community demand for pets.

Some of these factors can be controlled by shelter mission. For example, private rescues can control the number of animals coming in by denying entry to those they do not wish to admit. Municipal animal control organizations may not have this luxury. Large well-appointed shelters can increase or decrease transport from other shelters as needed. Likewise, private rescues with strict adoption policies can also control the “community” demand for pets by preventing adoptions to people they deem unacceptable; conversely municipal animal control agencies may waive fees for stray pets to allow low-income owners to redeem their pets. Well-resources shelters may decrease barriers further by committing to provide free follow-up medical care for some adopted animals.

Other factors cannot be controlled by mission, but are inherent in the process of sheltering animals. These include the provision of humane housing and humane care while in the shelter. The Association of Shelter Veterinarian’s Guidelines for Standards of Animal Care in Shelters document cites the Brambell Commission’s “Five Freedoms,” originally written in 1965 to promote livestock husbandry, as the benchmark for providing humane husbandry of shelter pets. The five freedoms are:

1. **Freedom from Hunger and Thirst**: by ready access to fresh water and a diet to maintain full health and vigor.
2. **Freedom from Discomfort**: by providing an appropriate environment including shelter and a comfortable resting area.
3. **Freedom from Pain, Injury or Disease**: by prevention or rapid diagnosis and treatment
4. **Freedom to Express Normal Behavior**: by providing sufficient space, proper facilities and company of the animal’s own kind.
5. **Freedom from Fear and Distress**: by ensuring conditions and treatment which avoid mental suffering.

To achieve sufficient animal welfare in the shelter setting, all of these freedoms should be addressed in the animal housing, animal feeding, and animal care protocols, and the “ASV Standards” document attempts to specify exactly what is necessary to achieve these freedoms in a small animal confinement setting. Basic references are provided below.

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husbandry in the shelter includes: intake physical examination and preventive care, sufficiently sized primary enclosures, the ability to hide and rest comfortably, provision of fresh water and at least one meal daily, medical and behavioral care as needed, behavioral enrichment through time with humans and/or other animals, and regular follow-up from staff tasked with ensuring the animal is moving as rapidly as possible toward its preferred outcome.

Recent research into cat housing has suggested that the size of housing makes a significant difference on cat stress and incidence of herpes recrudescence. To combat that, many shelters are renovating existing cat housing into double-compartment enclosures and using historical shelter metrics to calculate ideal in-house and yearly population capacity for the size and resources of their shelter. The same is true for animal shelters in the South with large dog populations: lengths of stay have significantly decreased by participating in shelter transport programs, and determining the "right" number of animals to transport can be challenging. Calculating capacity numbers is common practice for shelter consultants, but the equations employed are often straightforward and can be employed by shelter veterinarians without a consult if just a few data points and metrics are known. Animals moving through the shelter follow Little’s Law, which describes the relationship between the number of objects in a supply chain system (L), the time an object spends in the system (λ), and the arrival or departure rate of objects in that system (W) as:

\[ L = \lambda \times W \]

This equation can be used over and over again in the shelter to understand the constraints of space, time, staff, and money. Most basically, the number of animals in the shelter at any given time (L) can be calculated by multiplying the number of animals that enter a shelter over the course of a year or month (λ) by, on average, how long they stay (W). For example, if 90 cats enter a shelter on average each month (3 cats/day), and each stay an average of 30 days, the shelter will need to provide humane housing for 90 cats on a daily basis.

The same equation can be employed in calculating a shelter’s surgical capacity. How many animals can a surgical team provide services for (perhaps best to calculate summer and winter)? Useful information would be: the number of days/week that surgery is performed, the time of the entire procedure (induction to recovery), and the budget for surgical equipment, medication, and overhead. An example of using Little’s Law in this context would be: can we finish 25 cat surgeries if we start at 10am and need to leave by 3pm if it takes an average of 10 minutes to prep and 10 minutes to complete each surgery (including paperwork)? 10 minutes/cat (λ) x 25+1 cats (W) = 260 minutes (L). Divide by 60 min/hr to get hours, and you can be confident that 4.3 hours after 10am (2:20pm) you will be done with surgery.

The surgical budget can be calculated the same way when money replaces time in Little’s Law. For example, if a shelter has a year-long grant of $100,000 for feral cat surgeries, and it costs $45 dollars to spay or neuter a feral cat, do they have enough money to operate on all of the 200 cats/month they are expecting to serve? 200 cats/month is 2400 cats. $45 dollars per cat (λ) x 2400 cats(W) = $108,000 dollars. No, this is $8000 more than the grant will cover.

7 Scarlett, JM, Greenberg M, and Hoshizaki T. Every Nose Counts: Using Metrics in Animal Shelters. Maddie’s Fund, 2017
The number of staff needed to provide care for animals also follows Little’s law. Both the HSUS and NACA cite 15 minutes per animal per day as the minimal benchmark for feeding and housing, with 6 minutes for feeding at 9 minutes for feeding.\(^8\)\(^9\) My personal experience with shelter cleaning has been extremely variable: spot cleaning likely takes far less than this, but adding enrichment (another necessary part of meeting the Five Freedoms) obviously adds more time. Timing “average” animal care activities such as feeding, cleaning, and providing enrichment in individual shelters will be more accurate than relying on an industry standard, but for the purposes of this discussion, let’s go with it. For example, if it takes the average technician 15 minutes to clean each cage and feed each cat, and cleaning in the 60-cat ward must be performed between 7am and 10am (when the shelter opens), how many animal care technicians will be needed to be assigned to this room? 15 minutes (\(\lambda\)) times 60 cages (\(W\)) = 900 minutes. 900 minutes divided by 60min/hour = 15 hours. With 3 hours to perform this task, you will need 5 people to serve this ward.

When calculating housing numbers, using current numbers in Little’s equation merely provides a look at current operations. For example, if a shelter tells me they take in 90 cats every month, and that they have 90 cages always full, I can tell them without using any software 9 (or a calculator) that the average length of stay in the shelter for cats is 30 days (and vice versa). For healthy adult animals, lengths of stay over 14 days are considered “long-term” in the ASV Guidelines, and ideally would be shorter. Why does this matter so much? When cats only stay in the shelter for (for example) 15 days instead of 30, only 45 enclosures are needed to house 45 cats. That is 45 fewer mouths to feed, clean, provide litter, provide enrichment, and find homes for today. What this also means is that if the number of cats can be reduced, tiny cages can be “portalized” to provide better welfare for each cat, and lengths of stay will decline.

Length of stay is an enormous concern in shelters because it directly affects how many animals are in care, how many animals can be helped over time, how staff feel caring for the animals, and has lasting consequences on the animals that need shelter resources. Reducing lengths of stay in shelters is challenging and requires constant attention and a sense of urgency from every team, whether it’s management, behavior, medical, or volunteer. Methods for reducing lengths of stay attack both sides of Little’s Law: intake/outcome, and number of animals in house. Reducing intakes through TNR and managed admissions and reducing adoption barriers are all buzz words at shelter industry conferences right now. Providing humane housing so that animals don’t get sick and “detour” through isolation is extremely important. Solid preventive medicine through intake procedures and rapid isolation and treatment of ill animals keeps healthy animals and ill animals on track. Keeping abreast of the best (“fastest”) shelter medicine protocols improves length of stay for ill animals, such as those with dermatophytosis or respiratory disease. Daily rounds addressing the needs of individual animals allows different shelter departments to work together to keep animals moving rapidly toward adoption (or euthanasia), and helps create a culture of urgency around outcomes.\(^4\) Keeping animal:staff ratios low prevents staff (and veterinary) burn-out and prevents efficiency losses from training new hires. Finally, keeping the shelter within capacity allows shelters to provide their best care and help the largest number of animals their community will support.

CREATURES GREAT AND SMALL: SHELTERING EXOTIC ANIMALS

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Livestock, wildlife, and more traditional companion exotics enter animal shelters across the country every day. Some are surrendered by owners with long medical and behavioral histories, details about diet and preferences for one toy over another. Sometimes they come in after an owner dies, since companion birds in particular may have significant lifespans. Some animals are transported from other shelters with fewer resources to care for them, or with too many to care for. A surprising (to me) number of exotic pets come in stray, including snakes, tortoises, birds, rabbits, and iguanas. Some may be impossible to distinguish from wild animals; some may be dangerous to handle. The largest volume exotic animal intakes to shelters come from two sources: overwhelmed owners who didn’t realize how easily these animals reproduce or how much care they take, and neglect situations, where large numbers of animals are seized from owners who perhaps didn’t realize how overwhelmed they were. Finally, criminal animal abuse charges may result in the seizure of large numbers of fighting cocks.

Many of these exotic animals can be difficult to care for because they are not domesticated in the traditional sense of the word. In fact, these animals may be so exotic that identifying the genus and species may not be trivial. In cases like this, sending pictures to experts or employing google image searches can be extremely useful. Once the species is identified, important characteristics, including the identity of the individual animal, should be ascertained. Identification techniques include scanning for microchips (necks, backs, keels, withers, etc) and looking for tattoos or brands. Photos of stray animals should be posted on social media to help with identification and reunification efforts. These efforts are enhanced when the animal’s color, gender, life stage, and health status can be accurately described. Color and species are usually simple, however, in many bird, reptile, and amphibian species, gender and age may never be known.

Details about the individual animal can then be used to determine what particular intake, pathway planning, and husbandry needs the exotic pet will need in the shelter. Every animal entering the shelter should have a unique ID number and computer record, a photograph, an intake examination by a shelter technician or veterinarian (this may be a visual exam, depending on how handleable the animals is), and assessment of what the best outcome for the animal will be. Some animals will require surgery (rabbits), vaccination (ferrets), and endo- or ectoparasite treatment (routine or as needed). A location in the shelter should be identified, and a plan made for ongoing care.

Many “information sheet” or “care sheet” references exist online; some of these resources are more reliable than others. Info sheets from trusted sources should be used to make shelter specific protocols for the husbandry of that particular animal. Protocols should specify the appropriate primary enclosure, bedding, hiding/resting place, diet, water delivery, temperature (or range), shelter location, and enrichment plan. Ideally, protocols would be in place before intake, but given the number of species of exotic pets out there, it is impractical to have one for every animal. Priority should be given to commonly seen species in that shelter and location, based on intake history.

Husbandry is one of the most important considerations in caring for exotic pets in the shelter, for many reasons. First, the most common medical and behavioral problems encountered in exotic pets relate to deficiencies in their husbandry. Second, husbandry needs of exotic pets are often very different from cats and dogs. And finally, providing good husbandry in the shelter can help staff educate future adopters about the needs of the exotic pet, and set them up for a successful life with their new companion. Husbandry protocols are just as important for rescues and shelters that use foster homes to house exotic pets.

The primary enclosure for an exotic animal should be specified in the protocol, with attention paid to cage materials, cage shape, bedding substrate, air quality, heat, humidity, water, and a place for hiding/resting.
Reptiles in particular can be challenging to house because different species may variably require dry conditions or water deep enough for soaking, a high temperature or room temperature or a gradient, particular spectra of light, and a particular humidity range.\(^7,8\) This equipment can be expensive to buy, but may be sold to the adopter at the time of adoption, or cleaned and used for the next exotic pet intake.

Diet is a significant concern for exotic pets, wildlife and exotics, and poor diet is a major source of husbandry related medical issues, including nutrient deficiencies, poor body condition, poor hair coat, and obesity.\(^7\) Unexpected intake of reptiles, birds, amphibians, or ruminants may necessitate a grocery run, since live mice, fresh fruit, fresh vegetables, species-specific pellets, or hay may be urgently required. Shelter protocols should specify the diets of commonly encountered exotic pet species, and strive to provide the most complete and balanced nutrition available. For example, pelleted diets are preferred over seed medleys for rodents and birds,\(^7,8\) since many will pick and choose fatty sunflower seed and ignore more nutritious elements. However, if an animal is anorexic because the diet is unfamiliar, slow transitions to a healthier meal plan are likely to be more successful.

Keeping enclosures clean is a large part of good husbandry, but care should be taken in the selection of disinfectants. Concentrated beach in particular can be toxic to birds, reptiles, and amphibians when concentrated and sprayed. Bird-safe disinfectants are generally also safe for reptiles and amphibians; protocols should specify which of the shelter’s available disinfectants should be used to clean enclosures, the frequency of spot-cleaning and deep-cleaning, and the safe handling of the animals during this process.\(^1\)

Attention should be paid to the macro-environment in which the exotic pet's primary enclosure is placed. For example, the ASV guidelines specify that predator species should not be housed in the same room as prey species, since this places a significant amount of stress on prey species.\(^1\) The noise of barking dogs, screeching birds and clanging machinery may be disturbing to some animals (rabbits); conversely too quiet of a room may be stressful to others (social birds). Routine daily light and dark cycles should be maintained for all species in the shelter; this is easier when natural light is provided.\(^1\)

Daily enrichment should be provided to all animals in the shelter; this is a necessary part of meeting welfare needs. Enrichment can include sensory, manipulatory, environmental, forage/feeding, social, or training-based interventions.\(^8\) For example, small rodents may appreciate cuddling with volunteers or food puzzles, snakes may appreciate peg boards for climbing, and birds may enjoy water baths or large uncracked nuts. Animals of almost any species can be trained; some thrive on this activity such as social bird species, rats, and ferrets.

The most common medical concerns I see in small mammals, birds, reptiles and other exotics entering the shelter are those related to poor husbandry. These include all sorts of issues ranging from dermatitis, dysecdysis, vitamin deficiency, diarrhea, poor body condition, obesity, etc. Often, a short time with appropriate diet, enclosure, temperature and housing substrate can remedy these issues; others may require more intensive treatment such as vitamin or mineral supplementation, slow introduction to better food, refeeding plans, enucleations, beak and nail trimming, dental trimming, etc. Occasionally conditions may be so bad that euthanasia is warranted- this could include osteomyelitis from severe pododermatitis, bone fractures from metabolic bone disease, or pneumonia secondary to high levels of ammonia.

External parasites are common in small mammals and also birds; identify which are pathogenic and/or zoonotic before treating.\(^7,8\) These can include fleas, ticks, lice, and cuterebra larva. Other skin diseases may be mistaken for parasites, including dermatophytosis, which is zoonotic. Internal parasites are also common, such as coccidia in rabbits, and can have slightly different presentations compared to dogs and cats, such as hepatitis or GI obstruction.\(^7\) Like dogs and cats, fecal analysis or treatment for parasites if pursued should be dependent on clinical signs and shelter resources. Salmonella is commonly shed asymptomatically by both reptiles and birds; care should be taken to inform staff and adopters about zoonotic diseases.\(^8,9\)

Exotic animals involved in collisions with cars may present with fractures and wounds of various types. Distal fractures of legs and wings and shell fractures in turtles may heal with external coaptation; surgical fixation may be recommended for proximal and displaced fractures. This type of procedure is best performed by specialists, but if euthanasia is the alternative and safe anesthetic protocols are used, shelter veterinarians are encouraged to do diligent research and try what they feel comfortable doing. Amputation of legs and wings is uncommon but can be performed with good success in many species.\(^7,8,9\)
Oral disease can be the result of many etiologies, including poor dental occlusion and tooth root issues in herbivores, parasites in birds, fractures in carnivores, and a host of other infectious diseases. As much as possible oral exams should be attempted at intake. Using a dedicated ophthalmoscope attachment or tongue depressors or small dowels can be useful to visualize molars and examine bird mouths without being bitten. Dentistry is a common procedure for exotic pet veterinarians, and special equipment may be needed to address some of these problems; referral is recommended, but some shelter veterinarians have experience and resources.

The most common infectious diseases seen in exotic pets, besides parasites, infect the respiratory tract. Some of the etiologies of small mammals are shared with humans, dogs, cats and other species, such as *Bordetella bronchiseptica, Chlamydia psittaci*, influenza, and rabies. Others are species specific, such as some streptococcus species and herpesviruses. Physical exam and auscultation are often sufficient to diagnose respiratory disease, but radiography may also be useful. For very small animals, dental radiography can be used to take thoracic views. Although PCR and/or culture is recommended when a diagnosis is possibly zoonotic, empirical treatment for the most common pathogens is often successful. Some recommended courses of antibiotics are quite long, so a plan should be made for managing treatment in the shelter vs. foster vs. adopters continuing treatment at home.

As exotic animals age, reproductive, metabolic, and endocrine disorders become more common. Ferrets, for example, are predisposed to adrenal carcinomas, insulinomas, and lymphoma. 50-80% of rabbit does over 4 years of age are reported to have some stage of uterine adenocarcinoma. Egg binding is more common in older birds, possibly related to hypocalcemia or obesity. Ovarian cysts become increasingly common in older guinea pigs, and may lead to inappetence, weight loss, and abdominal haircoat loss from overgrooming. Mammary tumors are especially common in rats, but pituitary tumors, Zymbal’s gland tumors, and lymphoma are also common.

Some of these tumors can be prevented through routine spay/neuter, such as rabbit uterine and rat pituitary tumors. Others may actually increase in incidence due to gonadectomy, as has been postulated with ferret adrenal disease. Rabbit spay/neuter is considered a general practice surgery, and good practice for shelters adopting pet rabbits. Spay/neuter for other small mammals should be dependent upon shelter resources, anesthetic risks, and experience of the shelter veterinary team.

Exotic pets with serious unmanageable and untreatable illnesses should be humanely euthanized as soon as possible once ownership issues have been resolved. Some larger shelters are able to provide more treatments and care for exotic pets, and may agree to take animals on transport. For large pet birds, livestock, reptiles and amphibians, dedicated sanctuary placement may be available. Some jurisdictions have restrictions for pets and backyard game, including ownership of ferrets and roosters. If prohibited animals cannot be transported outside that jurisdiction, euthanasia may be required.

For healthy or treatable/manageable exotic pets, adoption is the ideal outcome. The adoption policy for exotic pets should be similar to that for cats and dogs: a conversational, educational process during which the experience level of the adopter is ascertained and every effort is made to set the adopter up for care success. Expectations for care of the particular animal should be described at length, and if possible, the primary enclosure should be sent home with the pet. The new owner should also be informed of the pertinent public health and legal considerations regarding their new family member. Restrictive, unfriendly adoption policies are no more likely to ensure pet retention than open, conversational practices, and likely increase exotic pet lengths of stay, impacting the shelter’s ability to care for more animals.

Resources:

2. Lafeber Info Sheets: Lafebervet.com
3. PetMD Species Conditions: www.petmd.com/
6. Banfield's Responsible Reptile Selection and Husbandry
   https://www.banfield.com/getmedia/f172a3cd-4bdf-49af-a1ae-0a1d510e127b/Responsible-Reptile-Selection_LR.pdf
Some may wonder at a lecture about canine distemper at a national conference. The incidence of distemper in the owned canine population in the United States is so low that nobody is studying or reporting it. The modified live distemper vaccine developed in the 1930s is so effective that it provides sterilizing immunity, possibly with just one vaccine, possibly within just a few hours of vaccination, to receptive dogs. Distemper virus is more discussed amongst wildlife circles these days, with many papers describing the decimation of wild animal populations, including endangered carnivores.

However, in certain parts of the United States, especially the Southeast and Southwest, distemper virus is a reasonable differential for sick puppies presented to shelters and private practitioners. Dogs may acquire distemper from other dogs, or from wildlife, such as raccoons and weasels. With increasing shelter-to-shelter transportation, both nationally and internationally, cases may be transported out of areas where vets remember that it can be a differential for a large host of symptoms. The long incubation period and subclinical shedding from some dogs can mean that CVIs are signed and dogs shipped or adopted without anyone aware of the possibility of infection. Unlike rabies, the infection is quickly propagated and easily spread: each infected dog can infect up to 10 naïve dogs. Finally, shelters are likely house more juvenile and unvaccinated dogs than any other institution; shelters that fail to vaccinate incoming dogs may propagate this airborne infection.

The spread of the distemper virus through the canine body depends on the animal’s humoral and cell mediated response. When exposed, those with competent antibody levels from vaccination, maternally derived immunity, or previous infection will mount an immediate response, and their antibodies will neutralize the virus before symptoms occur. In naïve dogs able to mount a strong immune response, distemper virus antibodies reach high levels approximately 1-2 weeks after exposure, and the virus may be neutralized before entering mononuclear cells. They may shed the virus, but will be mildly to a-symptomatic, and will not show neurological signs. For naïve dogs with weak immune responses, the virus spreads intracellularly, which leads to a classic presentation of distemper virus and a poor prognosis.

Symptoms of distemper are highly variable, ranging from subclinical, to fever, to respiratory and lymphoid tissue, then epithelial cells of the skin, GI, and urinary tract. Neurological signs caused by viral demyelination can be seen immediately or weeks to months after recovery from GI and respiratory symptoms. Bloodwork is not terribly diagnostic: lymphopenia and thrombocytopenia may be ascribed to stress instead of distemper, and elevated renal or liver values may be minimal or overlooked. Sometimes the first sign that a respiratory outbreak in a shelter is caused by distemper is the death of a puppy with neurological symptoms, or a dog that manifests multiple typical signs, such as both severe ocular and nasal catarrh and hardpad disease.

When suspicions are high in a coughing dog, the vaccination history should be scrutinized, new vaccines given to all unvaccinated exposed dogs, and a canine respiratory disease PCR panel should be performed on a respiratory conjunctival swab, since respiratory symptoms may be caused by multiple pathogens. Especially in multifactorial respiratory disease outbreaks, experts recommend swabbing at least 3-5 affected animals, or

10-30% of the population\(^4\) to get the best idea of which pathogen may be most likely causing the majority of symptoms. In a dog with more stereotypical signs, quantitative canine distemper rtPCR is about half the price of a multi-pathogen panel, and will give an idea of true infection vs expected levels of “vaccine” virus.\(^5\) Once canine distemper has been detected within the population, the vaccine history for all dogs should be scrutinized and addressed, and quantitative rtPCR should be performed on respiratory/conjunctival swabs from all exposed dogs (ie those breathing the same air) in order to best discover the asymptomatic shedding dogs within the population. While the PCR test is running, a conjunctival swab can be collected and stained with dif-quik to look for mucosal inclusion bodies; these can be diagnostic but a negative slide does not rule distemper out. Other tests, such as IFA, paired titers, or CSF antigen detection can be used and are diagnostic, but are far less practical in the animal shelter.

Any dog who tests “positive” or “intermediate” for shedding CDV on quantitative PCR should be isolated. Ideally, this would be in a separate shelter facility with separate staff and completely separate animal husbandry equipment. Treatment of affected dogs should be undertaken, commensurate with shelter resources, and euthanasia employed for those that are suffering and cannot be treated. Treatment includes the use of broad-spectrum antibiotics, nutritional and hydration support, and attentive nursing care. Dogs that develop neurological symptoms likely have a very poor prognosis or an extremely long course of disease and euthanasia should be considered.\(^6\) Once clinical signs have resolved, PCR testing of respiratory swabs or urine is recommended. Since shedding can be highly variable, two negative tests at least 1 week apart are recommended before releasing the dog into the community.\(^7\) This may take from 2 weeks to 6 months.

Exposed dogs who test negative on PCR should be immediately separated from dogs who have not been exposed. Approximately 50-65% of dogs entering shelters present with titers against distemper virus, and are very likely immune to the disease.\(^8\) Drawing blood to test each of these dogs for adequate titer levels is a good use of resources, since daily care in quarantine is expensive and keeps animals from getting adopted, which increases the care burden of (likely already overwhelmed) shelter staff. Bedside titer “combs” are available and are relatively inexpensive. Once resistance to CDV is assessed, PCR negative dogs with high antibody titers can be judged to be low risk and moved out of the quarantine area.\(^9\)

All new respiratory, GI, or neurological symptoms in dogs in the quarantine population should be regarded as suspicious, and PCR testing/conjunctival swabbing performed. Ideally, the dog should be isolated from other quarantine dogs as soon as possible but held back from joining distemper isolation until infection is confirmed by diagnostic testing. In 10-14 days after vaccination, asymptomatic dogs in the quarantine area may have their titers and PCR tested again. If antibody levels are high, and the dogs remain CDV negative, it is likely that they have developed adequate immunity and may be released into the general population. If the titer is still low, revaccination and retesting in 2 weeks is recommended. Quarantining dogs for 6 weeks without testing is actually more expensive than testing, less humane, and not recommended.\(^7\) The best management for distemper virus in both populations and individual dogs is prevention, and the best prevention is vaccinal.\(^6\) The use of modified live canine distemper vaccines at intake provides the herd immunity needed to stop even this very contagious disease. Puppies and unvaccinated dogs will always be a

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\(^7\) UC Davis Koret Shelter Medicine Program: Canine Distemper (CDV) updated 7/9/2010

\(^8\) Litster A, Nichols J, Volpe Prevalence of positive antibody test results for canine parvovirus (CPV) and canine distemper virus (CDV) and response to modified live vaccination against CPV and CDV in dogs entering animal shelters. A. Vet Microbiol. 2012 May 25;157(1-2):86-90

vulnerable part of your population; keeping them away from dogs with symptoms is part of the battle. Early recognition of a dog or dogs with signs of distemper and judicious use of diagnostic testing, both for disease and immunity, can significantly improve that dog’s prognosis and prevent spread throughout your shelter or community population.
Human rabies is found in over 150 countries worldwide, with over 95% of cases in developing nations in Africa and Southeast Asia. Although numbers are slowly decreasing, between 50-60,000 human deaths are attributed to canine variant rabies yearly. Children under 14 years old are overrepresented, making up over 40% of deaths. Young boys are also more at risk than young girls, likely due to patterns of social behavior and animal contact. Human rabies vaccination is expensive, often costing a large proportion of a yearly salary. Response to animal bites is also expensive, especially when governments do not cover the cost. To be successful, treatment must be initiated within a few days after exposure, and transportation and access to medical care may be challenging for rural residents in these countries.

In India in particular, 20-30,000 people died of canine variant rabies yearly during the early and mid-2010s, representing 700 cases in 100,000 or 1/150 people every year. Rabies control programs have been variously employed by state and local governments but there is no national reporting for human cases required, nor is animal surveillance reported. In recent years, shortages of IgG have prevented some people from getting treatment: these have been largely resolved due to new sources. Ambitious programs in concert with the WHO and GARC have aimed at coordinating NGO and governmental activities regarding control and surveillance, and efforts have paid off in some locations, including Goa, which has had no human deaths since 2018.

Studies have long shown that canine rabies control is the key to prevention of human rabies deaths. Control methods have variably included mass culling or killing of specific dogs, “tying out,” where suspect dogs are tethered or caged until they die (or don’t), and most recently, vaccination. Large fluxes and turnover in canine populations can interfere with these endeavors; the more puppies born every year, the more naïve dogs there are in the population. Spay/neuter and “responsible ownership” efforts combined with recurrent mass vaccination campaigns appears to be a compelling solution to this issue. In general, rabies is slow to spread amongst dogs compared to diseases like parvovirus or distemper virus. Calculated vaccination coverage in a stable population may be successful at as low rates as 40%, but with population fluctuation, most organizations are aiming for >60% dogs vaccinated to maintain the 40% buffer year-round.

Combined with efforts to manage dog populations, NGOs have shown that human education about rabies and risk factors is critical. This includes dog bite-prevention, and bite-care education. Providing intradermal PEP for free to those who have been bitten, improving cultural attitudes toward dogs, and the increasing involvement of well-funded NGOs has significantly improved both the vaccination and education efforts.
These organizations and the WHO have launched an ambitious goal of ridding the whole world of canine-variant rabies by 2030.\textsuperscript{5,6}

Historically, leash laws, requiring all dogs to be vaccinated and licensed, and actively collecting stray dogs have been credited with helping the US maintain a large canine population free of rabies. Humans living in the US today are at greatest risk for contracting rabies by contact with dogs when travelling abroad.\textsuperscript{7} With increasing individual and organizational transportation of dogs from countries with canine-variant rabies into the US and other countries without, the risk of acquiring canine-variant rabies within the bounds of the US has increased. Currently, the national controls are fairly lax. A rabies vaccine 4 weeks before entry if older than 12 weeks, notification of arrival, and a certificate of veterinary inspection is required for all countries, and a valid rabies certificate is required for “high-risk” countries. The CDC provides exceptions to these rules on a case-by-case basis. Dogs that are obviously ill may be denied entry.\textsuperscript{8} After several high-profile importations of dogs with inaccurate or adulterated rabies vaccination certificates, the US has temporarily banned the importation of dogs from one country, Egypt.\textsuperscript{9} Controls at the state level are frequently more strict, require additional testing (such as rabies titers), or require additional reporting.

The risk of acquiring rabies from canines in the US is significantly lower than acquiring rabies from cats; of animals submitted for rabies testing in the US, a yearly average of 255 (1.1%) cats were positive, vs 71 (0.3%) dogs between 2012-2016.\textsuperscript{7} In fact, cattle were more likely to be diagnosed as rabid (87, 6.8%) than dogs. For all of the animals, wild carnivore or bat variant rabies were diagnosed. Cats with unknown histories and injured strays make up a large proportion of cats seen by animal shelters. The incubation period for rabies can be several weeks, even in a small animal, the infection may go unknown or unnoticed until after adoption. Shelter controls should include: thorough history taking at intake, even for stray and un-owned animals, inquiries about encounters with wildlife, inquiries about health history, and quarantine holds if concerned. Shelter staff should be vaccinated for rabies and have their titers checked routinely.\textsuperscript{10}

When shelters take in animals with known or suspected bite wounds from wildlife, they should follow the guidelines of the rabies compendium. Euthanasia is recommended for animals without known rabies vaccination, but in the case that shelters elect to quarantine, vaccination should be given and dogs and cats quarantined for 4 months. For shelters that treat injured exotic pets and livestock, recommended post-exposure quarantine period is 6 months. In the event that aggression or neurological symptoms are noted, the animal should then be euthanized. This type of quarantine should be distinguished from a rabies “bite hold,” for which shelters are also routinely used by local and municipal health departments. When a domestic animal bites a person, this aggression could be the first symptom of a rabies virus infection, and salivary contact with blood could transmit the infection to that person. These animals should be quarantined for 10 days, since it usually takes 5-7 days for clinical animals to die. For shelters that handle injured wildlife, particular care should be taken when dealing with rabies vectors such as raccoons, skunks, foxes, and bats; some jurisdictions prohibit rehabilitation of wildlife species and in those situations, euthanasia is recommended.\textsuperscript{11}

\textsuperscript{5} Global Alliance for Rabies Control. https://rabiesalliance.org/
\textsuperscript{7} Rabies Surveillance in the United States during 2017. Xiaoyue et al JAVMA Dec 15 2018
SHELTER ANIMAL TRANSPORT: WHAT ARE WE DOING WRONG?

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One of the most powerful tools in preventing euthanasia of animals in shelters in the last decade has been a mass relocation of dogs and cats from areas of excess to areas of demand. Transport increases variety of ages and types of dogs and cats available for adoption at destination shelters, and allows shelters with limited resources to take advantage of the medical and financial resources of partner organizations. On a local level, transport may be arranged for a cat needing dental care from a shelter without a veterinarian to one with a vet on staff, from a rural shelter experiencing a hoarding seizure of a large number of kittens to an urban shelter with consistent kitten adoptions, or a young dog with behavioral challenges to a shelter with a certified behaviorist on staff. On a larger scale, source shelters with high numbers of canine (and increasingly, feline) intakes in the southern US often transport to destination shelters in the northern US, where companion animal intakes have dropped significantly in the last 2 decades, likely due to spay/neuter efforts, more resources, and cultural emphasis on responsible ownership.\(^1\) International transportation has been increasing on both a person-to-person and institutional level, with little to no oversight or guidance. This practice offers new hope for individual animals but poses distinct public health challenges.\(^2\)

Several models of transportation exist, including one-time direct shelter-to-shelter (or rescue-to-rescue) transfer, ongoing direct shelter-to-shelter transfer, multiple shelters to one shelter, one shelter to multiple shelters, shelter to individual, and a hub-and-spoke model, where several shelters send animals to one location, and then those animals move out to multiple destination shelters. There are no federal requirements for reporting transports, but the practice appears to be growing. The ~3000 rescue and shelter organizations voluntarily participating in Shelter Animals Count in 2016 reported 180,000 cats and 340,000 dogs taken in through transfers from other organizations in 2016; in 2018 they reported 240,000 cats and 380,000 dogs.\(^3\) Some shelters rely on transportation for the bulk of their animal intakes: for example, Oregon Humane Society took in more than 70% of the ~11,000 they adopted out in 2018 via transport, up from 55% in 2016. Likewise, at the Madera County Animal Services in Madera, California (a transport partner of OHS), ~60% of the 4300 animals that left that shelter alive in 2018 (85% of total intakes) did so through transportation programs. In 2014, before this partnership began, they euthanized 60% of all incoming animals.

Transport programs are most successful when relocation not only helps individual animals but acts as pressure release for overcrowded shelters, allowing them to spend limited resources on the animals left behind. Decreasing animal populations in source shelters not only prevents euthanasia, but can allow staff to implement better management practices and provide more local services, including spay/neuter and other preventive care. Sustainability, namely creating enough flow-through and capacity for source shelters so they can eventually take care of their incoming populations locally is often a goal of transport programs.

Of course, transportation has significant risks. One of the biggest is the introduction of infectious diseases into the population of the destination shelters. Diseases of major concern include respiratory diseases (discussed in the next lecture) such as canine distemper, feline calicivirus, Streptococcus equi subsp zoopneumonia, parainfluenza, pneumovirus, influenza, and others. Infectious diarrheal disease can also spread from transport dogs, including parvovirus, canine distemper, as well as GI parasites such as coccidia, giardia and trichomonas. Infectious dermatological diseases of concern include dermatophytosis.

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Heartworm disease incidence has increased in the northern US since Hurricane Katrina, likely due to animal relocation (with and without owners) since 2005 and warming climate. Unfamiliar regional diseases may cause diagnostic dilemmas for destination veterinarians, including *Ehrlichia canis*, Chagas disease, Coccidiomycosis, Salmon poisoning, *Brucella canis*, and Babesiosis, to name a few.

Transportation can illuminate other problems besides breaches in biosecurity, including failures of communication and unclear, unmet expectations. For destination shelters, receiving animals that are ill, behaviorally challenging, or otherwise difficult to place can soon overwhelm adoption programs counting on shorter lengths of stay. For source shelters, transporting all of the most adoptable animals may leave behind a population that is predominantly ill, behaviorally challenged, or otherwise difficult to place. The cost of transport can be high and source shelters may be saddled with this burden; destination shelters may feel extorted when asked to pay per animal. One of the most unpalatable parts of transport may be shelters who rely on “cull lists” to advertise to rescue partners, with which it can seem like animals only get rescued when euthanasia is threatened, and if euthanasia threats are not followed through, rescue partners feel no urgency.

Finally, transportation may be dangerous for the animals being transported. Several incidences of animal deaths during transport have been reported, including an incident involving 20 animals on an ASPCA relocation truck in 2019, and 11 dogs from carbon monoxide poisoning in Mississippi in 2018. Animals may break out of their crates during transport; incidences of dogs breaking into cat carriers during transport and killing them has also been reported. For the majority of animals, the stress of transport is the most dangerous part of the drive. This stress can be significant, and may increase susceptibility to infection or increase viral shedding. Transported cattle, for example, are known to present with mycoplasma pneumonia post-shipping; although not described in companion animal literature, many shelters complain of post-transport respiratory disease in dogs positive for mycoplasma on respiratory PCR tests.

In order to mitigate many of these problems, several organizations have come up with transportation guidelines for relocating dogs and cats. These include the 2019 Association of Animal Welfare Advancement’s Companion Animal Transport Program Best Practices, and the 2014 American Veterinary Medical Association’s Relocation of Dogs and Cats for Adoption. For example, both documents provide extensive recommendations for transport vehicle maintenance and operation. Both recommend that all animals crossing state lines have a valid CVI. Both recommend animals receive core vaccines including modified-live distemper and rabies, and receive a physical examination within 24 hours of transport. Transported animals should be treated for internal and external parasites; at minimum against hookworms and roundworms. Both organizations recommend dogs over 6 months old be tested for heartworm and started on preventive. Both recommend dogs receive behavior evaluation or screening, and both recommend animals wait at least 48 hours after sterilization surgery before relocation. Finally, both recommend following specific state and local regulations, including, for example, leptospirosis vaccination for dogs entering Michigan.

Other practice specific organizations have also created documents that recommend medical and behavioral interventions before and after transport. These include the 2010 Association of Shelter Veterinarians’ Guidelines for Standards of Care in Shelters, which outlines basic responsibilities of source shelters, transporting parties, and destination shelters. In partnership with the ASV, the American Heartworm Association published a guide to Minimizing Heartworm Transmission in Relocated Dogs in 2017. This document outlines minimal practices for source shelters, such as testing all dogs for heartworm and

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application of macrocyclic lactone preventive drugs and treatment if possible; if transportation cannot be delayed, dogs should be started on doxycycline. Receiving shelters should treat the dog for heartworm disease as soon as possible. Both documents recommend that source and destination shelters work together with a veterinarian to create preventive medical protocols for animals scheduled for transport.

In a 2016 article in the journal *animals*, Simmons and Hoffman conducted a survey of almost 50 shelters and rescues organizations participating in animal relocation to understand the logistics, dog selection factors, medical requirements, nature of shelter partnerships, and perceptions of long-distance dog transport programs at organizations across the US. They found that most destination organizations transported >100 dogs/year from around 1-5 partners, and around half contracted with independent transport companies for moving animals, especially in the NE US. Regarding medical care, intake “quarantine periods” were employed by 64% of the organizations, with a varying range of 2-14 days. Only 76% required dogs to undergo preventive medical treatments before transport; these requirements varied widely. For example, 57.5% of sampled organizations required rabies vaccination and 53.9% DA2PP vaccination before transport, while only 35.2% required heartworm testing, and 14.5% required treatment for fleas or ticks.

International transport shares many of the same risks with domestic transportation of companion animals, and like domestic transport, the lack of a systematic approach for monitoring means that it is difficult to quantify the risk of disease and other harms posed by this practice. The CDC reported that around 300,000 dogs were imported in 2006, but it was unclear how many of these were owned. A Canadian report from 2014 showed that approximately 1/5 of rescue organizations in that country imported dogs from overseas. Social media attention to international and extra-territorial adoptions increased significantly after the 2014 Sochi Olympic Games and Hurricane Maria in 2017. All of these factors suggest that the number of international companion animal rescue imports is high and growing.

In order to import dogs into the US, the CDC requires a certificate of rabies vaccination at least 4 weeks before travel from high canine-variant rabies risk countries. Due to particular recurring problems, dogs from Egypt or originating in Egypt but imported through a third country are banned from entering the US except with CDC written approval in advance. Certain jurisdictions may also require a USDA-APHIS CVI to travel from the airport into the destination city or into another state. Since these regulations are fairly lax, many shelters find importing dogs from outside the country straightforward, many owners easily buy pure-bred puppies from on-line international dealers, and many international sources target the US for exports.

The motivation to transport from crowded severely underfunded shelters in countries where animal welfare legislation is weak is clear. The risk is also clear: at least 3 dogs with canine variant rabies have been imported into the US in the last 15 years. The outbreak of H3N2 influenza in Chicago in 2015 was likely the result of importation of dogs from Asia (whether owned or rescue-bound is unknown). Different strains of more common diseases, such as canine distemper, could potentially affect not only companion animals but spread into local wildlife. Babesia, heartworm disease, and leishmania have increased their range into northern Europe due to infected dogs imported from other regions; this could easily happen in the US as well. A further concern for international transport is the current focus by many rescue groups on individual animals with dramatic stories, rather than a focus on helping a particular shelter or community, or a commitment to improving welfare for international companion animals long-term.

State or federal regulations on rescue-import are unlikely to prevent introduction of diseases from distant locations, since owners and “owners” will likely be exempt. But they may shut down or hinder life-saving efforts. In order to be able to continue to decrease euthanasia of companion animals both here and abroad, it

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is up to the shelters and rescue groups themselves to adopt responsible policies that minimize disease risk and animal harm. To accomplish this, source and destination organizations will need to collaborate to identify the most important risks and create transport protocols that apply appropriate preventive measures. Destination shelters should be prepared to help fund measures such as purchasing vaccinations or paying for testing when needed, and should visit the source shelter regularly. All relocating animals should be carefully screened by trained staff within 24 hours of transport, and removed from the roster if any concerns are noted. When ill dogs are being transported specifically to receive care at a destination shelter, all parties must be in agreement. All medical, behavioral, and travel records must accompany the animal to its destination, and must be disclosed to adopters.

If further disease outbreaks or public health breaches occur, regulatory bans and rules will be enacted that will prevent reasonable life-saving transportation efforts; this is already happening on the state level; for example, the NY state vet has recently banned the importation of known heartworm positive dogs. Additional research, including quantifying the number of animals relocated and from where, could help put these very real risks into a larger context, but responsible actions by shelters, especially careful health screening and comprehensive preventive health measures, taken both before and after transport, are the key to safeguarding this life-saving endeavor. As stated by the AAWA: “It is our responsibility to be vigilant, guard against abuses, and model quality transport to ensure continued access to this valuable tool that saves animal lives.”
Although many shelters have plenty of cats, some shelters in some areas of the US are experiencing shortages or significant reductions of kitten populations. As the average age of shelter cats gets older and more shelters try to adopt out animals with chronic health problems, standard operating procedures [SOPs] regarding the most common of these diseases are strongly recommended. SOPs should be based on evidence and science, with the underlying mission of the shelter and the goal of improving animal welfare as major influencing factors. For shelters with modest resources, average times to adoption and average costs of care may play a significant role in dictating policy towards particular diseases and conditions. Cats with “behavior problems” and “health problems,” even when relatively young, are likely to stay in the shelter for the longest lengths of time. When available, past performance on adoption of these cats should be accessed to determine what your community considers a “reasonable” shelter length of stay and a “reasonable” amount of veterinary time/dollar investment. In addition to welfare concerns for these cats when living in inadequate primary enclosure environments, volunteers and staff interact with these pets day after day and become very strongly attached. Showing and making these animals available and then reversing that decision may be particularly difficult in some shelters, and delicacy in communication is often required to satisfy multiple stakeholders. Pre-emptive education, clear communication by the medical team about SOPs and cat prognoses, and a united message by shelter management can significantly decrease rancor.

Alternatives to in-shelter care or euthanasia may be available for some cases, including foster based hospice care (aka “fospice”) or hospice adoptions. Clear guidelines detailing financial, time, and care decision responsibilities must be discussed up front with caretakers and shelter management. For some chronic disease cats, transport to a better resourced shelter may be possible, however, communication about outcomes between shelters should be very clear: animals should not be transported if euthanasia is likely to be the outcome at the receiving shelter.

The following is a brief discussion about the diagnosis, treatment, daily care and welfare expectations of cats with particular disease processes in the shelter, with the goal of helping shelter veterinarians construct SOPs for these conditions based on the most current knowledge and thinking in conjunction with expectations from their communities and shelter resources.

**Feline Leukemia Virus**
Our models of feline leukemia virus and how to diagnose this disease are being significantly called into question by current research and long-term follow up studies. Outcomes for cats with FeLV have a wide range- from ill kittens dying acutely of their disease to cats who abort the infection, or those who live full lives while persistently infected only to die of other age-related causes, to middle-aged cats with latent disease who experience a sudden unexpected recrudescence. Testing has two aims- first, to identify animals with persistent disease in order to isolate them from other cats and thus prevent transmission, and second, to help owners and potential adopters understand what the quality of life and life-expectancy of their cat might be. Negative tests are not guarantees of absence of infection, since some cats may harbor the virus in the bone marrow but be aviremic. New recommendations for shelters have included not testing every cat on arrival; benefits to this policy include not spending huge pools of money on SNAP testing, especially when cats are routinely housed in single cat or single litter enclosures. For cats housed in group situations, testing before introduction is still preferred. Draw backs to this policy include managing adopter and veterinarian expectations, and decision making about who to test. Having an option where new adopters can pay for the test at the time of adoption requires staff who can draw blood and accurately interpret tests to be available during all adoption hours. Alternatively, adopters can be referred to their vet for the test at their first visit; one problem with this policy is that owners become attached very quickly and positive test results may create a lot of drama; furthermore, the new adopter may have already mixed this cat with naïve cats in their home. When a cat tests positive, confirmation is needed. Using FeLV PCR is now recommended in lieu of a second snap
test or IFA in Australia.¹ These and other new recommendations on the management of FeLV in kittens and adult cats should be coming soon from the AAFP- policies should be updated accordingly when the new document is released.

Feline Immunodeficiency Virus
According to published studies, FIV transmission is extremely rare among household cats when all are neutered, and transmission of FIV from queen to kitten is also extremely rare.² Shelters and practitioners routinely use a snap test at admission or first vet visit to determine the FIV status of every cat, but this can be problematic. Firstly, healthy female cats and kittens are extremely unlikely to be positive, meaning that the positive predictive value of this test for these groups is fairly low. Follow-up tests are needed to confirm. Secondly, management of these cats in the shelter or home may not change as a result of the test. Like FeLV, new recommendations for shelters include not testing every cat, but unlike FeLV, the disease is much less likely to be problematic in multi-cat household adoptions. Rational FIV testing policies could include testing all cats going into group housing, testing intact adult males, or testing adult intact males with any signs of wounds or scars. Vets should also know that prevalence of FIV may be very different in other countries, and that vaccination is commonplace in Australia and New Zealand, so testing imported cats may be recommended. For cats that unexpectedly test positive, new recommendations include using a different brand of snap test, using the FIV PCR in lieu of Western Blot, and in rare cases of discordant results, using virus isolation.¹

Chronic kidney disease
Chronic non-congenital kidney disease is the most common cause of death of older cats. CKD is found in 30-50% of cats over 15 years old and starts in some cats as early as 6 years old.³ Every shelter is impacted by cats with renal disease. Because progression is not necessarily linear, and because cats may have different levels of impact to their welfare at different stages, many shelters struggle to know if and when euthanasia is appropriate. Nevertheless, nephron damage is usually irreversible and progressive. Recommendations for management by shelters includes creating a diagnostic rubric based on IRIS staging and the individual cat’s welfare in light of current symptoms. SDMA testing and biochemistry panels can be useful, but USG and azo-stick can provide a rapid, inexpensive diagnosis. Policies regarding diagnosis should reflect the mission and resources of each shelter, and treatment/diet options that are likely to be available to the average owner should be employed by the shelter. For example, cats that require daily SQ fluids, phosphate binders and a special diet are unlikely to be good adoption candidates in most communities. For shelters that are willing to adopt out otherwise healthy IRIS stage I and II cats, monitoring of cats on the adoption floor is extremely important, since these cats are likely to be long-term shelter stays and their disease may progress to stage III or IV rapidly. Considerations should be made for behavior and other health conditions (dentistry, heart disease, etc) which might further impact welfare and the ability to treat kidney disease if needed. Median survival for cats in IRIS stages at the time of diagnosis in one study were: Stage Iib median 1,151 days (range 2-3,107), stage III had a median 778 (range 22-2,100) and stage IV was a median 103, (range 1-1,920.)⁴

Feline Hypothyroidism

Hyperthyroidism in cats has been recently linked to exposure to flame retardant chemicals, and is diagnosed in about 10% of cats over the age of 9 years old. In the shelter, these cats are harder to recognize because common symptoms are similar to other diseases (PU/PD, weight loss) and simple bedside testing is not available. Suspect cats may not always present with an obvious thyroid slip. Sending out for total or free T4 on thin older cats with ravenous appetites who might otherwise be good adoption candidates is recommended when shelter resources allow. Management is also complicated, since treatment of the disease can reveal previously masked kidney failure, and excess thyroid hormone effects on the RAAS system, which can lead to hypertension and permanent damage to the heart. To date, there is no test that can successfully predict whether cats will become azotemic following treatment. Instead, biochemical bloodwork looking for renal disease is recommended after an initial four-week course of treatment with methimazole. Although this medication is not particularly expensive, housing a geriatric animal in the shelter for a month just to decide to euthanize it after post-treatment tests reveal kidney disease (which happens in 15-49% of diagnoses) might be considered a poor use of shelter resources and more importantly, less than great welfare for the cat. Sending the cat to foster on treatment for a month only to euthanize it after testing takes an especially resilient foster parent if you want to maintain their relationship with the shelter. This is why many shelters euthanize hyperthyroid cats after diagnosis. Those that attempt to adopt these cats out after the month-long treatment trial have the option of adopting on daily methimazole, performing thyroidectomy, or, in far more rare circumstances, sending the cat for radioiodine treatment. Thyroidectomies are simple to perform (especially intracapsular method which preserves the parathyroid glands) but have the possibility of severe side effects if the parathyroid glands are damaged or hypothyroidism if both glands are taken, as well as a higher rate of hyperthyroidism recurrence than iodine 131 radiation treatment. However, in cats who are resistant to daily medication, this technique may provide a new owner relief for many years, possibly for life. Thyroidectomized cats may also have a shorter length of stay, since being on daily medication can be a barrier to adoption. For shelters wanting a way to break down

**Feline Diabetes**

Unlike thyroid disease, diabetes mellitus is relatively simple to diagnose in the shelter (understanding that stress-related hyperglycemia must be ruled out) using a bedside glucometer and/or urine dipstick. However, this disease is particularly difficult to treat successfully in the animal shelter setting. First, cortisol plays a large part in insulin-glucose regulation. Cats that are stressed in the shelter, have concurrent diseases such as periodontitis, renal failure, or heart disease are not great candidates for adoption; therefore, complete bloodwork is recommended if treatment is being pursued. Likewise, it may be beyond the means and care capacity of the medical staff to treat ketotic cats who require emergency treatment in the shelter. Second, daily management of stable cats with diabetes in the shelter requires trained personnel to give injections 12 hours apart, and close daily observation of eating and urinating, with clear communication with medical staff when appetite is sub-normal. For shelters starting insulin treatment for a newly diagnosed diabetic, glucose curves and/or fructosamine level monitoring are recommended after the first few weeks and until stable are essential to determine the proper dosing and (hopefully) any remission events. If remission is the goal, veterinarians should use the more expensive glargine or detemir and do their best to keep the cat’s BG below ~200mg/dL. Dietary management using a low-carbohydrate high-protein diet and reducing obesity in this population is optimal, while maintaining a consistent good appetite. It is understandable that with all of these challenges, many shelters have a policy of euthanasia for all diabetic cats. For shelters able and willing to provide treatment and management until adoption, significant investment in humane housing and enrichment

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is critical: first to reduce stress, and second with the understanding that the length of stay until adoption for these cats may be very prolonged, —months to years— since adopters willing to take on these challenging cats are quite rare.

**Feline Heart Disease**

Heart disease is common in older cats, affecting up to 25% over 10 years of age, and is often occult.\(^8\)

Although murmurs are not greatly correlated with hypertrophic cardiomyopathy (in fact, cats with HCM but no murmur often have worse disease), snap pro-BNP can be a very useful part of a diagnostic rubric in cats with murmurs. Cats with murmurs should also be screened for anemia and tested hypothyroidism. Listening for gurgle rhythms or other arrhythmias are more sensitive markers of HCM than listening for a murmur, since these are more likely to be correlated with the disease. Radiographs and ultrasound are also available at some shelters. Although the sensitivity and specificity of radiography for hypertrophic cardiomyopathy may not be great, radiographs can pick up on congestion or DCM if present. Ultrasonography for diagnosis of hypertrophic cardiomyopathy is rarely part of a shelter vet’s toolbox, but may be available on referral. Since much of the heart disease in our older cat population is occult, we may not know about it until after adoption or until anesthetic death occurs.

**Tritrichomonas foetus**

Tritrichomonas foetus is a motile protozoan that occasionally causes chronic diarrhea in cats, most commonly found in cats living in large colonies in unsavory conditions, and should be on the list of differentials for any feline large bowel diarrhea. Although not traditionally considered a chronic disease, treatment and diagnosis are somewhat complicated. For example, treatment with common GI antibiotics such as metronidazole, tilosin, or sulfa drugs may resolve the infection temporarily, and fecal samples while on these drugs may be negative, but the organism and symptoms quickly reappear when these antibiotics are discontinued. This is because the organism can hide deep in intestinal crypts, where medications have difficulty reaching. Diagnosis on feces for cats not on antibiotics may be done cheaply and effectively with fecal tritrichomonas culture (same pouch as for cattle) and occasionally with direct wet mount of feces (not very specific), but the test with the fastest turn-around time and highest specificity is the more expensive fecal PCR. Ronidazole, a metronidazole relative, is currently the most specific treatment available, and seemed very promising at first. However, some cats cannot tolerate the therapeutic doses and manifest neurological disease or vomiting, and up to 36% of cats may have only partial to no response to treatment.\(^9\)

Unlike many of the other chronic conditions listed here, cats may clear this disease on their own after several months to a few years, but may have prolonged intermittent large bowel diarrhea episodes in the meantime. Even when cats respond to therapy in the shelter, adoption can be a difficult proposition: not too many people are willing to take on a kitten or cat who has the possibility of recurrence of chronic diarrhea, especially a contagious cause that could potentially affect their cats at home. Furthermore, since many of these cats come from hoarding or cruelty situations where their behavior toward humans may preclude treatment, many shelters elect to euthanize the worst cases.

**Chronic small bowel disease**

Middle-aged to older cats who present with chronic small bowel disease can be particularly challenging to manage in the shelter, since daily care is onerous for staff, work-up is prolonged, and can be invasive, expensive, and yield inconclusive results. The two most common causes of this syndrome are IBD and lymphoma, and since both can cause similar hematological and biochemical profiles on bloodwork,\(^10\) the best


diagnostic technique for cats who fail to respond to symptomatic diarrhea therapy (ie probiotics, antibiotics, bland diet, or hydrolyzed diet) is intestinal biopsy. Shelters are often well set up for abdominal surgery, but histopathology may be deemed expensive. For shelters pursuing diagnosis, care should be taken during patient selection, and behavioral suitability and other diseases (such as hyperthyroidism and trichomonas) ruled out first. Surgeons should be careful to take multiple samples of various parts of the GI tract, including but not limited to liver, pancreas, lymph nodes, duodenum, jejenum, and ilium, as well as any segment that is determined by the surgeon to appear abnormally thickened or inflamed. Colonic samples should be taken only if large bowel symptoms are also noted. Lymphoma (or intestinal mast cell disease or adenocarcinoma) is unlikely to be an adoptable condition, so shelters should be prepared to euthanize these animals when diagnosis is achieved, which, according to one study, was approximately 40% of chronic small bowel cases. Using prednisone to symptomatically treat cats without biopsy is not recommended, since most adopters are not anticipating a cancer diagnosis they may receive from their GPs down the road. Many shelters find IBD cats extremely difficult to manage; in shelters where IBD is also a condition where euthanasia is employed, middle-aged to older cats with chronic small bowel disease should be worked up as described, and then euthanized without biopsies if infectious or hypersensitivity-type conditions are ruled out.

Although it may seem counter-productive in many of these cases to spend money on diagnostics only to euthanize an animal, without spending the money, cats that can be managed will not achieve the benefits to their welfare that proper care and eventual adoption can give. Defining a medical budget and (mostly) sticking to SOPs can take a lot of the ethical anxiety out of decision making for veterinarians, staff, volunteers, and community stakeholders. SOPs should be regularly revised to reflect the latest scientific understanding and treatment options for these conditions.
Questions:

1. When should a shelter test a cat for FeLV, if they are no longer testing every cat?
   a) **When a cat is not responding to treatment of routine diseases**
   b) When a cat is being moved to the adoption floor
   c) When it’s been 14 days since the last test
   d) When a cat in the next cage over tests positive for FeLV

2. Which test would NOT be the most appropriate use of shelter funds for initial workup of an elderly polyuric/polydipsic cat?
   a) Azo stick
   b) **Thyroid hormone**
   c) Urine specific gravity
   d) Blood glucose

3. Which shelter protocol for hyperthyroid cats is the LEAST appropriate?
   a) Cats are tested, and euthanized if T4>4.0
   b) Cats are tested, sent to foster for 4 weeks on methimazole, tested for renal failure, and then adopted with 2 weeks of medication to go home.
   c) **Cats are tested, and sent home for adoption on methimazole.**
   d) Cats are tested, kept in enriched housing for 3 weeks on methimazole, tested for renal failure, then thyroidectomized and adopted.

4. Cats with chronic diarrhea from *Tritrichomonas felis* should be treated
   a) with metronidazole or tylosin
   b) **with ronidazole**
   c) with panacur
   d) no treatment; these cats are unlikely to respond to any of the above.

5. When created in advance shelter protocols for treatable/manageable disease can do all of the following EXCEPT:
   a) **Create complete consensus about every case**
   b) Prevent volunteer and staff drama about euthanasia decisions
   c) Provide fair and balanced use of limited medical and shelter resources
   d) Ensure decisions are based on evidence and prognosis, keeping feline welfare paramount
Gastric dilatation volvulus: It doesn’t have to make your stomach turn

Objectives
• To be able to effectively stabilize a patient with GDV pre-operatively
• To efficiently and effectively perform surgical gastric derotation and incisional gastropexy
• To understand pre-operative prognostic factors and their accuracy in predicting gastric necrosis
• To improve the post operative care of patients with GDV to maximize their successful release from the hospital
• To understand the importance and effectiveness of prophylactic gastropexy in at risk dog breeds.

Introduction
Gastric dilatation-volvulus (GDV) is a true surgical emergency, but surgical skill is only one component of successfully treating a GDV patient. Patients with GDV present in hypovolemic and cardiogenic shock and require aggressive pre-operative resuscitation. As well as improving the cardiovascular status, patient stabilization can improve gastric wall perfusion, prevent further bacterial translocation and sepsis and reduce the incidence of ventricular arrhythmias. Importantly, patients with an increased time between presentation to the hospital and surgery tend to have better outcomes, which speaks to the importance of effective emergency stabilization prior to general anaesthesia and surgery.

Post operatively, GDV patients require 24 hour care and monitoring, so referral to a 24 hour facility should be considered, where available. The prognosis for patients with GDV is fair to good, with appropriate timely treatment. The most important prognostic factor is presence of gastric necrosis necessitating partial gastrectomy. However, the challenge of GDV cases is that the ultimate determination of gastric wall health must be made intra-operatively. Even so, there are several prognostic factors have been shown to be predictive of gastric wall necrosis. For those patients suspected to require partial gastrectomy, referral to a specialty centre is strongly recommended as the surgical procedure and the post operative care required by these patients is significantly more challenging than for those who have adequate gastric wall viability. This lecture aims to provide a framework for working up and treating a case of GDV, so that you can approach your next case methodically and efficiently, whether you decide to perform the surgery yourself or to transfer the patient, after stabilization, to a referral facility.

Pathophysiology
• Acute gaseous distention of the stomach that is accompanied by a twisting of the organ on its mesenteric axis.
• Most commonly aerophagia leads to gastric distention and then volvulus, but distention can occur primarily in the case of gastric foreign bodies and gastric neoplasia.
• Increase in intragastric pressure leads to decreased flow through caudal vena cava. This results in portal hypertension, systemic hypotension and progressive cardiogenic shock.
• Pylorus moves ventrally from right to left, coming to rest dorsal and to the left of the oesophagus.
• Complex pathophysiology requires assessment and consideration of the following:
  o Blood flow
  o Cardiac dysfunction
  o Gastric wall necrosis
  o Bacterial translocation
  o Reperfusion injury

Approach to a clinical case
• Gain history – duration of clinical signs, prior episodes of clinical signs may offer prognostic information.
• Physical exam – focus on cardiovascular stability initially, perform more thorough exam once patient is more stable.
Prior to surgery, consider the following:
- Minimum database bloodwork
- Analgesia
- Assess/treat cardiac arrhythmia
- Treat hypovolemic shock
- Right lateral abdominal radiograph
- Decompress stomach
- Evaluate prognostic factors

Cardiovascular stabilization
- Place 2 x wide bore (18G+) cephalic catheters
  - Draw blood at time of catheter placement for minimum database
- Administer crystalloid bolus of 20-40ml/kg rapidly. This equates to roughly 1L for an average standard poodle or 2L for a Great Dane.
- +/- Colloid (hydroxyethyl starch) bolus 10-20ml/kg
- Reassess HR, BP
- Repeat as necessary
- Connect ECG, evaluate for VPCs
- Oxygen therapy

Analgesia
- Opioids preferred, pure mu agonist
  - Fentanyl 3-5mcg/kg bolus, then CRI 3-5mcg/kg/h
  - Methadone 0.2-0.5mg/kg IV
  - Hydromorphone 0.05-0.1mg/kg IV, may cause emesis
  - Avoid butorphanol
- Avoid NSAIDs
- Lidocaine CRI 25-50mcg/kg/min

Evaluate ECG
- Ventricular arrhythmias
- 40-70% dogs with GDV, 48-72h post operatively
- Myocardial ischaemia and myocardial depressant factors - specific cause unknown
- Treat VPCs if:
  - Ventricular tachycardia (HR > 130-160); R on T; multiform VPCs; patient hypotensive despite adequate volume
  - Treatment:
    - Lidocaine CRI 2mg/kg IV bolus, then 30-80mcg/kg/min CRI
    - Analgesia
    - Improve perfusion – IV fluids, gastric decompression

Radiographs
- Right lateral abdomen is diagnostic, other views if stable (avoid VD)
- “Double bubble”, “popeye arm”, “smurf hat”
- Soft tissue band between cardia and pylorus - differentiate from gastric dilation
- Evaluate for free peritoneal gas and gastric intramural gas
- +/- Thoracic radiographs – evaluate for aspiration pneumonia, metastatic disease

Antibiotics
- Bacterial translocation - GI venous stasis, gastric wall compromise
- Ischemia-reperfusion injury
- Cefazolin or Unasyn (ampicillin-sublactam) - administer at induction, q 90min intra-operatively
Bloodwork
- Minimum database
  - PCV/TP, BG, lactate, venous blood gas for pH, electrolytes
- Pre-operatively
  - CBC – evaluate WBC, platelets
  - Chemistry – evaluate kidney function, liver function, albumin, electrolytes
  - PT/aPTT – risk of DIC, consumptive coagulopathy

Gastric decompression
- Trocharization
  - Right (or left) side, behind ribs
  - Avoid spleen - percuss, U/S
  - Clip, sterile prep, gloves, 14G catheter, can repeat
- Orogastic tube
  - Difficult in awake patient
  - Sedate (or induce)
  - Large bore tube, lube, +/- intubation - risk of aspiration
  - Do not force - may perforate esophagus

Anesthesia
- Premed
  - Opioid (pure mu agonist)
  - +/- Benzodiazepine
    - Midazolam 0.2mg/kg IV
    - Diazepam 0.2mg/kg IV
  - Avoid acepromazine, α₂ agonists
- Induction
  - Propofol, alfaxalone IV to effect
- Maintenance
  - Isoflurane + O₂
  - Fentanyl CRI 5-10mcg/kg/h
- Lidocaine CRI 25-50mcg/kg/min

Surgical approach
- Ventral midline celiotomy, xyphoid to prepuce/4th mammary glands
- Remove falciform
- Balfour retractor, moistened lap sponges
- Expect hemoabdomen due to avulsion of short gastric arteries
- Dog with GDV will have greater omentum covering stomach (compared to dog with gastric distention only – some dogs will have derotated by the time of surgery)

1. Gastric derotation
- Perform first
- May need to decompress stomach: needle + suction or orogastric tube
- Stand on patient’s right side
- Right hand grasps pylorus (on left side), left hand forms fist and “kneads” cardia (displaced to right) dorsally, while pylorus is pulled ventrally and to the right (towards surgeon)

2. Abdominal exploration
- Perform complete abdominal explore - allows stomach time to “declare itself”
- Palpate carefully for gastric foreign body, evaluate spleen
- Hemostasis of short gastric arteries

3. Assessment of gastric wall viability
- Subjective assessment by experienced surgeon 85% accurate
• Colour
  o Pink, bright red usually ok
  o Dark red, purple questionable
  o Black, grey, white resect
• Gastric wall slip
  o Mucosa suffers most ischaemic necrosis
  o Thin wall consider resection
• Peristalsis
  o Improvement in peristalsis more likely to be viable

4. Partial gastrectomy if necessary
• Challenging surgery, referral is ideal for these cases
• May need to call owner intraoperatively if large area of gastric necrosis
• Stay sutures, isolate stomach from abdomen
• Ligate blood supply close to greater curvature
• Incise back to bleeding serosal tissue
• 2 layer closure if location permits
  o Mucosa/submucosa – simple continuous appositional
  o Seromuscularis – continuous inverting
  o 2-0 or 3-0 PDS
• Preferred over invagination
• +/- splenectomy

5. Gastropexy
• Many techniques described
• Recurrence rate of GDV close to 0%
• Gastric dilation may still occur
• Incisional is technically simple, effective
  o Pexy site on stomach: 2-3 cm oral to pylorus, ventral surface, between curvatures, 4-5cm long
  o Pexy site on body wall: RIGHT transverse abdominis, just caudal to last rib, parallel to skin incision, 1/3 distance from ventral to dorsal
    1. Stand on dog’s LEFT side
    2. Towel clamps on RIGHT body wall, assistant elevates
    3. ID last rib, diaphragm insertion (rib 11)
    4. Place 2, partial thickness stay sutures of 2-0 PDS
       a. 2 separate needles
       b. Knot to stomach
       c. Leave long ends, leave needle attached
    5. Make partial thickness (seromuscular) incision in gastric wall between stay sutures
       a. New #15 blade
       b. Visualise submucosa, do not enter lumen
    6. Suture each 2-0 PDS stay suture to transverse abdominis at site of proposed pexy
       a. Leave needles on, ends long
    7. Make incision in transverse abdominis between stay sutures, full thickness
    8. Suture each seromuscular layer of gastric wall to cut edge of transverse abdominis
       a. 2-0 PDS, simple continuous appositional
       b. Tie to long ends of stay sutures
       c. Suture dorsal incision first!

Post operative care
• Crystalloid +/- colloid fluid therapy
• +/- treat electrolyte imbalances
• Analgesia
  o Fentanyl CRI 3-5mcg/kg/h
  o Methadone 0.2-0.3mg/kg IV q 4-6h
  o Lidocaine CRI 25-50mcg/kg/h
• Continuous ECG if available
• +/- Treat ventricular arrhythmias
• Antibiotics – discontinue 24h after surgery if no gastric necrosis
• Antiemetic, prokinetic, antacid therapy
• Begin feeding 12-24h post operatively
• +/- NG tube – gastric decompression, enteral feeding

Complications
• Ileus – Tx metoclopramide, cisapride, NG tube
• Vomiting – Tx maropitant
• Gastric ulceration – Tx PPI, H2 blocker
• Peritonitis – ongoing gastric necrosis, inadequate gastric resection (re-operate/euthanasia)
• Sepsis – rule out gastric necrosis/perforation
• DIC – may require plasma transfusion, heparin

Prognosis
• Most recent studies: 73% - 90% survival
• Factors associated with increased mortality:
  o Clinical signs for > 6h prior to presentation, recumbent/moribund patient
  o Gastrectomy or splenectomy
  o Hypotension
  o Gastric necrosis
  o Peri-operative ventricular arrhythmias
  o Peritonitis, sepsis, DIC
  o Increased time between presentation and surgery decreased mortality rate
• Lactate
  o Marker of tissue perfusion
  o Normal < 2.5mmol/L
  o Trends indicate success of resuscitation
    ♠ < 6.0mmol/L  99% survival; > 6.0mmol/L  58% survival
    ♡ Cutoff 7.4mmol/L 82% accurate for predicting gastric necrosis, 88% accurate for predicting outcome
    ♠ ≤ 9.0mmol/L  90% survival; > 9.0mmol/L  54% survival
    ♡ > 42.5% change in lactate  100% survival


Prophylactic gastropexy
• Lifetime risk for predisposed breeds: 4% - 37%
• Prophylactic pexy reduces mortality rate in predisposed dogs by 29 times
• Timing – at time of spay/neuter, ideally close to adult size
• Traditional ‘open’ vs laparoscopic-assisted - less post operative pain, faster return to function with laparoscopic-assisted pexy.

Final tips for GDV success
• Adequately stabilize patient – don’t panic!
  o Extra time spent stabilizing improves prognosis
• Be aware of hyperdynamic shock – adequate BP does not rule out shock
• Check ECG and treat arrhythmias
• Have an assistant scrub in
• Trocharize rather than orogastric tube for pre-operative gastric decompression
• Decompress intra-operatively before derotation
• Palpate as many normal stomachs as possible
  o Gastric wall slip, check anatomy
• Patients who ‘walk in’ generally do well!

References
**Gastrointestinal foreign bodies: Minimizing the risk of post operative dehiscence**

**Objectives**
- To discuss healing of the small intestine and factors leading to failure of intestinal healing (dehiscence)
- To focus on specific techniques to maximize successful intestinal healing, including:
  - Pre-operative stabilization and anesthesia considerations
  - Surgical technique
  - Post operative care and monitoring

**Introduction**
Gastrointestinal foreign bodies can be some of the most rewarding surgical emergency cases. However, a simple procedure can quickly turn into a life-threatening situation if inadequate intestinal healing results in septic peritonitis. Failure of intestinal healing can occur due to a number of patient and surgeon factors, and understanding these factors is fundamental in the prevention of dehiscence. Intestinal wound healing differs from other parts of the body in significant ways, so as a surgeon it is important to take steps pre-operatively to maximize intestinal perfusion, intra-operatively to respect the delicate intestinal tissues and post-operatively to support patients until their appetite and intestinal motility has normalized. Case selection is key when learning gastrointestinal surgery, as the technical difficulty of cases can vary from simple to very complex. This lecture aims to provide some important principles in gastrointestinal surgery so that you can approach your next case with confidence.

**Healing of the gastrointestinal tract**
The phases of gastrointestinal healing are the same as other tissues: inflammation, proliferation and maturation (figure 1). However, several important differences exist between intestinal healing and skin healing. When gastrointestinal wounds heal, there is a *lag phase*, which occurs during the first 3-5 days post operatively. During this phase of healing in the GI tract, there is both collagen synthesis and collagen lysis, due to collagenase activity. This results in a 64% decrease in GI wound strength in the first 48 hours after surgery. Success of the intestinal repair therefore depends on the ability of the remaining collagen to hold suture. This is why gentle tissue handling and maintenance of GI perfusion are so critical, as low oxygen saturation and tissue trauma can impair collagen synthesis and lead to an overall failure of repair. Additionally, there are several other important differences between GI and skin healing:
- Increased bacterial numbers, both aerobic an anaerobic in GI tract.
- Constant vascular perfusion of skin compared to GI, can rapidly fluctuate with changes in cardiac output (e.g. shock hypoperfusion of GI).
- Increased wound shear stress in GI due to constant peristalsis and intestinal motility.

The stomach heals faster, regains more of its original strength following surgery and is generally more ‘forgiving’ than the small intestine – dehiscence of gastrotomy incisions is very rare in otherwise healthy patients. Novice surgeons should start by practicing gastrotomy techniques and gradually progress to simple enterotomies before attempting linear foreign body surgeries or intestinal resection-anastomosis.

**Incidence and prognosis of dehiscence following intestinal surgery**
The reported incidence of dehiscence of small intestinal incisions is 7-16%. Cats may be less likely than dogs to develop peritonitis following intestinal surgery (Ralphs et al, JAVMA 2003). Septic peritonitis resulting from enterotomy or anastomosis leakage is a surgical emergency and carries a guarded prognosis, with a reported mortality of approximately 50% (range 5-85%). Intensive postoperative care is required for these patients to treat systemic inflammatory response syndrome (SIRS) at a cost of ~$10,000 in most referral hospitals.

**What factors contribute to intestinal dehiscence?**
Several studies have evaluated risk factors for patients undergoing intestinal surgery. Some of the most consistently reported are:
• Hypoalbuminaemia ≤ 25 g/L (Ralphs et al, JAVMA 2003; Grimes et al, JAVMA 2011)
• Intra-operative hypotension: MAP <60mmHg (Grimes et al, JAVMA 2011)
  o Hypovolaemic shock decreased vascular perfusion to GI
  o If cardiac output is adequate, hematocrit levels up to 15% below normal do not
    impair gastrointestinal wound healing.
• Multiple GI incisions in a single surgery; linear GI foreign body (Schwartz et al, Vet Surg
  2018)
• Pre-operative septic peritonitis: 38% dehiscence for dogs with pre-operative peritonitis,
  6% dehiscence for dogs without pre-operative peritonitis (Grimes et al, JAVMA 2011).

Detection of pre-operative septic peritonitis
Given the detrimental effect of preoperative septic peritonitis on intestinal wound healing, it is vital
that patients be evaluated for gastrointestinal perforation prior to surgery (see list below). Bear in
mind that very dehydrated patients may not have abdominal effusion initially, but that it may
become apparent after fluid resuscitation. Patients with septic peritonitis present some of the
most challenging surgical and post operative patients and referral to a 24 hour facility with
specialist care is strongly recommended.
  • Paracentesis: 1-3cm caudal to umbilicus or U/S guided
  • Cytology: toxic and degenerate neutrophils +/- intracellular bacteria, foreign material
  • Blood-to-fluid glucose difference > 1.1mmol/L
  • Blood-to-fluid lactate difference below -2.0mmol/L

How can intestinal dehiscence be prevented?
It is important to consider both local and systemic factors when performing gastrointestinal
surgery. Local factors include meticulous preservation of blood supply to intestine, through gentle
tissue handling. Wounds should be closed without tension and with a full-thickness, appositional
suture pattern that incorporates the submucosa, the holding layer of the small intestine. Systemic
factors include maintain adequate cardiac output (aim for MAP 90mmHg) and optimal oxygen
saturation levels at the wound edges. Pre-operative fluid resuscitation, close monitoring of blood
pressure under anesthesia, adequate analgesia and maintaining patient body temperature will all
aid in maintaining perfusion of the intestinal wound edges. Early enteral nutrition within 12 hours
of surgery, through either oral feeding (if tolerated) or placement of a feeding tube provides
enterocytes with energy and protein for repair. This is particularly important in hypoproteinemic
patients.

Pre-operative stabilization goals
  ¥ Rehydrate, improve volume status
    o Crystalloid bolus 10-20ml/kg IV over 20min, repeat until HR, BP normalize
    o +/- colloid 5-10ml/kg IV bolus
  ¥ Analgesia
    o Methadone 0.3-0.5mg/kg IV, fentanyl CRI 3-5mcg/kg/h IV
  ¥ Prevent vomiting/aspiration antiemetics
    o Maropitant 1mg/kg IV q 24h
  ¥ Protect oesophagus, stomach antacids
    o Pantoprazole 1mg/kg IV q 12h

Antibiotic prophylaxis
  • Gram positive and gram negative bacteria
  • First generation cephalosporins (Cefazolin) or Ampicillin-sublactam (Unasyn)
  • 1st dose: at induction, then q90min intra-operatively
  • Discontinue within 24h of surgery
  • Extended use of antibiotics does not prevent infections and increases the incidence of
    resistant bacteria.

Surgical tips for success
The lecture will cover specific techniques for gastrotomy, enterotomy and intestinal resection-anastomosis. Intestinal surgery requires the surgeon to be a perfectionist and it is well documented that experienced surgeons have a lower complication rate than novices, so mindful practice is important. Useful methods include practicing intestinal suturing on cadaver tissues – if small animal cadavers are not available then pig or sheep intestine can also be used. Practice gentle tissue handling, particularly avoiding grasping the intestinal edges with forceps, as this can lead to increased inflammation and disruption of intestinal blood flow, which in turn contributes to poor wound healing. Use the forceps to ‘spread’ the edges of an open hollow viscus, rather than grasping them. Start with gastrotomy surgery and gradually progress to enterotomies where clinical signs are acute and resection-anastomosis is unlikely to be necessary. Since linear foreign bodies are more likely to have extensive intestinal damage that may require resection, patients with suspected linear obstructions should be treated by a more experienced surgeon.

When closing enterotomy incisions, use 4-0 monofilament absorbable material (polydioxanone [PDS] is ideal). A simple interrupted, full thickness appositional pattern is most consistent for novice surgeons and care should be taken to incorporate the submucosa into each bite and to avoid eversion of the mucosa wherever possible. The affected section of bowel should be isolated from the rest of the abdomen with moistened laparotomy sponges and contaminated gloves and instruments should be replaced with separate, sterile gloves and instruments when the enterotomy is complete.

Assessment of intestinal viability
Determining whether an intestinal resection-anastomosis is necessary in cases that have not yet perforated can be challenging. Decision making during intestinal surgery can be subjective and improves with surgical experience. Factors that can be used to assess whether intestine is viable enough to heal include:

- **Colour**
  - Pink, bright red  usually ok
  - Dark red, purple  questionable
  - Black, grey, white  resect
- **Intestinal wall thickness**
  - Thick – usually ok, indicates edema
  - Thin – usually indicates loss of layering, impending perforation
- **Peristalsis**
  - Improvement in peristalsis  more likely to be viable
- **Perforation**
  - Carefully evaluate mesenteric border of intestine, this is the most likely site of perforation

Post operative care
Post operatively, patient body temperature is maintained through active warming. Opioid analgesia (pure mu agonists) are provided either intermittently or via continuous infusion until the patient is able to take oral medications. NSAIDs should always be avoided following gastrointestinal surgery. Crystalloid fluid support is provided to continue to correct dehydration, correct electrolyte imbalances, provide maintenance and manage ongoing losses (GI secretions, vomiting, diarrhea). Antiemetic medication (e.g. maropitant) is helpful to reduce stress on suture lines caused by vomiting and to improve appetite. Except in cases of pre-operative septic peritonitis, antibiotics are discontinued within 24 hours of surgery. Enteral feeding is provided within 12 hours post operatively and a nasogastric feeding tube can be useful to suction gastric residuals and prevent nausea, as well as to feed a liquid diet. Post operative monitoring should include PCV/TP, USG and bodyweight daily, electrolytes if derangements were present pre-operatively, gastric residuals if an nasogastric tube was placed and abdominal FAST scan to monitor peritoneal effusion if available. A more detailed discussion of post operative care and complications is covered in the next lecture.
References


Gastrointestinal surgery pearls: Tips and tricks you won’t find in the textbook

Objectives:
• To maximize available pre-operative imaging of vomiting patients for early detection of GI foreign bodies
• To share some useful surgical techniques
  o Approach to linear foreign bodies in dogs and cats
  o Gaining good visualization of all parts of the GI tract (covered in lecture)
  o Managing luminal disparity (covered in lecture)
• To discuss complications of GI surgery and how to manage them

Maximizing pre-operative imaging
1. Radiographs of the vomiting patient
   • Take three views: right lateral, left lateral, VD
   • LEFT lateral shows “what’s LEFT” in the stomach
     o Helpful for gastric and linear FBs
     o Gas in pylorus (on right side) will rise
   • Radiographs equivocal and no signs of sepsis? Repeat in 8-12 hours, treat symptomatically, consider abdominal ultrasound.

2. Detecting linear foreign bodies
   • 50% of dogs with linear FB have no evidence of SI dilation (Sharma, 2010)
   • Comma-shaped (“paisley-shaped”) or “interrupted” gas pattern
   • +/- gastric foreign body on LEFT lateral (dogs)
   • Intestinal plication, loss of serosal detail
   • Note that normal fat cats will have intestinal ‘bunching’ due to intra-abdominal fat, with intestines located on right side of abdomen on VD. Use plication, gas pattern, ultrasound to differentiate.
   • Ultrasound is usually diagnostic

3. Detecting pre-operative intestinal perforation using imaging
   • Loss of serosal detail
   • Free peritoneal gas - cranial to liver, not associated with GIT
     o Serosal and luminal margins of small intestine visible
     o Horizontal beam radiograph will highlight peritoneal gas dorsally.
   • Free fluid on FAST scan
   • Cytology: septic suppurative inflammation

Surgical approach to canine linear foreign bodies
The same general surgical principles apply to linear foreign bodies as were discussed in the previous lecture for enterotomy. Exploration of the abdomen in cases of intestinal plication should be very gentle and is often cursory until the linear foreign body has been removed, to reduce the risk of intestinal tearing along the mesenteric border. In dogs, 67-83% of linear foreign bodies are anchored at the pylorus and most extend to the jejunum. Note that 40% of dogs with linear FB will have septic peritonitis at the time of surgery, so the surgeon should be experienced and prepared to perform intestinal resection-anastomosis in these cases. If you do not feel comfortable with intestinal R&A, referral to a more experienced or specialist surgeon is recommended.

The first step in removing linear foreign bodies is to ‘release’ the anchor point of the foreign body via gastrotomy. The foreign material should be removed from the stomach without excessive traction on the linear portion in the small intestine. The material is cut, the gastrotomy incision is closed and the foreign material is then gently ‘milked’ down the duodenum and jejunum to remove plications. Ideally, all of the foreign material is removed through a single, jejunal enterotomy, aboral to the affected bowel. However, in cases where additional damage to the intestine is likely (foreign material very abrasive), multiple enterotomies may be performed, but this may increase the likelihood of post operative dehiscence. Most canine linear foreign bodies that do not have intestinal perforation can be removed in a single gastrotomy and a single
enterotomy. In cases of intestinal perforation or where a resection-anastomosis is deemed necessary due to questionable intestinal viability, the foreign body can be milked into the section of bowel to be resected and then removed en bloc.

**Surgical approach to feline linear foreign bodies**
The principles of surgery for feline linear foreign bodies are similar to those in canine patients. However, several important differences exist. Cats are more likely to ingest string, whereas dog foreign bodies tend to be thicker fabric or toy material. 50-63% of feline linear FB are anchored around the base of the tongue so a thorough oral examination should be performed in every cat with gastrointestinal clinical signs. Conservative management of cats with string foreign bodies is only appropriate in cases with *no clinical signs* (i.e. string is an incidental finding) and is reported to have been successful in 47% non-clinical cases. 33% of cats with linear foreign bodies have intestinal perforation at the time of diagnosis.

Once the anchor point of the foreign body has been determined, it should be released to relieve the plicated bowel. However, unlike the thick fabric ingested by dogs, string can be difficult to palpate in the intestine and can be embedded in the intestinal wall at the mesenteric border. Therefore, it is usually helpful to make a small, mesenteric enterotomy midway along the plicated region of bowel and to grasp the string with a mosquito hemostat, prior to release of the plications. Then, the string can either be cut from under the cat's tongue, or if the material is anchored at the pylorus, a gastrotomy can be performed as for dogs. The string can then be gently milked down the duodenum while *very gentle* traction is placed with the hemostat. Cats are more likely than dogs to require more than one enterotomy to remove all of the foreign material, to avoid the string tearing through the mesenteric border of the intestine.

**Selected complications of gastrointestinal surgery**

1. **Septic peritonitis**

   This is the most serious complication of intestinal surgery and is a surgical emergency. Prompt detection of leakage from an enterotomy or anastomosis site is crucial to give patients the best chance of a successful outcome. Despite repeat surgery and critical care, the reported mortality of septic peritonitis following intestinal surgery is ~50%. If septic peritonitis is diagnosed, referral to a specialist facility for repeat surgery and post operative care is strongly recommended.

   Dehiscence of intestinal incisions usually occurs in the first 3-5 days after surgery. This corresponds to the ‘lag phase’ of intestinal healing (see previous lecture). Dehiscence in the immediate post operative period is uncommon except in cases of surgeon error. Clinical signs include inappetence or anorexia (particularly in a patient who was previously eating), vomiting and abdominal pain. Pyrexia and a palpable abdominal fluid wave may also be present. Bloodwork may show band neutrophilia. Repeated abdominal FAST scans are helpful in quantifying peritoneal effusion in the post operative period – a mild amount of peritoneal fluid is expected post operatively in patients without preoperative peritonitis, but this should decrease each day. Radiographs are not useful, as free peritoneal gas may be present in any patient who has undergone abdominal surgery for up to 2 weeks post operatively.

   Definitive diagnosis of septic peritonitis requires abdominocentesis and cytologic evaluation of peritoneal fluid. Obtain fluid directly from abdomen and make direct and centrifuged smears, then stain with Diff Quick. Evaluate microscopically for large numbers degenerate neutrophils, intracellular (or large numbers of extracellular) bacteria or plant/food/foreign material. Submit fluid for bacterial culture. Note that blood-to-fluid glucose and lactate differentials are *not* reliable for detecting septic peritonitis post operatively. If sepsis is confirmed, broad spectrum IV antibiotics should be started immediately and the patient should be transferred directly to a referral facility, if possible. A detailed discussion of surgery and post operative care for septic peritonitis is beyond the scope of this lecture. If clients decline repeat surgery, euthanasia is the only reasonable alternative – medical management is not appropriate.

2. **Ileus**

   Ileus is a common complication of gastrointestinal surgery, particularly following linear foreign
body removal or intestinal resection-anastomosis. Clinical signs of anorexia, nausea and regurgitation begin within 24 hours of surgery. Laparotomy, intestinal manipulation, long operative time and extensive resection all contribute to sympathetic overactivation, which reduces normal gastrointestinal motility. The syndrome is exacerbated by anesthesia, opioid use, sepsis and electrolyte derangements.

Treatment of ileus is focused on treating medical underlying causes (sepsis, electrolyte abnormalities) and stimulating normal gastrointestinal motility. Enteral feeding should be started within 12 hours of surgery at \( \frac{1}{4} \) resting energy requirement (RER), either as a continuous infusion (e.g. Clinicare) or in bolus feedings q4-6h. I place a nasogastric feeding tube intra-operatively in all cases of R&A and most linear foreign bodies – it can easily be removed if the patient begins eating and they are very useful for suctioning gastric residuals which improves patient comfort. Prokinetic medications are also helpful and I usually start these immediately post operatively. Some useful prokinetics are:

- Metoclopramide CRI 1-2mg/kg/d
- Cisapride 0.1mg/kg PO q 8h
- Erythromycin 0.5-1mg/kg IV q 8h

Analgesia should be adjusted to the lowest possible dose of opioids that allows patient comfort while maximizing normal GI motility. Pure mu agonists (especially fentanyl, hydromorphone) cause more ileus than methadone or buprenorphine so it can be helpful to transition patients with ileus to less potent opioids.

3. Short bowel syndrome

The normal length of canine and feline small intestines is five times the length of the patient's trunk. Short bowel syndrome (SBS) is a condition of malabsorption and malnutrition that occurs following resection of 50-85% of the small intestinal length. When performing a resection-anastomosis, it is therefore important to remove all abnormal intestine, but not to be overzealous with resection of viable bowel. Clinical signs include persistent, watery diarrhea and weight loss. Thorough and frank client communication is crucial in cases where a large small intestinal resection is required. Management of SBS can be lifelong and involves frequent feeding (6-8x per day) of a highly digestible diet. Adaptation of the small intestinal may occur but usually takes weeks to months. The prognosis for patients with SBS is worse if the small intestinal resection is more distal (ileal resection) and/or if the ileocecal valve is resected.

Conclusions

In summary, to maximize your success with gastrointestinal surgery, case selection is key. Set yourself up for positive patient outcomes by starting with simpler cases (gastrotomy, single enterotomy) in systemically well patients with an acute onset of clinical signs. Referral is recommended for patients who are systemically ill, have linear foreign bodies, protracted clinical signs or septic peritonitis, especially if you are not experienced with intestinal resection-anastomosis and comfortable assessing intestinal viability. Practicing gentle tissue handling and intestinal suturing on cadaveric tissues will greatly improve your surgical confidence and efficiency and patient outcomes. A surgical assistant is invaluable, particularly in cases of resection anastomosis and for novice surgeons. Pre-emptively treating ileus post operatively will shorten your patients’ hospital stays and speed their recoveries. With careful case selection, deliberate practice, good mentorship and critical evaluation of outcomes, surgery for gastrointestinal foreign bodies can be some of the most rewarding cases in practice.

References


**Going with the flow: A practical guide to canine lower urinary tract obstruction**

**Objectives**

- To review the pathogenesis of urethral obstruction in dogs
- To provide a practical framework for emergency and surgical management of patients with urethral obstruction
- To discuss less invasive techniques for management of canine obstructive urolithiasis

**Introduction**

Lower urinary tract (LUT) obstruction in dogs and cats is a common medical emergency. Obstruction of the urethra can be either benign, due to urolithiasis, or malignant, due to neoplasia (usually transitional cell carcinoma). Treatment of a patient with urethral obstruction involves identifying and correcting life threatening azotemia, electrolyte and acid-base abnormalities, identifying the cause of obstruction, restoration of a patent urethra and in many cases, surgical removal of urolith(s). Successful treatment of LUT obstruction requires a comprehensive understanding of the pathophysiology of obstruction, the regional anatomy and the treatment options, which may be non-surgical (cystoscopic basket retrieval, laser lithotripsy) or surgical. Surgical procedures involve open surgery (cystotomy), minimally invasive surgery (percutaneous cystolithotomy – PCCL) or salvage procedures (scrotal urethrostomy).

**Diagnosis of urethral obstruction**

A history of stranguria, dysuria or hematuria usually precedes LUT obstruction. Signalment and history may help to differentiate between benign and malignant obstruction – transitional cell carcinoma usually has a more insidious onset of signs and is common in older, female dogs, particularly Scottish terriers, Shetland sheepdogs and beagles, although any sex or breed may be affected. Patients with urethral obstruction who present with an unstable cardiovascular status should be stabilized immediately (see below). In stable patients, diagnostic imaging should be performed to determine the nature and location of obstruction. Plain radiographs can identify uroliths >1mm diameter and two lateral views should be taken: one with the pelvic limbs extended to highlight the bladder and one with the legs pulled forward to show the pelvic and penile urethra. Collimation should include the perineal region. Plain radiographs have a 25-27% incidence of false negative findings for urate, cysteine and calcium phosphate uroliths.

Pneumocystography or double-contrast urethrocystography should be considered in patients with suspected urolithiasis and equivocal plain radiographic findings. Ultrasound can also be used to diagnose urolithiasis and false negatives occur in 3.4-6.5% of cases (equivalent to pneumocystography or double-contrast cystography). Ultrasound may also detect evidence of a bladder or trigonal mass. In some cases, urethroscopy or cystoscopy is required to detect neoplasia of the pelvic urethra or trigone region.

**Emergency treatment of patients with urethral obstruction**

Urethral obstruction is a medical, rather than a surgical emergency. Initial physical examination and diagnostics should focus on diagnosing and treating cardiac arrhythmias, correcting electrolyte and acid-base disturbances and rehydration without inducing fluid overload. Initial diagnostics should include PCV/TP, BG, serum electrolytes, acid-base status, BUN and creatinine. An ECG should be evaluated for spiked T waves, short QT interval, wide QRS, or absence of P waves due to hyperkalemia.

- **Emergency treatment of hyperkalaemia**
  - **Calcium gluconate** (10%) 0.5 to 1 mL/kg IV over 10 to 20 minutes. Does not alter serum K+, cardioprotective. Onset within minutes, duration 1h. *Monitor ECG for bradycardia.*
  - **Dextrose**: 0.5 to 1g/kg IV, with or without exogenous insulin 0.5 to 1 IU/kg (IV or IM). Drives K+ intracellularly, lowers serum K+. Onset 1 h, duration several hours. *Monitor BG.*
  - **Sodium bicarbonate**: 0.5 to 2mEq/kg IV over 15 min. Drives K+ into cells in exchange for H+ ions. *Monitor acid-base status.*
Cystocentesis can be performed to decompress the bladder of obstructed patients to allow for correction of azotemia and electrolyte derangements. There is a low risk of bladder rupture if cystocentesis is performed by an experienced person in a relaxed, sedated, well restrained patient. Urine should be saved for urinalysis and culture. IV fluid diuresis can then be initiated with potassium deficient fluids (e.g. 0.9% NaCl).

Re-establishing urethral patency
Once obstruction due to urolithiasis has been diagnosed and the location of the obstructive urolith(s) has been identified, urohydropulsion should be attempted to move obstructive uroliths back into the bladder. In male dogs, uroliths are commonly lodged at the base of the os penis and in female dogs obstruction may occur just proximal to the urethral papilla. A digital rectal exam can identify uroliths in the pelvic urethra. *Urohydropulsion is strongly preferred over urethrotomy*, which has a high complication rate and is ineffective in dislodging uroliths from the proximal (pelvic) or distal (within the os penis) urethra. Additionally, urethrotomy makes future attempts at urohydropulsion much more difficult.

Urohydropulsion in dogs requires very heavy sedation and epidural or general anesthesia (ideally). Attempting the procedure in an awake or inadequately sedated patient often is unsuccessful and is more likely to result in patient pain and complications such as bladder rupture or urethral tear. General anesthesia is only be performed once patients are cardiovascularly stable. Performing the procedure in the radiology suite allows for easy assessment of stone location.

**Canine urohydropulsion step-by-step**
1. Premedicate and induce general anesthesia.
2. Perform epidural – ideal to relax skeletal muscle of urethra and prevent spasm.
3. Perform cystocentesis to empty bladder.
4. Take lateral abdominal radiograph to check location of stone(s).
5. Clip and aseptically prepare prepuce (or vulva in female dogs).
6. Non-sterile gloved assistant palpates urethra per rectum and places ventral pressure on urethra to occlude it.
7. Veterinarian places sterile, lubricated red rubber catheter into distal urethra and advances as far as obstruction, then digitally occludes penis around catheter.
8. While the assistant occludes the pelvic urethra, the veterinarian slowly injects saline into red rubber catheter and holds pressure on syringe – this dilates the urethra around the uroliths.
9. On count of ‘three’, assistant will release occlusion of pelvic urethra while veterinarian maintains occlusion of distal penile urethra and puts firm pressure on syringe to force saline through urethra into bladder and move uroliths.
10. Repeat radiographs and attempt to pass red rubber catheter into bladder to check success of procedure.
11. Multiple attempts may be necessary – remember to check bladder size and drain after each attempt via catheter or cystocentesis to prevent iatrogenic bladder rupture.
12. Once stones have moved into bladder, place indwelling (Foley) urinary catheter to prevent re-obstruction and allow further stabilization, or proceed directly to surgery for cystotomy or PCCL.

**Surgical treatment of urethral obstruction**
A detailed description of the cystotomy procedure will be provided in the lecture. Key points include:

- Ensure that the prepuce or vulva is included in the surgical field to allow normograde and retrograde passage of a catheter intra-operatively.
- Exteriorize the bladder and isolate from the abdomen with moistened laparotomy sponges to prevent urine contamination of the abdomen. Cystotomy performed on *ventral* bladder surface.
- Use stay sutures at the bladder apex and bladder walls after entering the bladder to allow visualization of the lumen.
- Obtain sample of bladder mucosa for culture +/- histopathology.
- Close using 3-0 or 4-0 monofilament, rapidly absorbable suture (e.g. Monocryl/poliglecaprone 25) in a simple interrupted or simple continuous single layer, full thickness, appositional pattern.
- Leak test bladder and place additional sutures or inverting pattern if concern for urine leakage.
- Repeat radiographs (+/- positive or double contrast cystography) to ensure complete stone removal. Incomplete stone removal is reported in 20% dogs after cystotomy (Grant, 2010).

Patients should be maintained on IV fluid diuresis for 12-24h post operatively to encourage residual blood clots to be flushed from bladder. Broad spectrum antibiotics (cephazolin or amoxicillin-clavulanic acid) are continued for 7 days while awaiting culture results, since 76% dogs with cystic calculi have a urinary tract infection. An indwelling urinary catheter is usually not necessary, except in cases with pre-operative bladder rupture, severe urethral inflammation or urethral tear. Uroabdomen occurs in <1.5% of patients following cystotomy but minor complications (hematuria, stranguria) occur in 37-50% of cystotomies and are generally self-limiting (Thieman-Mankin, 2012).

Alternative methods for cystolith removal

1. Lithotripsy
   - Using laser energy or shockwaves to crush or fragment uroliths – laser is safest and most efficient for LUT uroliths.
   - Holmium:yttrium-aluminium-garnet (Ho:YAG) laser used for canine cystic or urethral calculi.
   - Urolith fragments removed endoscopically via basket or via voiding hydropulsion.
   - Complications/failure of procedure due to small urethral diameter, bladder rupture, undetected residual uroliths.
   - Female dogs (larger diameter urethra) and/or dogs with low urethroliths make best candidates.
   - Small male dogs with multiple uroliths are not usually good candidates.
   - Reported to be as effective as cystotomy in removing canine uroliths but lithotripsy takes average of 20-25min longer to perform (Bevan, 2009). Equivalent cost, similar short term complications for both procedures.

2. Percutaneous cystolithotomy (PCCL)
   - Alternative option for small patients and male patients with larger numbers of small uroliths.
   - Miniature laparotomy and cystotomy incision, then laparoscope or cystoscope passed into bladder.
   - Retrograde flushing via urethral catheter, uroliths removed via suction or basket retrieval through instrument channel of cystoscope.
   - Advantages over surgical cystotomy: decreased requirement for post operative analgesia, allows magnified evaluation of bladder mucosa and uroliths.
   - Disadvantages over surgical cystotomy: longer operative time, more expensive. (Arulpragasam, 2012)

3. Voiding hydropulsion
   - NOT appropriate for patients with urethral obstruction
   - Useful for especially female patients with recurrent uroliths that are detected early (1-2mm diameter).
   - General anesthesia, bladder filled with saline via catheter, patient positioned vertically and even manual pressure applied to bladder to express uroliths through urethra.
   - Radiographs taken after procedure to ensure complete removal.
   - Urine culture and stone analysis performed as for cystotomy.

References:


Open wounds: We’ve got you covered! Demystifying the world of wound management

Objectives

- To review normal wound healing and how it differs between surgical wounds and open/chronic/contaminated/infected wounds
- To provide an algorithm for the management of open wounds in practice
- To review open wound management techniques and products available
- To discuss methods of wound closure and timing of closure

Introduction

Open wounds are common in both general and specialty practice. They can be very rewarding cases and do not often require a large amount of expensive equipment, so can be effectively treated in a primary care setting. However, treatment can often take time, patience, money and owner commitment to achieve a successful outcome. As veterinarians (and surgeons in particular), we have a tendency to want to ‘fix’ things. The key with open wounds, particularly contaminated or chronic ones, is to resist the temptation to close the wound straight away and instead prepare the client (and yourself) for the long haul. Achieving a healthy, non-infected granulation bed is the holy grail of open wound management – it provides a microbiologically resistant, vascular substrate, after which various methods of wound closure can be considered. Although open wound management can be expensive and time consuming, it is less expensive and time consuming than complications (dehiscence, infection) that arise when a wound that is closed too early.

Primary closure

Primary closure is indicated for surgically created wounds (or clean lacerations, e.g. cut on glass) that are clean or clean-contaminated. The ‘golden period’, 3-6 hours after creation of the wound, applies only to clean wounds and refers to the time taken for bacterial colonization of the wound from the environment. Contaminated wounds, or wounds with extensive tissue trauma (e.g. bite wounds, crushing/shearing injuries) are not good candidates for primary closure, even if they are treated immediately. If there is any doubt at all that the wound may be contaminated, if there is damage to the vascular supply of the wound edges, or if debridement is necessary, a wound should be left open for at least 24 hours, bandaged in a sterile fashion with a moist primary layer and reassessed. If the wound appears healthy and non-infected the following day, with no ongoing necrosis and healthy wound edges, the wound can be closed at that time (delayed primary closure), or open wound management can be continued. There is no detriment in the long term to leaving most wounds open a little longer to ensure the wound environment is healthy prior to closure. More complications occur in practice due to closing wounds too early than from failure to close a wound soon enough! The remainder of the lecture will focus on open wounds that are not suitable for primary closure at initial presentation.

Approach to an open wound case

1. **Triage.** Goals: stabilize patient, reduce microbial burden, prevent contamination
   - Assess and stabilize cardiovascular status first
   - Provide IV fluids, analgesics
Perform initial diagnostics: thoracic radiographs, FAST scan, point of care bloodwork

Brief orthopedic/neurologic exam prior to sedation, pay attention to neurovascular supply of wounded region

Wear gloves – protect wound from nosocomial infection

Copious irrigation of wound with tap water or sterile isotonic solution

Cover wound with antibacterial contact layer, temporary sterile dressing

Continue with remainder of patient work-up (e.g. long bone radiographs, complete bloodwork)

2. **Definitive wound care.** Goals: decontaminate, debride necrotic tissue, provide moist wound environment, antimicrobial treatment

Sedate or anesthetize patient

Protect wound (fill with sterile lubricant), clip *wide* region around wound

Prepare surrounding skin for aseptic surgery (avoid chlorhexidine in wound but on surrounding skin is fine)

Irrigate wound with balanced warm electrolyte solution
• Perform *deep tissue culture* and sensitivity. Culture the wound after it has been clipped, cleaned and lavaged.

3. **Judicious debridement.** Goals: remove foreign material, contaminated, devitalized or necrotic tissue

• Aseptic technique (sterile skin preparation, sterile instruments, cap, mask and sterile gloves)

• Be conservative, remove only tissue that is certainly non-viable, allow questionable tissue time to ‘declare itself’

• Explore wound, including subcutaneous pockets

• Layered debridement - superficial to deep

• Non-surgical debridement (e.g. wet-to-dry bandage)

4. **Moist wound management.** Facilitate debridement, promote granulation tissue formation, epithelialization

• Moisture at wound surface facilitates autolytic debridement through cytokine-rich exudate

• Maintains cells, cytokines, growth factors at wound surface
Current standard of care

Topical antimicrobials +/- systemic broad-spectrum antibiotics (1st generation cephalosporin or amoxicillin-clavulanic acid)

Primary layer – non-adherent surface +/- topical antimicrobials (Table 1)

Secondary layer – draws exudate away from wound surface (e.g. sterile 4x4 or sterile laparotomy sponges)

Cover with soft padded bandage or tie-over bandage

Bandage should be changed every 24 hours initially, then the frequency of bandage changes can be decreased as the amount of wound exudate lessens. Once a healthy granulation bed is present, it is often beneficial to leave dressings on for a week at a time, so as not to disrupt epithelialization.

Table 1: Examples of useful primary layers for open wound management

<table>
<thead>
<tr>
<th>Level of wound exudate</th>
<th>Primary layer</th>
<th>Antibacterial component</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dry (granulation tissue)</strong></td>
<td>Petroleum (+/- chlorhexidine) impregnated gauze</td>
<td>None/chlorhexidine/topical antibiotic</td>
</tr>
<tr>
<td></td>
<td>Non-adherent dressing + topical antibiotic ointment</td>
<td></td>
</tr>
<tr>
<td><strong>Mildly exudative</strong></td>
<td>Calcium alginate or hydrocolloid</td>
<td>Leptospermum honey</td>
</tr>
<tr>
<td><strong>Heavily exudative</strong></td>
<td>Alginate or foam</td>
<td>Silver, leptospermum honey</td>
</tr>
</tbody>
</table>

Second intention healing vs secondary closure
Once a healthy granulation bed is present, there are a number of methods by which final epithelialization may occur. For smaller wounds in low-motion areas, moist wound management can be continued and the wound can be allowed to gradually contract and epithelialize via second intention healing. Advantages are that surgery is not required and therefore this can often be simplest and most cost effective method of closure. Disadvantages are that the process can be time-consuming, new epithelium is fragile and easily damaged and contraction of the scar tissue may impede function.
Alternatively, once a healthy granulation bed has been established, surrounding skin can be mobilized free of the granulation bed and closed over the top (secondary closure). This surgery should occur in the operating room using aseptic technique as for clean surgery. The skin edges are sharply dissected from the granulation tissue and carefully undermined. Minimal debridement of the skin edges is necessary and it is not necessary to remove the granulation tissue as it contains important growth factors and cytokines that can assist in healing. A closed suction drain (e.g. Jackson-Pratt drain) should be placed at the time of surgery for larger wounds to treat dead space and prevent seroma formation.

If there is excessive tension on the wound edges at this stage, then tension-relieving reconstructive techniques, local subdermal plexus flaps, axial pattern flaps or free skin grafts can also be performed at this time.

**Negative pressure wound therapy (NPWT)**

Negative pressure wound therapy, sometimes referred to as vacuum assisted closure, or ‘the VAC’, is an alternative to traditional bandaging as a method of open wound management. Following initial lavage, culture and debridement of devitalized tissue, coarse open cell polyurethane foam is cut to size and placed in the wound bed. The skin edges are secured to the foam and an impermeable adhesive plastic dressing is placed to create an airtight seal. Although a wall suction unit can be used to perform NPWT, a specialized suction unit is preferred as a specific pressure can be achieved. Suction is applied continuously at -125mmHg. The unit removes exudate from the wound and the application of negative pressure improves wound perfusion, reduces edema, stimulates granulation tissue formation and may decrease bacterial colonization. This results in more rapid formation of healthy granulation tissue and means fewer bandage changes are required. NPWT can be used on wounds in most locations but is ideal for large wounds and those in locations that are difficult to bandage. Although the setup costs are higher than conventional bandages, final costs can be comparable for large wounds where large daily bandages under sedation would be required.

**Chronic wounds**

Most wounds will progress through the following phases of wound healing:

1. **Inflammation**
   - Day 0-3
   - Removal of diseased tissue by phagocytosis
   - Predominant cells are neutrophils and monocytes, coagulation cascade creates fibrin seal

2. **Proliferation**
   - Day 4-12
   - Repair of wounded tissue, fibroblasts create collagen
   - Restoration of blood flow, extracellular matrix, epithelium

3. **Maturation (remodeling)**
   - Collagen synthesis complete 4-5 weeks
   - Collagen maturation 12-18 months
   - Return of proportion of pre-wound strength

Chronic wounds do not progress past the proliferation phase and usually present as a region of poorly formed granulation tissue. A checklist approach (table 2) is helpful when presented with a chronic wound, to ensure that both local and systemic factors have been addressed. This helps to identify why the wound is not healing, so that a patient-specific diagnostic and treatment plan can be generated.
Table 2: Factors that may hinder normal wound healing

<table>
<thead>
<tr>
<th>Factor</th>
<th>Treatment/management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excess motion</td>
<td>Splint or external skeletal fixator, patient confinement</td>
</tr>
<tr>
<td>Tension</td>
<td>Tension relieving techniques, revise closure with flap or graft</td>
</tr>
<tr>
<td>Pressure</td>
<td>Adjust bandaging, make ‘doughnuts’ over pressure points</td>
</tr>
<tr>
<td>Fluid accumulation</td>
<td>Absorbent bandages, debride eschar, NPWT, active suction drain</td>
</tr>
<tr>
<td>Necrotic tissue</td>
<td>Serial debridement, remove eschar</td>
</tr>
<tr>
<td>Ischaemia</td>
<td>Correct systemic hypotension, correct anemia, check for thromboembolism</td>
</tr>
<tr>
<td>Infection</td>
<td>Culture and sensitivity, treat with appropriate topical and systemic ABs</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Biopsy, previous chemotherapy or radiation therapy</td>
</tr>
<tr>
<td>Underlying osteomyelitis, sequestrum or exposed bone</td>
<td>Radiographs, debride sequestrum, forage exposed bone</td>
</tr>
<tr>
<td>Immunocompromise</td>
<td>Treat/manage underlying endocrinopathy, discontinue iatrogenic steroids, correct malnutrition, hypoalbuminemia (also consider FIV, effects of chemotherapy)</td>
</tr>
</tbody>
</table>

Take home messages

¥ Prepare yourself and your clients for the long haul
¥ If in doubt, treat with moist open wound management

¥ Optimize wound environment - use checklist for chronic wounds

¥ Debride sparingly, allow tissue to ‘declare itself’

¥ Prevent nosocomial infection, culture all wounds, use rational antibiotic therapy based on sensitivity results

¥ Goal is viable, non-infected tissue or healthy granulation tissue

¥ Take photos along the way – it helps you and the clients to see progress!

References


What lies beneath: Managing thoracic trauma from the ER to the OR

Objectives:
- To discuss the mechanisms and sequellae of penetrating thoracic injuries in dogs and cats
- To review a practical guide to triage and stabilization of patients with thoracic trauma
- To discuss diagnostic imaging of patients with thoracic trauma
- To develop a framework for decision making regarding surgery in patients with penetrating thoracic trauma

Introduction
Thoracic trauma is a common emergency presentation in dogs and cats and may present as either blunt force trauma (e.g., hit by car, fall from height) or penetrating thoracic trauma (e.g., animal bite wounds, penetrating foreign bodies). This lecture will focus specifically on penetrating thoracic injuries, with an emphasis on decision making regarding surgery in these patients.

Sequellae of penetrating thoracic trauma include muscle trauma, rib or sternum fractures, flail chest, traumatic body wall hernias, diaphragmatic hernia, pneumothorax, hemothorax, pulmonary contusions and lung laceration. Patients frequently present with an unstable cardiovascular status. Not all penetrating thoracic injuries warrant surgical exploration and cardiovascular stabilization followed by diagnostic imaging are required prior to making a decision to proceed to emergency thoracotomy. In retrospective studies of dogs and cats who underwent surgery for thoracic trauma, the perioperative mortality rate was 14.6% for dogs and 13% for cats (Lux, 2018). A recent retrospective study of cats who suffered thoracic dog bite wounds found an overall mortality rate of 27% (Frykfor von Hekkel, 2019).

Patient assessment and stabilization

Patients with penetrating thoracic injuries should be taken to the treatment area for immediate triage and evaluation. Supplemental oxygen is administered by mask if respiratory distress is present and an IV catheter should be placed. Minimum database bloodwork (PCV/TP, blood glucose, venous blood gas (i-STAT) and BUN) can be collected from the catheter and SPO2 should be monitored. Initial physical examination focuses on the cardiovascular system, lungs and brain. IV fluids are administered to treat hypovolemic shock and restore blood pressure, but should be used judiciously in patients with pulmonary contusions.

Opioid analgesia is preferred but hydromorphone and morphine can exacerbate panting and so are ideally avoided in favor of fentanyl or methadone. Bupivacaine intercostal nerve blocks can also be performed in cases of rib fractures for additional analgesia. A more detailed physical examination can then be performed, with careful palpation of the ribs and intercostal musculature and evaluation of the orthopedic and neurologic status of the patient. Evaluation of the abdomen via abdominal-focused assessment with sonography (A-FAST) is also indicated. Lacerations over the thorax should be noted as even small puncture wounds can cover large thoracic wall defects and significant intrathoracic trauma. Chest wounds that obviously allow environmental air to enter the thorax should be immediately clipped, cleaned and a sterile airtight dressing placed. Intravenous, broad-spectrum antibiotic therapy should be initiated.
Pneumothorax is common in small animals with penetrating thoracic injuries, and was present in 50-61% of cats and 24% of dogs who presented with dog bite injuries (Cabon, 2015; Frykfors von Hekkel, 2019), and in 46% of dogs and 37% of cats who underwent surgery for thoracic trauma (Lux, 2018). In patients in severe respiratory distress, immediate thoracocentesis may be indicated prior to diagnostic imaging to treat pneumothorax and improve ventilation. Thoracocentesis for pneumothorax is best performed dorsally, with the patient in sternal recumbency. In some cases, intubation and mechanical ventilation may be necessary to provide oxygenation while further assessment is carried out. If pneumothorax is recurrent or continuous after external thoracic injuries have been sealed, one or bilateral thoracic drainage catheters should be placed. Soft flexible Mila chest tubes work well for this purpose and are easily placed in the ER. Intermittent or continuous thoracic suction can then be applied, until either pneumothorax spontaneously resolves or until the patient is stable enough to undergo surgery (see below).

Thoracic radiographs are taken once patient stability permits, taking care, particularly in cats, not to further stress the patient to the point of respiratory collapse. Radiographs are evaluated for pulmonary contusions, pneumothorax, pleural effusion, rib fractures, diaphragmatic hernia, subcutaneous emphysema and fractures of the surrounding vertebrae and long bones. Thoracic-focused assessment with sonography (T-FAST) is also useful in the diagnosis of pneumothorax and pleural effusion. A full description of the technique has been described by Lisciandro et al (2008). Although T-FAST is a reliable method to detect pleural effusion, there are conflicting results regarding its accuracy in detecting pneumothorax. One study found T-FAST had an accuracy of 90% (Lisciandro, 2008), while another study found T-FAST to be unreliable in detecting pneumothorax when compared to thoracic computed tomography (Walters, 2018).

Initial wound management should be efficient and involve clipping and cleaning skin surrounding thoracic wounds, removal of debris and contaminated material and gentle lavage with warm, sterile, isotonic solution. A sterile bandage should be placed and cling film or adhesive plastic drapes are useful to achieve an airtight seal. Further wound exploration and debridement can occur once the patient is more stable and a decision has been made as to whether exploratory thoracotomy is necessary (see below).

**Decision making in emergency thoracic surgery**

1. Indications for thoracic surgery following penetrating trauma

In human medicine, emergency department thoracotomy (EDT) refers to open thoracotomy in the emergency room and is recommended only in patients with penetrating thoracic injuries who present pulseless but with signs of life. The survival rate reported in the human literature for EDT is 11.7% (Seamon, 2015). In veterinary patients, EDT is not practiced and the decision to proceed to surgery is made following initial patient assessment and stabilization (see above).

Several studies have suggested indications for surgery in small animal patients after thoracic trauma (Scheepens, 2006; Cabon, 2015; Frykfors von Hekkel, 2019; Peterson, 2015). In some cases, surgery is clearly indicated, such as in patients with impalement injuries, severe ongoing hemorrhage, large full-thickness thoracic wall defects, severe intrathoracic hemorrhage, diaphragmatic hernia or if cardiovascular stability cannot be achieved (e.g. continuous pneumothorax). In other patients, particularly those with thoracic puncture wounds +/- flail chest following thoracic bite wounds, decision making is more challenging. Although there is no consensus, general guidelines for the need for thoracotomy include rib fractures, flail chest, pulmonary contusions, pneumothorax, or greater than three thoracic radiographic lesions (Scheepens, 2006; Cabon, 2015; Frykfors von Hekkel, 2019). Unlike penetrating abdominal bite wounds, there is less concern for pyothorax following penetrating thoracic trauma and bite wounds can be successfully managed without surgery in some cases. However, in the author’s experience, patients with thoracic bite wounds who fulfill the above criteria generally have
extensive damage to intercostal musculature +/- intrathoracic structures that benefit from surgical intervention.

If a decision is made that exploratory thoracotomy is necessary, referral to a specialist centre is strongly recommended. General anesthesia with mechanical ventilation is necessary, surgery must occur within a clean operating room and the surgeon must be comfortable with thoracic anatomy and experienced in procedures such as lung lobectomy and diaphragmatic herniorrhaphy. In a retrospective study of dogs who underwent thoracic surgery following trauma, 11% had diaphragmatic hernia, 26% required partial or complete lung lobectomy and 50% had muscle defects that required reconstruction (Lux, 2018). Flail segments of the thoracic wall can be stabilized to an external brace or to adjacent ribs, or can be treated conservatively with muscular and skin reconstruction and analgesia via bupivacaine intercostal nerve blocks. Fractured ribs rarely require surgical fixation and can be sutured to adjacent ribs to provide some stability and improve comfort. Indwelling thoracic drainage catheter(s) are placed post operatively to remove residual pleural fluid and air and to monitor cytology. Subcutaneous, closed suction drain(s) may also be necessary if extensive dead space is present.

If thoracotomy is not deemed necessary but partial thickness penetrating wounds are present, these are treated with general principles of open wound management once the patient is cardiovascularly stable. The surgeon should be prepared that skin wounds are ‘the tip of the iceberg’ and often overly much larger thoracic wall defects and extensive muscle damage. In some cases, there may be no externally visible puncture wound but extensive subcutaneous damage to the thoracic wall. General anesthesia and intubation with positive pressure ventilation are therefore indicated for exploration of all but the most superficial skin wounds, given the risk of pneumothorax during wound exploration. Extensive exploration of thoracic wounds outside of the OR should generally be avoided due to the high likelihood of entering the thoracic cavity. More superficial wounds are lavaged, gently explored, debrided and moist wound management applied along with a cross-my-heart thoracic dressing. Once wounds are free of infection, they can be closed or left open to heal via second intention.

2. Timing of surgery following penetrating thoracic trauma

Patients who have sustained penetrating thoracic trauma frequently present in shock and are at risk for systemic inflammatory response syndrome (SIRS) – 55% of canine and 57% of feline patients requiring thoracotomy fulfilled SIRS criteria pre-operatively in retrospective studies (Lux, 2018). General anesthesia and surgery are therefore high risk and clinicians must be aware that surgery in the early post-traumatic period may worsen the inflammatory cascade, resulting in multiple organ failure and death – the ‘second hit phenomenon’ (Peterson, 2015). Timing is therefore a balancing act between not operating too soon and creating a ‘second hit’, while at the same time not increasing morbidity and expense by delaying surgery. Current recommendations are therefore to perform surgery as soon as a patient is cardiovascularly stable, or immediately if cardiovascular stability is not possible without surgical intervention.

Take-home messages

• Patients with penetrating thoracic injuries present challenging emergency cases.

• The presence of a penetrating thoracic wound, rib fracture(s) or flail chest are not necessarily indications for surgery.

• Initial assessment and stabilization and diagnostics to evaluate the extent of intrathoracic injury are performed prior to definitive management of thoracic puncture wounds.

• Results of diagnostic imaging and patients’ response to stabilization measures will dictate whether thoracic surgical exploration is indicated.
If thoracotomy is deemed necessary, referral to a specialty centre is recommended if possible, as soon as the patient is hemodynamically stable.

Some penetrating thoracic injuries can be managed successfully conservatively, without surgery.

Extensive exploration of thoracic wounds outside of the OR should be avoided, and even patients with seemingly superficial thoracic puncture wounds should be intubated and positive pressure ventilation provided for wound exploration.

References:


Advanced Dental Procedures, What’s Possible

Benita Altier LVT, VTS (Dentistry)
Pawsitive Dental Education LLC

As veterinary practices have become more modern, sophisticated and technologically advanced, so has our ability to perform veterinary dentistry to a much higher level than was ever thought possible. Through specialization of the profession and a wider availability of these specialists, we are able to offer our clients’ referrals for more advanced care to board certified veterinary dentists.

As veterinary technicians and veterinarians we need to be completely aware of what kinds of dental care and treatments are available, and when to offer a referral instead of opting for more basic dental care in hospital.

The primary concern that we often see in dogs and cats is periodontal disease; however, if teeth can be salvaged instead of extracted through periodontal surgical techniques and home care, then through these treatments we could benefit the patient over the long term, to retain important teeth for their function.

We will cover some of the many options for advanced dental care briefly to offer basic information on what types of care is available and why it would be recommended.

**Advanced Periodontal Therapy**

Larger and more important teeth can be difficult to extract even with significant periodontal disease, which can result in horizontal or vertical bone loss, furcation bone loss and tooth mobility due to loss of attachment. When we look at teeth through clinical observations and measurements as well as radiographically, we must assess the true extent of the pathology. A tooth can be evaluated on a root by root basis as well as an individual side of each tooth root. A tooth with significant bone loss (>50%) on a tooth root’s surface may have a very poor prognosis even with advanced periodontal surgery, especially if the bone loss is all the way around the root or what is called a four-walled defect. The area in between a multi-rooted tooth’s roots is called a furcation and if the bone is lost from this area it reduces the success of an advanced procedure even further.

**Total Attachment Loss:** This is the sum of the measurement of any gingival recession on the root’s surface, as well as any pocket depth beyond that gingival recession. If gingival recession is not present then it is just the measurement of any periodontal pocket depth beyond what may be considered to be a normal sulcular depth for that specific tooth, in that specific pet’s mouth. This differs depending on the size of the animal, size of the tooth and length of the tooth root specifically.

In order to measure total attachment loss, you must use a periodontal probe with clearly marked 1mm increments and measure from the marginal gingival edge to the bottom of the sulcus or periodontal pocket if there is attachment lost. The bottom of the sulcus is normally attached to the tooth’s surface at or very near to the cementoenamel junction (CEJ). When attachment is lost at this point a periodontal pocket is created and a pathological process begins. The periodontal probe should be used with a gentle hand, in line with the vertical axis of the tooth and walked around the tooth’s structure recording measurements in at least four places around each tooth root. Whenever these measurements are greater than what would be considered a normal sulcular depth around that particular tooth, the measurement should be recorded on the patient’s dental chart.

Conditions such as gingival hyperplasia can create a false pocket depth and not true attachment loss so careful measurement of the excess gingival tissue and noting if the bottom of the sulcus is at the CEJ is important to determining the extent of attachment on these teeth.

If the bone loss or total attachment loss is <50% and there is not significant furcation involvement, or less than a four-walled defect, it may be possible for advanced periodontal surgical techniques, frequent follow
up care (possibly under anesthesia) and daily homecare which is a commitment that the client must make when attempting to “save” important or strategic teeth.

If a periodontal pocket depth exceeds 5mm, it is recommended that open curettage be performed with the use of flap surgery to facilitate the visualization of the bony defect and exposed root surface and allows the practitioner to treat the area to the best of their ability to get the best possible outcome from periodontal therapy.¹

Techniques to perform flap surgeries are fully described in several dental text books and can be learned by veterinarians at wet labs taught by veterinary dentists on the subject, however if surgical procedures are indicated that are beyond the practitioner’s skill level then referral may be the preferred option.

**Dental Radiographs**

Radiographs must be obtained to fully assess the extent of any suspected bone loss. Evaluation of a full set of intraoral dental radiographs will help determine the success of any proposed advanced dental procedure, as well as give the veterinarian a baseline to monitor the progress of treatment. If your veterinary practice does not have the ability to obtain those dental radiographs and the client is interested in advanced dental care and saving teeth rather than extraction, then considering referral from the onset may be in the best interest of the patient.

**Advanced Periodontal Flap Surgeries**

**Apically Repositioned Flap**

This technique can be used to help attached gingiva lay over any remaining alveolar bone, it requires that there is at least 2mm of gingiva to extend towards the crown.¹ This surgery moves the gingiva down onto the root surface after the area is cleaned of unhealthy bone, granulation tissue and debris; and then the area is allowed to heal.² This procedure can be performed on mandibular incisors to allow for a reduction in periodontal pocket depths, allow for daily cleaning by the client and to allow easier cleaning of areas of furcation exposure on multi-rooted teeth.²

Contraindications for this procedure would be >50% bone loss especially on a four-walled defect, grade three (3) tooth mobility and the presence of less than 2mm of attached gingiva before surgery.¹

**Laterally Positioned (Pedicle) Flap**

Indications: When the root surface of a single tooth is exposed significantly due to a cleft that extends to or near the mucogingival line.¹

Contraindications: Tooth mobility due to loss of bone on more than one wall of the alveolar socket, furcation bone loss or lack of commitment on the client’s part for daily homecare and more frequent follow up professional dental care.¹

Carefully created and planned vertical releasing incisions, and the creation of a donor flap which is moved laterally over the area and sutured, is required for this technique.¹ The goal is to partially cover this exposed root surface and allow for at least 2mm of attached gingiva to help preserve the health of this particular tooth, the area of tissue that is exposed from the donor site will heal in by second intention.¹,³

**Free Gingival Graft**

Indicated in specific individual teeth with a cleft like defect that are free of endodontic disease and tooth mobility is not present.³

Contraindicated if endodontic disease is the cause and endodontic disease is not treated first.³ Concurrent periodontal disease must be treated and controlled, if there is tooth mobility the success of this technique will be poor.³ Success will also depend on the client’s willingness to perform daily recommended homecare and follow up treatment with the veterinarian.³
In this procedure a gingival graft is obtained from a donor site separate from the site to be treated and often on the buccal surface of attached gingiva over the maxillary canine, this site offers the largest expanse of tissue. The donor graft is carefully harvested using a template and careful technique is used to avoid damage to the periosteum under this split thickness of tissue. The donor tissue is then used to graft over the recipient site with careful surgical techniques that are fully described in dental surgical texts.

Guided Tissue Regeneration:

The goal of this type of advanced periodontal therapy is to help facilitate the development of cementum on the root’s surfaces and the regeneration of healthy periodontal attachments. Barrier membranes that are either absorbable or non-absorbable are specifically positioned to prevent granulation tissues from invading the area and to allow bone and periodontal ligament cells to develop in the area where they have been destroyed by periodontal disease.

The use of bone inductive materials can assist in such procedures where significant bone has been lost in two and three walled bony defects, and areas of class two (F2) furcation bone loss in multi-rooted teeth.

Endodontics

Endodontics is the discipline in veterinary dentistry that provides treatment of disease involving the inside tissues of the tooth. The inside tissues of the tooth are highly neurovascular and are easily susceptible to trauma that can cause significant inflammation and lead to irreversible damage that can cause tissue necrosis and death. Concussive injury to a tooth can cause the pulp to bleed inside of the tooth into the dentinal tubules or expose the pulp to the oral cavity as in the case of a complicated crown or crown root fracture. Other causes of endodontic disease are near pulp exposures such as in an uncomplicated tooth fracture into the porous dentinal tubules, a carious lesion or cavity, severe abrasion or attrition, or bacterial invasion through the animal’s own bloodstream through the apical delta into the root canal and pulp chamber of the tooth.

Root canal procedures are performed to retain strategic important teeth in their alveolar bony sockets, to retain the function of these teeth even though after the procedure is performed the vital portion of the inside of the tooth is removed and replaced with an inert filling material and then the access and fracture sites if applicable are filled with a composite filling material.

Root canal procedures are not recommended on all fractured teeth, if a successful procedure is to be performed, specific criteria should be met. Often, the tooth will not be restored to its original height before the fracture occurred, because the weakened tooth would be more prone to further damage. Annual monitoring of a tooth that has had root canal treatment is recommended, dental radiographs should be obtained to evaluate the continued success or failure of the procedure.

Restoration of teeth affected with a Carious Lesion

True carious lesions are not common in dogs and especially not common in cats, however if they are found they can be restored after careful evaluation with dental radiographs. Some carious lesions involve the pulp and without conventional endodontics should not simply be restored with cavity preparation and restoration material. If concurrent periodontal disease is also present the prognosis for such teeth may be significantly worse.

Restoration of Enamel Defects

Restorations are performed for several reasons in veterinary dentistry. When we choose to restore enamel defects, which can be acquired defects due to tooth wear: abrasion or attrition or a congenital condition such as that which occurs when the enamel does not form correctly before adult tooth eruption occurs. Enamel formation is complete prior to eruption of the teeth in dogs, by the age of 105 to 220 days after the puppy is born, the permanent dentition should have all of the enamel formation completed. Cases of dental trauma, localized infection or systemic infection causing a fever, hypocalcemia or the
use of certain drugs during this vital enamel forming period can cause defects or malformations in the enamel on unerupted permanent dentition.5

Amelogenesis imperfect (AI) is an inherited defect that has been found to be fairly rare in dogs and also rare in people, it seems to be most common amongst these species located in remote areas where the genetic pool is less diverse.5 Hypoplastic, hypomaturation and hypocalcification are the three subcategories of these types of genetic enamel malformations.5 Enamel hypocalcification leaves the resultant enamel very fragile even though the depth of the enamel is within normal limits, it has failed to undergo mineralization and is easily removed from the dentin below it.5

The goal of enamel restorations is to prevent any further destruction of the surrounding enamel and to protect the pulp and dentin from damage due to changes in temperature, bacterial invasion and further wear or loss of enamel substance.1 Occasionally in the case of attrition we will choose to restore an important tooth, such as a canine tooth, and extract a less important tooth such as an incisor, if these two teeth are rubbing on each other and causing the lesion or enamel defect.4 If the defect is caused by forces on another object other than another tooth, then the source of that wear must be removed to prevent further wear and loss of the restorative material on that tooth or teeth.4

Often a flowable type of composite is used to repair areas of enamel hypoplasia or enamel defects.4 First the enamel defect must be prepared to accept and retain the composite material that will help to restore the tooth to a more normal contour and function.3, 4 Preparation entails the debridement of diseased or damaged enamel and contouring the edges with a pear-shaped bur used in a high-speed handpiece, cooled with water spray.4 Then a hand instrument called an excavator, is used to further prepare the area.4 An acid etchant is then used to remove the smear layer from the exposed dentin and to create an environment where the restoration will bond more effectively through micromechanical interlocking.1, 4

An unfilled resin, which is a bonding agent, which will help the flowable composite attach more readily, is then light-cured onto the surface of the prepared defect.4 Composite is then flowed into the defect and allowed to slightly overfill the area, the composite is then cured with a special dental curing light and then finished with a fine diamond bur or polishing discs so that the edges of the composite are not detectable when investigated with the tip of a shepherd’s hook explorer.4

Depending on the cause of the defect, one or more teeth could be affected; the act of restoration should help to increase the durability of the tooth as opposed to its weakened state without intervention. Care must be taken to inform the client regarding the possibility of lost restorations, the likelihood of further treatment and that the habit or behavior that caused the abrasion if that is the case, must be prevented to avoid further damage to these teeth.4

Orthodontics in Veterinary Dentistry

Orthodontics deals with the correction of malocclusions or abnormally positioned teeth that may be causing tooth on tooth trauma or attrition, or tooth on soft tissue trauma which can cause the patient pain when the mouth is opened and closed.1 It is important to become familiar with the basic skull types, how these occlusions are evaluated and categorized, and what constitutes a comfortable, pain free, occlusion for our patients.

Orthodontic abnormalities should be recognized by the general practitioner early on in the pet’s life. During the primary, mixed or early eruption of permanent dentition phases, the teeth should be carefully evaluated and any abnormalities of this progression should be noted. Persistent primary or retained deciduous teeth can further exacerbate the problems that could be occurring due to teeth erupting out of their proper and ideal positions. At risk breeds are those with jaw relationships that are outside of the normal limits associated with mesocephalic skull types. These dogs and cats should be monitored closely for any signs of malocclusion, and early intervention through interceptive orthodontics should be performed if indicated. If the practitioner diagnoses a malocclusion, referral to a veterinary dentist may be the best option.
Commonly found malocclusions in dogs and cats are:

Lingually displaced mandibular canines: When this occurs the cusps of the canines are tipped too far lingually and may begin to cause soft tissue trauma to the hard palate. Occasionally this condition occurs due to the lack of a space or diastema, between the lateral maxillary incisors and the maxillary canine teeth either bilaterally or unilaterally.

Rostral Cross-Bite: In this condition, some or all of the mandibular incisors are positioned in front of or rostral to the maxillary incisors rather than the preferred scissor bite.

Caudal Cross-Bite: This is diagnosed when one or more of the mandibular pre-molars or molars occlude buccally to the maxillary teeth above them. A more extreme example is when the maxillary fourth pre-molar tooth occludes palatal to the first molar of the mandible on that same side of the mouth.

Level Bite: When the maxillary incisors and the mandibular incisors occlude right on top of each other causing attrition to the crown cusps over time. This could also occur with maxillary pre-molars causing occlusion to occur with mandibular pre-molars on the same side of the mouth which could cause attrition.

Mesioversion of maxillary canine teeth: The maxillary canine teeth are tipped too far forward causing a reduction in the diastema between the maxillary third pre-molar and the maxillary canine tooth on that side. The crown tip can also cause trauma to the patient’s lip.

Malocclusions can be caused by numerous issues, during the periods of growth spurts the four quadrants of the patient’s skull grow independently. During these times if there are traumatic incidences, mechanical forces at play causing an interlock which could inhibit the normal growth, genetic limitations or factors, poor nutrition or other risks these malocclusions can occur.

Orthodontic movement involves precise and specific dental procedures that should not be undertaken by the untrained professional; however careful observation and a basic understanding of occlusion can really assist us in making the proper recommendations for further treatment rather than disregarding these conditions as unavoidable and untreatable situations. Early and careful intervention may be required even when only primary dentition is involved, to prevent more involved orthodontic intervention later on in life.

Summary

Always keeping the patient’s best interest in mind can assist the veterinarian or technician in making the proper observations, and then recommendations, for more advanced dental treatment and procedures. Developing a working relationship with a referral veterinary dentist can be very helpful in obtaining advice via a phone consultation on a specific case. Radiographic and photographic interpretation can be facilitated via internet communications. Having a financial quote ready for the client should they choose referral for more advanced treatment options, will be important when they need to make treatment decisions. Open communication with the client will increase the general understanding of the findings and diagnoses and help the client feel more comfortable with your recommendations and referral.

References

In our profession, veterinary dentistry, ergonomics and workplace safety have often been overlooked as our focus has been on improving patient care while we minimized the importance of insuring a healthy, comfortable and injury-free environment for the provider of that care to work on a daily basis. Human dentistry has over the years provided us with some research and insight on ways to provide dental services to patients without causing the dental operator to suffer from musculoskeletal disorders that result from a disparity between the physical capabilities of the dental operator and the physical requirements of the procedures to be performed.

Participatory ergonomics (PE) has been defined as: “The involvement of people in planning and controlling a significant amount of their own work activities, with sufficient knowledge and power to influence both processes and outcomes in order to achieve desirable goals” according to JR Wilson. We have learned that work-related musculoskeletal disorders cause a significant amount of lost work time, decreased productivity and job satisfaction, bodily injury to workers and many other undesirable outcomes. Gaining valuable quantitative and qualitative information through PE can assist your hospital in creating change.

**Goals of participatory Ergonomics in Veterinary Dentistry**

- Reduction of risk factors associated with WMSD
- Decrease injuries and lost time from work due to WMSD
- Prevent decline of worker’s abilities over time
- Increase productivity and efficiency
- Increase communication and understanding between all levels of the organization
- Empower individuals to create cost effective solutions
- Increase job satisfaction
- Increase patient care and safety
- Ongoing evaluation and future planning

**Process of Participatory Ergonomics**
1. Assess, identify and analyze risks
2. Formulate recommendations: feasible, achievable, efficient
3. Plan of action
4. Implementation of solutions
5. Periodic re-evaluation
6. Document success and failures

Successful PE programs have shown that changes to organization, practices and design of working environment, combine to make significant improvements. All staff members should gather information, voice their opinions, make suggestions and create solutions to cultivate “a new way of working” that serves our goals.

There are many levels of concern regarding ergonomics and dentistry; here are three that should be evaluated through PE.

1. Operator posture during procedures
   a. Adjustable height procedural tables: The table and the patient must be adjusted to the operator to ensure a comfortable and seated posture maintaining the normal curve of the spine of the operator. Furthermore, patient comfort and retention of body heat are imperative.
   b. Seating: Proper seating should support a neutral spinal posture and avoid nerve compression in the legs of the operator.
   c. Lighting and Magnification: Head mounted lighting and magnification is critical to prevent awkward postures thus reducing eye, neck and shoulder strain while supporting a neutral spinal posture.
   d. Power equipment: Equipment location, height and handpieces all play a role. Swivels, integrated lighting, a comfortable grip, handle diameter and straight air and water lines can make a difference on high and low-speed handpieces.
f. b. Hand instrumentation: Handle diameter, weight, grip and color coding can increase user comfort and efficiency.¹

g. 3. Organizational and Scheduling Ergonomics

h. a. Stress and repetition are risk factors for WMSD.⁵,⁸,¹¹ Proper scheduling of patients and operators requires a responsive and receptive leadership team involving PE to ensure the staffing is appropriate to support a physically and emotionally safe workplace.⁸

i. b. Patients with high-complexity and increased treatment times should not be scheduled back to back for the same operators. Breaks are imperative. Teamwork is essential to prevent tasks from overworking one person.

Participatory Ergonomics in veterinary dentistry can create a safe, satisfying and collaborative workplace for everyone involved. This should be our ongoing desire because those that benefit truly matter to the success of our profession; the staff, patients and clients we serve.

Cumulative Trauma and Repetitive Strain disorders involving the muscles, nerves and bone structures are among the nation’s most serious occupational hazards. They can have a serious economic impact on the practice and may lead to practitioners’ early retirement from the profession due to inability to perform the tasks required of them.²

Veterinary dentistry is a precise and detailed discipline in which the human operator is required to use small and large muscles of their upper bodies as well as concentrated use of their hands, eyes and mind to accomplish treatment of the oral cavity. These procedures are often very time consuming and exhausting given the nature of veterinary dentistry being focused primarily on treatment of disease, rather than primarily on prevention alone. Often the term “dental prophy” is confused with performing periodontal therapies, which is much more involved treatment of a patient’s oral health.

The muscles, joints, tendons and ligaments of the hand, wrist, upper arms, shoulders, neck and back are often stressed repeatedly to the point of fatigue and then to exhaustion within the scope of one difficult dental case. Over time, this can lead to a chronic disorder and if not allowed to fully heal, this chronic disorder can lead to permanent and irreversible changes in the joints, nerves, vasculature and muscles that are involved in the injury.¹¹

Injuries of this nature often present with very similar symptoms such as pain in the neck, back or shoulders, hand or wrist. Numbness or tingling of hand or fingers and also loss of strength in these areas may occur.⁹ If an injury is suspected, a physician should be consulted for proper diagnosis and treatment so that healing can occur before permanent damage has been caused.

We, being the caring providers that we are, often overlook our own discomforts in order to keep providing patients with treatment. Tending to be a very stoic group of individuals, we look out for the needs of others first. We must remember that if we are not healthy and physically able to perform procedures, we will be of no help to those who need us.

Prevention of these injuries and physical disorders through ergonomics principles in veterinary dentistry is crucial to ensuring that dental operators do not have to suffer injury and require treatment in the first place. Remember that ergonomics is not just about manufacturers designing equipment that we can use and hold that will automatically alleviate any risk that the job may entail; it is also about using our minds to manage the situation to prevent un-necessary risk that may cause these types of injuries. This may involve having ergonomically designed equipment and an ergonomic layout for our work spaces as well as using time management and scheduling strategies to reduce some of the repetitive stresses that can lead to injury.

Some patient scheduling strategies that can be employed to reduce strain during the day are:

• Avoid scheduling two or more consecutive patients that require extensive extractions, root planing or other advanced dental procedures that require constant use of pinch grip type hand instrumentation.
• Avoid scheduling more than one larger breed patient in a row.
• If you are healing from an injury, allow yourself time to heal before committing to treat a patient that will require you to repeatedly re-injure the area of concern.
• Train others in the practice to perform dental procedures to help allow for breaks between patients for each operator. During these breaks, you may perform other tasks that do not require stress of the same musculoskeletal areas.
• Practice mini-breaks during the procedure to stand and stretch strained muscle areas.

Operator posture is of major concern when practicing dentistry. Everything from the chair that the
operator sits on to the height and adjustability of the dental table can have a large impact on maintaining a neutral posture during the procedures that are being performed.

- Operator’s shoulders should be in a relaxed, even position.
- Neck should not be tilted forward more than 15 degrees from upright.
- Avoid tilting head to one side.
- Avoid hunching shoulders up and keep the weight of the head evenly over the spine.
- The back should not be tilted any farther forward at the waist than about 20 degrees from upright and the operator should not slump or hunch the back.
- Ideally, the forearms should be parallel to the floor when in a working position and the elbows should not be elevated significantly above the hands.
- Upper arms should align with the vertical line of the torso.
- Hands and wrists should be positioned such that the “pinky” finger is slightly lower in orientation compared to the thumb side and the wrist should be in a neutral position avoiding flexion, extension or wrist deviation toward the “pinky” finger side.

A neutral posture overall minimizes the extension or flexion of all body parts that may deviate them from a natural or relaxed position. That is not to say we must stay in a rigid non-compromising posture but just like an airplane, we are constantly trying to realign ourselves with our flight path so that we may remain on course to our destination. Our plan is to reduce fatigue and stress on the joints so continually working toward a more neutral posture in these areas will help to accomplish our goals of injury free motions. Change of our ingrained poor habits takes much conscious effort and dedication on our part and must take place over time to develop newer and healthier habits. Our muscles have learned to hold our skeletons in a certain manner and have compensated for our lack of conscious posture control so the muscles must un-learn and re-learn a new and better way by our active redirection during the procedures. Equipment that can assist our efforts to learn new neutral postures includes:

- An adjustable chair suited for veterinary dentists and technicians.
  - Must adjust for the height of the operator and the table on which the patient rests.
  - Have a stable base with a ring foot rest so that the user’s feet can safely rest there and the user’s thighs are parallel to the floor when seated.
  - Another seat option is the saddle type seat which allows the user to be in a neutral sitting position with the feet on the floor, legs spread apart and the spine in a more naturally curved position which is said to aid in the operator’s balance, ease of movement and hand-eye coordination.
- Adjustable table for the patient.
  - Adjust the patient for optimum flexibility to facilitate a neutral posture overall with an adjustable height table.
  - Reduces lifting of heavy patients or awkward movements to accommodate patients.
- Instrument Handles
  - Choose larger diameter (3/8”), round handles with a texture grip to help facilitate an easy grasp without having to use excessive pinching forces.
  - Purchase elevators and luxators with handles that fit into the palm of the operator properly and allow the fingers to rest near the working end of the instrument.
  - Frequently change from one type of procedure involving hand instruments to another to avoid excessive time performing one specific type of motion such as that involved in extensive root planing.
  - Choose instruments with lighter weight handles.
  - Maintain instrument sharpness and do not attempt to use broken or damaged instruments.
  - Use instruments in the areas of the mouth for where they are intended. Proper instrument adaption will aid in maintaining a neutral wrist posture as often as possible.
- Gloves:
  - Choose hand-specific right and left gloves to help reduce stresses on hand muscles and joints from ill-fitting ambiidextrous exam gloves.
  - This will aid in instrument stabilization in the hand without having to use excessive force due to the lack of slipping that occurs when gloves are too loose.
  - Do not wear gloves that are too tight.
• Lighting and magnification of working area
  • Using an eye loupe with magnification can be very important to maintaining a neutral head and neck posture.
  • Avoids tilting the head forward and attempting to see your working area.
  • A light mounted on the frame of the eye loupes can offer "line of sight" lighting so that the light shines where you are looking. This eliminates the shadow we tend to cast with our head when we are between the light source and the working area.
  • Direct visualization of the working area will reduce eye strain, neck and shoulder strain and allow us to maintain a more neutral spinal position as well.

• Ultrasonic scalers
  • The correct and effective use of ultrasonic scalers can reduce the need for excessive use of hand instrumentation alone.
  • The scaler handle should be held with very light forces and a fulcrum should be used to balance the movements by the hand.
  • The user should be familiar with the specifics of their particular equipment so that it can be used properly to avoid damage to the tooth or soft tissue structures as well as reduce any strain on the operator’s hands.
  • Hearing protection should also be utilized with these units.

Organization of the dental area should allow for access to commonly used items without requiring excessive reaching or twisting of the body. Cords and equipment should be kept up off the floor if possible to avoid any tripping hazards in the working area. The use of safe lifting procedures (bending at the knees and squatting, rather than bending at the waist) will help reduce back injuries when lifting equipment or patients in and out of the dental area as well. Use of eye and respiratory protection is required to prevent inhalation of biological material or inadvertent damage to ocular structures from flying debris.

As with any other possible exposure to radiation hazards, radiology safety procedures should be followed. The dental technician should never hold the sensor, film or cone while an exposure is being obtained and should remain a safe distance from the primary beam (6 feet) preferably behind a lead shield or apron wearing a Dosimetry badge for monitoring of exposure.

Ergonomics and safety in the veterinary dental profession have become of increasing importance to us as veterinary dental technicians due to our continued drive for excellence in the field. We are finding more pathology than ever before with the common use of thorough methods of diagnosis with radiology and the trained professional, thus increasing the time we are taking to completely diagnose and treat the anesthetized patient. With this in mind, we need to be even more diligent in learning and acquiring new habits to maintain neutral postures, use proper techniques and equipment, and practice healthy habits so that we may remain physically functional to perform dentistry on veterinary patients for years to come.

References
Dentistry. Vol 22 No 2. 124-130
When we think of the words “a dental” which we have commonly come to know as really any dental care that is done under general anesthesia for our veterinary patients, we are really over simplifying what is, or should be, occurring when we undertake professional dentistry for animals. Comprehensive Oral and Radiographic Evaluation with professional scaling and polishing is the basic procedure that all animals need.

Dentistry is a large discipline that requires its own specific set of skills, knowledge, correct instrumentation and equipment to accomplish prophylactic and therapeutic treatment. Extensive training and practice by the practitioner are required to ensure the correct methods are used to reduce or eliminate infection and pain in our patients’ mouths.

A thorough knowledge of anatomy and pathology as previously discussed is a starting point for the professional dental treatment of veterinary patients. Only after diagnostic methods have been employed by an oral exam, complete charting of the oral cavity and obtaining dental radiographs, can we determine the extent of the disease and create a treatment plan for the patient. When the diagnostics have been completed then the actual treatment can begin.

If there are no indications of periodontal disease, fractured teeth, tooth resorption, missing or unerupted teeth, supernumerary, crowding, mobile teeth or other situations that will need more advanced treatment, the dental prophylaxis can begin. Prophylaxis, which means the prevention of disease, really can only apply to healthy mouths that need to be thoroughly cleaned of any plaque and tartar to help prevent pathology that may develop if the plaque and bacteria that it contains is allowed to stay in contact with the oral soft and hard tissue structures.1

If the patient’s mouth is currently in a state of active disease such as that of periodontitis, where there is destruction of the periodontal ligament and alveolar bone occurring, this requires more extensive treatment which should correctly be called “Periodontal Therapy.”

Periodontal therapy requires more involved and invasive treatment of pathology to bring their mouth back to a healthy state. This should be discussed with the client to help inform and increase understanding of why disease has already occurred and why treatment of this disease will require longer anesthetic procedures, possible tooth extractions and dedication to homecare procedures if the client desires to try and save teeth instead of having teeth extracted. Sometimes the periodontium has undergone such destruction that saving teeth even through more advanced methods is simply not a feasible option due to a poor prognosis for the tooth, lack of commitment to homecare or future anesthetic visits, to allow follow-up therapy to be performed.

General anesthesia is required for all veterinary dental patients and should be undertaken with all of the same precautions that would be allowed for any surgical candidate. Pre-operative blood/urine or other diagnostic testing should be performed and interpreted. An estimate for treatment should be presented to the client with a contingency plan for further authorization once the patient is fully evaluated under anesthesia and radiographs have been obtained. Prevention of hypothermia, hypotension or other anesthetic complications should be carefully assessed and steps should be in place to manage the patient as closely as possible to prevent any situations that could be avoided.

A multimodal pain management protocol should be taken into consideration as well, especially if periodontal surgery or extractions are part of the dental treatment plan. The use of regional dental anesthetic blocks is fast becoming a standard of care in oral and dental pain management when painful procedures are to be performed.
Use of a cuffed endotracheal tube is a must to prevent accidental aspiration of fluids or debris from the oral cavity into the airways. This tube should be monitored closely for any obstructions and the cuff should be checked again several minutes into the procedures to ensure a secure seal without causing any trauma to the trachea by over-inflation.

There are several steps involved in the Comprehensive Oral and Radiographic Evaluation (CORE Dental Procedure) including professional scaling and polishing. Additional treatment needed would go in the category of oral surgery (extractions), periodontal therapy, orthodontics or other advanced dental procedures.

**Basic Dental Instrumentation**

<table>
<thead>
<tr>
<th>Item</th>
<th>Recommended</th>
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<tbody>
<tr>
<td>Explorer/probe:</td>
<td>Probe/explorer combination (UNC15/23) General use</td>
</tr>
<tr>
<td>Scalers-dog:</td>
<td>Towner/Jacquette Sickle Scaler</td>
</tr>
<tr>
<td>Scalers-cat:</td>
<td>Morse 0-00</td>
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<tr>
<td>Curettes-dog:</td>
<td>Gracey 1/2</td>
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<tr>
<td></td>
<td>Barnhart 1/2 Universal curette</td>
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<tr>
<td></td>
<td>Columbia 13/14 Universal curette</td>
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<td></td>
<td>Columbia 4R/4L Universal curette</td>
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<tr>
<td>Curettes-cat:</td>
<td>Double ended NV series feline curette (Shorter working end is ideal for cats)</td>
</tr>
<tr>
<td>Sharpening kit:</td>
<td>India slip stone (for reworking instruments), Arkansas stone kit (daily sharpening), honing oil and plastic test sticks.</td>
</tr>
<tr>
<td>Power Instruments:</td>
<td>Ultrasonic scaler of either a piezoelectric, magnetostrictive stack insert or a magnetostrictive type with a ferrite rod insert. This scaler must have a water source to cool and irrigate the working end while in operation. It can be a stand-alone unit that is attached to a pressurized water bottle or integrated into a dental delivery unit that houses a compressor, which uses compressed air to drive the slow-speed handpiece, ultrasonic scaler, high-speed handpiece and air/water syringe.</td>
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<tr>
<td></td>
<td><strong>Low-speed handpiece</strong>: The handpiece is either driven by an electric motor pack or integrated into an air driven dental delivery unit.</td>
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<tr>
<td>Prophy Angle:</td>
<td>This attaches to the low-speed handpiece and allows a prophy cup to be placed on the working end. This is used to polish the teeth after the cleaning. Disposable angles are important to prevent patient cross-contamination.</td>
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<tr>
<td>Prophy paste:</td>
<td>These should be single use individual cups of prophy paste, to prevent patient cross-contamination, which when used on the teeth following the ultrasonic and hand instrumentation of the tooth will reduce any microscopic grooves created in the enamel to eliminate a rough, plaque-retentive surface that can be created by scaling and curettage. Fine, medium or coarse grits are available, however it is usually recommended to choose a fine or medium grit paste to prevent removal of too much enamel when polishing and to create a smooth surface. Some prophy pastes contain fluoride so be aware that fluoride can interfere with some</td>
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</table>
restoration procedures and should not be used in these cases on a tooth that will have a restoration performed.³ Flour pumice is a good choice for those needs. **Do not use a fluoride type of prophy paste if you plan to use Sanos® Sealant on the patient.**

Chlorhexidine Oral Solution: This 0.12% chlorhexidine solution is used to irrigate the oral structures to help reduce bacterial aerosolization exposure to the operator and bacteremia to the pet’s bloodstream.

**The Fifteen Step C.O.R.E. Dental Procedure with or without Oral Surgery-Advanced Care**

**Step 1: The basic oral/facial/skull examination:**

This exam should occur in the awake patient. This helps us to create a treatment plan for the client prior to anesthesia. This is just the initial exam however. Please see the notes in the pathology and anatomy section.

**Step 2: Photographs:**

With the use of a digital camera we can take before photos of the oral cavity and hard and soft tissues of interest so that we may offer the client a visual comparison.

**Step 3: Radiology:**

Full mouth dental radiographs are an important step in further diagnostics and documentation of oral structures and should be obtained prior to commencing treatment. Some prophy pastes can be seen on radiographs so post-treatment radiographs should be obtained prior to the polishing step.

**Step 4: Oral exam on the anesthetized patient:** This is a more thorough evaluation of the oral and facial structures including a complete occlusal evaluation. (Evaluate occlusion prior to the placement of the endotracheal tube on the sedated patient.)

**Step 5: Charting:**

Thorough documentation as previously discussed is a must in locating any areas of concern or pathology and working towards creating a plan for further diagnostics and treatment. The use of the instrument called the explorer/probe is necessary during this step. Also a dental mirror can further enhance our ability to visualize the areas of concern. Proper lighting and magnification will greatly increase our ease of recognition of oral structure abnormalities. A complete chart that allows full documentation of pre and post treatment with multi-directional views of each tooth will assist in complete and accurate documentation as well. Decontamination of the gross calculus may be necessary at this time to help facilitate correct charting.

**Step 6: Treatment plan:**

The creation of a treatment plan for each and every tooth. Paring the clinical findings from the oral examination, documented by charting and the radiographic findings to create this plan. If the plan is to truly perform only prophylactic procedures such as supra and subgingival cleaning and polishing then regional anesthesia and a more complicated treatment plan will not be necessary.

**Step 7: Regional Anesthetic Blocks:** These should be performed now and given a few minutes to take effect before more painful stimulus is caused by extractions or sub-gingival curettage.

**Step 8: Advanced periodontal therapy, exodontics, endodontics or other procedures:** Prioritize these procedures as directed by the veterinarian.
Step 9: Post extraction Radiographs: As indicated by changes to tooth or bone structure these radiographs should be obtained prior to suturing the extraction sites to avoid the need to undo sutures to retrieve root or tooth structure that was inadvertently left behind during the extractions.

Step 10: Re-evaluate Occlusion as necessary: This can be an important step if extractions of major teeth were performed to insure that there are no complications from the patients jaws closing more fully or if we are extracting teeth to alleviate any current malocclusion issues.

Step 11: Dental Cleaning Procedure:

If the patient does not require any other procedures as outlined above then step 7 will be omitted. The dental prophylaxis consists of removal of gross calculus and then removal of all dental calculus from the crown surface or supragingival. The use of the ultrasonic scaler to do most of the major work and then the hand scalers to perfect the work are helpful for this.

The ultrasonic scaler, which operates at a vibration range of 18,000 to 45,000 cycles per second, is utilized to break up the calculus or tartar deposits on the coronal surface of the teeth. The handpiece should be carefully held in the hand with a comfortable grip only using the active sides of the instrument’s tip with the handpiece held parallel to the long axis of the tooth.

The instrument tip should be continuously in motion over the surface of the enamel in a cross-hatching pattern. Care should be taken to keep moving on to the next tooth and allowing each tooth to cool down in between sessions of cleaning. Ultrasonic vibrations can generate enough heat that if persistent can cause thermal damage and possible necrosis to a tooth’s vital inner blood and nerve supply. Water spray, in a fine mist, further reduces the heat that is created and should be sufficient and always in use. The water also helps rinse the debris off of the tooth as it is being scaled clean.

Subgingival calculus and plaque must be removed if there are periodontal pockets below the gingival margin. This is a very important step if truly effective dental cleaning is desired. If there is any debris left below the gum line in the gingival sulcus or in a periodontal pocket then the prevention of oral infection and disease will not be accomplished.

The use of curettes which can safely be inserted under the gingival tissues and into periodontal pockets is recommended. Due to the rounded back and toe of the curette, this instrument, which will reduce the likelihood of damage to the attachments at the bottom of the sulcus if used properly, should be used instead of a sharper pointed scaler.

Curettes must be held in a modified pen grasp, a fulcrum should be established, the instrument should be adapted to the surface to be cleaned, the blade of the instrument should be engaged and then the down or cleaning stroke performed. Overlapping strokes in different planes will ensure that the surface that needs to be cleaned will be completely cleaned.

Different variations of dental curettes are available and help with instrument adaption whether working in the most rostral portion of the mouth on incisors or adapting the instrument to caudal pre-molars and molars. Choosing the correct instrument for the area to be curetted is important to successful adaption, effective plaque and tartar removal and prevention of operator injury due to inappropriate handling of the instruments at awkward angles.

Periodontal bactericidal ultrasonic debridement is the final step in ultrasonic cleaning. A specially made periodontal tip insert is required for this procedure or some dental ultrasonic units are already equipped with a tip that can be safely inserted sub-gingivally. Please consult your ultrasonic equipment manual regarding which tips are safe to insert under the gum line into the sulcus, and at what setting the machine should be turned down to, reducing the frequency of vibrations to a safe level for this purpose.
Periodontal bactericidal ultrasonic debridement occurs due to the ultrasonic sound waves causing microscopic bubbles to form and then implode in the gingival sulcus, cavitation. These implosions can cause the bacterial cell walls to be disrupted and along with the water rinsing through the area at a certain pressure further reduces the concentration of bacteria within the space.\(^1\)

**Step 12: Polish tooth crowns:** This important step helps to create a smooth, non-plaque retentive surface so that the teeth will remain free of plaque.\(^3\) Polishing can remove any plaque that was missed during the scaling and curettage phase of cleaning and helps to remove stains from the enamel.\(^1\) This requires that a **prophy cup**, usually a fairly soft cup, be attached to the working end of a **disposable oscillating prophy angle**. This is attached to the slow-speed handpiece either on the motor pack dental unit or the air-driven dental delivery unit which rotates at between 1,000 to 3,000 rpm.\(^3\) The oscillating disposable prophy angle reduces excessive heat from being generated by rotational forces on the tooth surface and also prevents the patient’s hair from winding around the angle when in use near the patient’s hair on the lip or cheek areas.\(^7\) Disposable, one-use prophy paste cups further prevent patient cross-contamination from the alternative of a multi-use large container of paste.\(^1\) Metal non-disposable prophy angles can be used but generally are not oscillating and require cleaning and maintenance to keep them functioning correctly.

Ample prophy paste should be applied or rubbed onto the tooth surface via the non-spinning prophy cup prior to commencing polishing. It is the prophy paste and not the cup that does the actual smoothing of any enamel defects so this is an important step. By smoothing the paste on the teeth in quadrants prior to polishing you avoid spraying as much prophy paste around the mouth and onto the operator. The use of prophy paste helps to reduce the friction on the surface and minimizes the heat that is generated as well. The choice of prophy paste will depend upon whether fluoride is desired and the grit of the paste required.\(^1,2,3\) Standard paste is either fine or medium grit and usually contains fluoride.\(^3\) Course prophy paste can be used to remove stains from the enamel, however it will remove more enamel and also should be followed up by a fine paste as the ending step to polishing.\(^1,2\)

All coronal surfaces, buccal, palatal or lingual, mesial and distal should be polished in a systematic fashion by starting at the most caudal teeth and working towards the midline or central incisor in each quadrant. The prophy cup should be applied to each surface with only enough pressure to slightly flare the cup out onto the surface and into the gingival sulcus area.\(^1\) Thermal damage to the tooth pulp can occur if the oscillating prophy cup is kept on the tooth for more than a few seconds.

**Step 13: Irrigation of sulcus and teeth:** The gingival sulcus is a prime place for left over debris to accumulate after a thorough dental prophylaxis or periodontal therapy has been performed. If this debris is allowed to stay in the sulcus it will act as an irritant and source of further inflammation or possibly even a periodontal abscess in the future.\(^1,3\)

Potential debris is dental calculus, cellular debris, prophy paste and plaque containing harmful bacteria. We must gently lavage this debris out of the sulcus either by using the three-way syringe on our dental machines to use air and water together to rinse all of the prophy paste and debris from the crowns and sulcus. In addition to this, we may choose to utilize a 6-12 cc syringe filled with a 0.12% chlorhexidine gluconate oral solution and rinse the sulcus around each tooth completely with a 22-28g blunt tip needle or cannula.\(^1,2\)

**Step 14: Post treatment/cleaning photographs:** These digital images along with images of the dental radiographs if applicable can be shared with the client along with the pre-treatment photos to further illustrate the remarkable difference that dental prophylaxis, periodontal therapy or more advanced dental treatment can make for their pet. These can be printed out in color for the client to take home, shared with the client via e-mail or sent to a specialist for their evaluation if necessary to help facilitate future treatment and care. Photos and radiographs also help us to follow visual changes in the patient’s dental health over time. These serial photographs can really show a client the progression of disease if we neglect homecare or professional cleanings.
Step 15: Application of dental sealant products or fluoride if indicated: Fluoride Foam: Application of fluoride foam is controversial but may have some benefits to patients, such as decreased tooth sensitivity especially if dentin is exposed on the coronal or root surfaces because it acts to seal exposed dentinal tubules, an anti-plaque and antibacterial effect because it inhibits bacterial metabolism; and it can help the enamel resist decay. Cavities in dogs are rare and extremely rare in cats so this may not be a viable reason to apply fluoride in our veterinary patients. The down sides to using fluoride are possible toxicity if chronically used in higher than recommended doses and the interference of fluoride with certain restorative, bonding or sealing agents.

If fluoride is applied it should be applied to cleaned, polished, lavaged and dried tooth surfaces. The fluoride foam should be allowed to remain in contact with the enamel or dentin for 3-5 minutes, after that it should be carefully wiped off with dry gauze, do not rinse fluoride foam off with water because it will inactive the fluoride.

Oravet™ Sealant:
Merial Oravet™ is a non-toxic waxy polymer that is applied to the clean and dry tooth surfaces of both cats and dogs. The professional application is of higher viscosity than the thinner homecare kit. It is the base application for the prevention of dental plaque adherence to the enamel surfaces of the tooth crowns. It should be applied up under the gingival margin on the surfaces of the crown and in the sulcus to prevent plaque from accumulating under the gum line. The manufacturer recommends that the client begin the homecare kit applications two weeks after the initial professional application to keep the thickness of sealant in place on the enamel surfaces.

Sanos®: AllAccem, Inc
Sanos® is a product that was developed to help improve gingival health and prevent periodontal disease by providing a liquid bandage like barrier that when applied to the gingival sulcus stays in place for up to 6 months. It has a V.O.H.C.(Veterinary Oral Healthy Council) label for prevention of plaque and tartar accumulation and it prevents gingival inflammation that may be caused by the plaque bacteria invading under the gingival margin. It is easily applied to clean and dry teeth and gingival sulcular surfaces with the brushes in the kit. It dries quickly and is clear to slightly opaque in color. It is non-toxic and approved for use in both cats and dogs. Note: If the practitioner plans to use Sanos® on the teeth and gingiva then a fluoride application is not recommended.

References
4. www.vohc.org
5. www.fda.gov/ForConsumers/ConsumerUpdates
Pediatric Veterinary Dentistry-What You Need to Know
Benita Altier LVT, VTS (Dentistry)
Pawsitive Dental Education LLC

Presentation Goals:
Understand normal eruption and exfoliation of dentition in canine and feline patients.

Create a greater understanding of dental malocclusions and recognize ways to advocate for the patient's comfortable and pain free mouth closure.

Gain a basic understanding of the Angle Classification system to classify occlusions in veterinary patients and the difference between atraumatic and traumatic occlusions and what this means for the patient.

The Angle Classification system will be discussed so that the dental technician will have a basic understanding of how to evaluate canine and feline patients for malocclusions and how these traumatic occlusions can be corrected with interceptive orthodontics.

Methods of basic interceptive orthodontics will be discussed to help alleviate these traumatic occlusions when seen in the general or specialty dental practices.

What is interceptive orthodontics?
The practice of using interceptive orthodontics in the growing young canine or feline patient involves either the extraction of retained or persistent deciduous teeth or the extraction of deciduous teeth in the very young patient to prevent dental interlock and growth issues of the patient's mandibles or maxilla. The main goal is to prevent malocclusion issues that could be alleviated through carefully timed and strategic extractions.

How does the normal eruption of the deciduous teeth progress in a puppy and in a kitten?
Three to four weeks after birth, kittens should have erupted all of their deciduous incisors and canine teeth. In the dog the incisors and canines should be present in the mouth by the age of three to six weeks. The remaining pre-molars sequenced from '06 to ‘08 in each quadrant should be in by the age of five to six weeks in both the kitten and the puppy.

What should the dental technician or veterinarian look for in the puppy or kitten's mouth to indicate that interceptive orthodontics may be necessary?
Many puppies and kittens are born with a slightly overshot maxillary occlusion. This allows for easier nursing. This is more correctly referred to as a distocclusion or Angle Class II malocclusion. As the puppy or kitten matures from being a nursing neonate to an independently eating, the mandibles tend to go through a growth spurt as the deciduous teeth continue to erupt.

Monitoring the three to eight-week-old puppy or kitten for this growth spur and concurrent eruption of their deciduous teeth, can help us to predict or intercept if a dental interlock situation occurs.

What is a dental interlock and why does it occur?
A dental interlock occurs when the deciduous teeth erupt and the mandibles are not growing rostrally yet. As this occurs, the crown cusps of the deciduous teeth protrude into the maxillary soft tissues of the palate and the attached gingiva. Due to the fact that the mandibles are still shorter than their possible potential they occlude distal and lingual to the ideal occlusion for a young animal. The sharp tips of the deciduous canines and incisors traumatize the soft tissues and cause a stopping point every time the animal closes its mouth. A painful condition persists, as well as the interlock, setting up a situation that may stop the mandibles, even in a genetically normal dog or cat, from reaching their full growth potential.

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Some breeds of dogs are more prone to this situation due to their genetically inherited conformation. However, this Class II distocclusion is an abnormal occlusion in any type of dog and is found to be extremely rare in a cat.¹

As the puppy or kitten continues to mature, the deciduous teeth will undergo a normal process of root resorption which allows the deciduous tooth to exfoliate at a certain time and the adult tooth to continue to erupt into the mouth to replace it.

For the feline patient, the deciduous incisors should fall out and the adult incisors should erupt at 3.5 to 5.5 months of age.³ Between 4-5 months of age the pre-molars will exfoliate and the adult pre-molar teeth will erupt. The deciduous canines should be replaced by permanent canines at 5-6 months of age.³ The adult molars in the cat will erupt from 5-6 months of age as well.³ There are not any deciduous molars in the mouth of a cat.³

In the canine mouth the deciduous incisors fall out somewhere between 3-5 months of age, the deciduous canines exfoliate and the adult canines erupt between 5-7 months of age.³ Between the ages of 5-6 months the puppy will lose any deciduous pre-molars and erupt the adult dentition to replace them and the molars that do not have any pre-cursor deciduous teeth will erupt between 4-6 months of age.³

If the dental technician and the veterinarian monitors the teething process closely we can recognize any eruption abnormalities whether they are malpositioned teeth, a severe malocclusion, failure to exfoliate the deciduous dentition as the adult dentition erupts, nonerupted adult teeth or other uncomfortable mouth closure issues. As these issues occur a treatment plan should be created to facilitate the removal of any retained deciduous teeth prior to a major malocclusion occurring.

Corrective measures may be strategic extractions of retained deciduous teeth, extractions of adult dentition to correct the malocclusion or a referral to a veterinary dentist for further orthodontic evaluation and treatment as required. Possible advanced options may be orthodontic movement of teeth, vital pulp therapy or root canal therapy after crown reduction or other possible tooth saving procedures.

What is the Angle Classification System and how does this help us to classify malocclusions?

Originally created for human dental patients, this classification system has been adapted to assist us with classification of occlusions in veterinary patients as well. The classifications below are considered part of a group of symmetrical skeletal classifications. In other words, the skeletal development of the dog or cat has symmetry from one side of the head to the other.

**Class 0:** Considered a normal occlusion for both the dog and cat patient.

**Class 1: Neutroclusion.** (MAL/1) malocclusion. This occurs when one or more teeth are not in their proper place, however the general jaw relationship between the maxilla and the mandibles is normal.

**Class 2: Mandibular Distocclusion.** (MAL/2) malocclusion. When this occurs, the mandibular teeth are occluding caudal to the normal position in relationship to the maxillary teeth. Often the mandibular canines are not able to fit into the proper space interdental to the maxillary third incisors and the maxillary canines. This type of classification can be due to either the length of the maxilla or the mandibles or both.

**Class 3: Mandibular Mesiocclusion.** (MAL/3) malocclusion. The mandibular teeth occlude rostral or in front of their normal relationship with the maxillary teeth. This could be due to the maxilla being too short or the mandibles being too long. This is considered a normal occlusion for some breeds of dogs and cats.

**Asymmetrical Skeletal Malocclusions**

Skeletal malocclusions can either be defined as being in an abnormal rostro-caudal relationship, an abnormal side to side relationship or an abnormal dorso-ventral orientation.

When the skull of the dog or cat is in an abnormal rostro-caudal relationship it means that one side of the patients face has a normal dental arch and tooth alignment while the other side of the face is either in mandibular distocclusion or mandibular mesiocclusion.
If the patient has side to side asymmetry of the skull, the midline of the maxilla and the mandibular incisors is lost. The midline at the central incisors on both the maxilla and the mandibles do not line up.

**Mandibular-maxillary asymmetry in a dorso-ventral direction** means that there is an abnormal vertical space or OB (open bite) between the teeth in opposing dental arches when the patient's mouth is closed.

**What are some common methods or treatments that would help to alleviate a painful mouth closure in the young feline or canine patient?**

Once a problem has been recognized then a treatment plan can be developed to help prevent malocclusions or at least give the growing puppy or kitten the best opportunity to have a comfortable occlusion, free of pain and discomfort.

Interceptive orthodontics may involve extraction of deciduous teeth in the pediatric dog or cat to alleviate a dental interlock situation or to extract the retained deciduous teeth in the younger adolescent dog or cat at the time they should be exfoliating.

A full dental evaluation under anesthesia, evaluation of the patient's occlusion prior to placement of an endotracheal tube and a set of full-mouth dental radiographs should be obtained to assess the extent of the issue looking for retained deciduous teeth, or in the case of a very young puppy or kitten when deciduous teeth are being extracted at eight-twelve weeks of age, the location of the adult tooth buds.

Once the dentition and the occlusion has been clinically evaluated and diagnostic radiographs have been obtained and interpreted and a diagnosis made by the veterinarian, a plan for treatment and/or extractions can be made. Often the extraction of retained deciduous teeth will help the corresponding adult teeth move into a proper position to prevent the malocclusion in the first place if the jaw relationship is not severely maloccluded in the first place. If the jaw relationship is such that this normal occlusion becomes impossible then more invasive orthodontic procedures may be necessary.

Occasionally extractions are performed on newly erupting adult incisors or other non-essential teeth are extracted to make a space for the more important canine teeth to fit. Occlusions such as the (MAL/3) mandibular mesiocclusion can cause the maxillary incisors to occlude directly with the soft tissues that are just lingual to the mandibular incisors. This occlusion can be very painful causing dents and sores to develop. This trauma to the soft tissues can cause loss of attached gingiva on the lingual aspect of these mandibular incisors creating early periodontal disease and bone loss.

Options to alleviate this kind of dental malocclusion are crown reduction and vital pulpotomy or root canal therapy for all of the maxillary incisors that are causing the trauma or extraction of those incisors if the former is not a feasible option.\(^1\)

**Masel chain** is an elastic type of chain that is used to stretch between orthodontic buttons or other wired loops which are attached to anchor teeth and the tooth that needs orthodontic movement. This type of **continuous force orthodontic movement** can help facilitate simple movements of the teeth if a malocclusion does occur due to retained deciduous teeth or another type of malocclusion.

**Linguoverted mandibular canine teeth (MAL/1)** are an example of a malocclusion often caused by waiting too long to intervene and extract retained deciduous mandibular canine teeth. The adult mandibular canines erupt in a position that is tipped in towards the tongue instead of tipping buccally towards the lips and fitting into the space interdental to the lateral maxillary incisors and the maxillary canines. If this occurs, orthodontic movement often is required to position the mandibular canine teeth to tip out towards the lips. If there is a space already between the lateral maxillary incisor and the maxillary canine for the mandibular canine to tip into when the patient's mouth is closed, then sometimes "**ball therapy**" can be practiced to put **intermittent force** on these teeth to cause a normal occlusion to occur.
What is "ball therapy" and when is it indicated?

Ball therapy is the use of a toy ball that is of the correct size for the dog patient to easily hold in the rostral part of the mouth in between the erupting mandibular canines. Having the patient hold this ball for a few minutes several times per day will help put intermittent forces on these canines, encouraging them to tip buccally instead of erupting straight upward and hitting the soft tissues of the palate causing trauma. The ball should be of a smooth surface and the right diameter to fit easily in between the mandibular canines without either being too small or too large. Ball therapy should not be attempted if the mandibular deciduous canines are still present in the mouth or if there is not a diastema or space on each side of the maxilla for the mandibular canines to tip into when the mouth is in a closed position.

Conclusion:

Early intervention and a very proactive approach is necessary to provide effective interceptive orthodontics to our canine and feline patients. In order to offer them the best chance to reach their genetic growth potential, avoid painful mouth occlusions, tooth trauma on other teeth or soft tissues. The dental technician and veterinarian must strive to provide a thorough oral exam and occlusal evaluation at every puppy and kitten visit and beyond to the time of fully erupted adult dentition. Using a pediatric type of dental chart for these exams to document what type of occlusion the patient has and what teeth are erupted and what teeth are not, is very important. The period of mixed dentition where the deciduous teeth and adult teeth are present in the mouth at the same time can be a normal process, however it is important to understand when this may be progressing into an abnormal situation and that deciduous teeth are being retained too long. As we learn more and come to recognize the warning signs of an impending malocclusion we will become better equipped to provide appropriate care recommendations and referrals as necessary to make sure that the growing puppy or kitten has the best chance for a comfortable and function dental occlusion in the future.

References available upon written request
A general understanding of terminology, husbandry and common dental diseases or injuries of the oral cavity are vital to the technicians’ ability to obtain a thorough history of the rabbit or rodent patient as well as to assist the veterinarian in performing a complete examination of the structures of the head and oral cavity of the patient, obtaining dental and skull radiographs as well as providing anesthesia support, correct instrumentation and assisting in procedures.

**Terminology**

**Anisognathous:** Maxillary arch is wider than that of the mandibular arch, occlusion of the rabbit.\(^1\)

**Apical Germinal Tissue:** The tissue that originates the formation of tooth structures.

**Aradicular:** Without a root.\(^1\)

**Brachyodont:** This type of tooth has true anatomical roots with a true apex, short crowns and they do not continuously grow.

**Caecotrophs:** digested food and bacterial products that are encapsulated and often consumed directly from the anus by rabbits. The amount consumed is often directly related to the current energy and protein levels in the diet.

**Caries:** Demineralization of teeth, often resultant of a high-carbohydrate diet and can result in tooth resorption.\(^1\)

**Chinchilla lanigera:** A member of the rodent family. Originating from the Andes mountains of South America.\(^4\)

**Clinical Crown:** The portion of the crown that is exposed in the oral cavity above the gingival margin.

**Collagen Fibrils:** The weak tissues that comprise the periodontal ligament in elodont teeth due to their continued eruption throughout the life of the animal.\(^1\)

**Diphyodont:** Having both deciduous and permanent sets of teeth.\(^6\)

**Elodont:** Continuously growing teeth with "open roots". There are no "true" anatomical roots.\(^3\)

**Hypsodont:** Teeth with long crowns.\(^1,6\)

**Guinea Pigs:** A member of the Caviidae family often called cavies. They are from South America originally and are a part of a group that includes several species of rodents. They are social animals.\(^4\)

**Lagomorpha:** Rabbits belong to this order of species.\(^4\)

**Leporidae:** Rabbits belong to this family.\(^4\)

**Mandibular Symphyseal Separation:** Common traumatic injury in small mammals often due to falls or being dropped.\(^1\)

**Mandibular Prognathism:** Mandible is long in comparison to the maxilla. This is not usually true in rabbits.

**Maxillary Brachygnathism:** Malocclusion common in small rabbit breeds where the maxilla is too short. Genetic in origin this can cause incisor overgrowth un-related to cheek teeth elongation or a non-fibrous or abrasive diet.\(^1,3\)

**Monophyodont:** One single set of teeth, rodents are monophyodont.

**Myiasis:** Parasitic infestation of a mammal by fly larvae (maggots).\(^1\)
Periodontal Disease in Rabbits: Due to a weak periodontal ligament structure and the ability of food to become impacted between cheek teeth when they become elongated, periodontal disease is fairly common in rabbits.¹

Pulp Necrosis: Death of the pulpal tissue inside of the tooth.

Pulpitis: Inflammation of the pulp at the center of the tooth.

Rats: (Rattus norvegicus) originated from the Norway rat. They are commonly kept as pets and used in research facilities. They are part of a group of rodents called Murine rodents along with mice, hamsters and gerbils.² They possess continually growing incisors, but their cheek teeth are not continually growing, have true anatomical roots and are brachyodont in type.²

Reserve Crowns: The part of the crown that lies below the marginal gingiva.

Rodents: Including Guinea Pigs, Chinchillas and Degus as well as others. They possess no canine teeth and two pairs of elodont incisors.²

Silicate Phytoliths: Silica particles stemming from the epidermis of a plant.

Supragingival Crown: This is the portion of the crown that is visible above the gingival margin intraorally.⁶

Spurs and Spikes: These often develop on the lingual occlusal surface of mandibular cheek teeth and on the buccal surface of maxillary cheek teeth due to lack of abrasion by improper diets in animals with elodont teeth.¹ ³

RABBIT DENTISTRY

Understanding the dentition of the rabbit:

Deciduous dentition: Rabbits do have deciduous dentition however it is exfoliated within the first few days to a month after birth and is rarely of any issue.¹ ² ³

Adult Dentition: Adult rabbits have (4) maxillary incisors: (2) that are towards the labial surfaces and (2) incisors that are palatal called "peg teeth". Rabbits have (2) mandibular incisors.¹ Canine teeth: (0). Premolars: (3) maxillary and (2) mandibular. Molars: (3) maxillary and (3) mandibular.

The dental formula looks like this: 2 (I 2/1, C 0/0, P 3/2, M 3/3) for a total of 28 adult teeth¹ ²

The mandibular teeth grow at a faster rate than the maxillary teeth.¹ As an example the mandibular incisors may grow in the normal rabbit at a rate of 2.4 mm per week compared the maxillary incisors growing at a rate of 2 mm per week.³

Enamel thickness varies around the surface of these teeth as well, it is thicker on the labial surfaces and thinner on the lingual aspect of the incisors.³ On the cheek teeth the enamel is thicker on the lingual surface of the maxillary pre-molars and molars and on the buccal surfaces of the mandibular cheek teeth.³

Rabbit and Rodent Examination and Evaluation:

Often reported clinical signs of disease may be fairly vague such as a recent refusal to eat favorite foods, scattered food, weight loss, lack of feces, grinding of teeth, ocular discharge and or facial swellings, un-kempt fur appearance, and an accumulation of caecotrophs.¹ ² Also drooling (slobberers), obvious elongation of incisor teeth or nasal discharge may be obvious in the awake patient.² ³ These can often be signs of dental disease and should be further explored with a clinical oral and facial examination by the veterinarian.¹ Due to the fact that these diseases are often chronic in nature but not often presented until the animal is severely debilitated these are often emergency situations and should be handled as such.³
Obtaining History: A full and thorough history of the patient must be obtained including information about the daily diet and habitat that the animal lives in. The veterinary technician should obtain information regarding the companions that the animal lives with and information on travel and exposure to the outdoors or any unsupervised areas.

Weight: The patients' weight is very important information. We may not have a previous weight to compare this to, however moving forward weight, diet and habits will be extremely important to monitoring the patients' progress after treatment.

Examination of the conscious rabbit: Examination of the oral cavity and facial structures should follow obtaining history. This can be accomplished by the use of a standard otoscope and size 4 or 6 mm cone, however only about 50% of the pathology will be recognized and thus an exam under significant sedation or anesthesia will be necessary after this initial cursory examination if there are areas that cannot be inspected, the patient will not allow examination or if dental disease is strongly suspected.

Equipment required for examination:
Standard veterinary otoscope and cone, Cheek or pouch dilators, mouth speculum to open mouth vertically, magnification loupes and lighting. Optional: lighted bi-valve nasal speculum.

Radiographic Evaluation: This is very necessary for diagnosis of disease, planning and evaluation of treatment procedures and outcomes. Common Radiographic Views: Dorsoventral, lateral, rostrocaudal and two oblique views.

Evaluation and Interpretation of Dental Radiographs:
Supragingival crown length and the shape of the occlusal surfaces should be evaluated.
What is the apical position for each tooth compared to a normal patient's radiograph?
Alveolar bone quality should be evaluated looking for areas of ankylosis or lucency.
To what degree is there rostral convergence of the palatine bone and the ventral border of the mandibles?

Common Dental Diseases in Rabbits and Their Treatment:
Abscessing of cheek teeth or incisors: Commonly abscessed cheek teeth that have caused bony changes on the ventral aspect of the mandible require aggressive extracapsular surgical treatment often with associated removal of bone and any teeth involved in the abscess. Radiographs must be performed prior to treatment and extraction planning to assess the full extent of the diseased area. Post abscess antibiotic therapy is often done by packing an antibiotic impregnated substance or beads into the defect to provide a high dose of antibiotics that is not absorbed systemically to any great degree. The surgical incision is closed over the application of the medication to ensure that it remains antibiotics are often used concurrently as well for two to three weeks post-extractions or treatment.

Carious lesions: Often diet related, if these are small then they can be removed by burring away the lesion. They are prone to return and can lead to issues of abnormal wear of opposing teeth.

Cheek teeth elongation: This complex situation requires careful evaluation clinically and radiographically and is a very common finding in our rabbit patients. Occlusal adjustment is necessary using general anesthesia, a straight nose-cone on a slow speed handpiece using a special bur or a long-shanked taper fissure bur. Soft tissues should be protected from damage as well as the proper occlusal plane of angulations restored to these cheek teeth.

Incisor Elongation: Almost always this is secondary to a cheek teeth elongation, however in rare circumstances this can be an isolated issue. Treatment is done with a high-speed handpiece and a cross-cut taper fissure bur to reduce the height of the crowns to function. Care must be taken to avoid pulp exposure and the risk of pulpitis or pulp necrosis. Treatment could include shortening of incisors every three to six weeks, dietary change and/or extraction.
**Periodontal Disease**: Pockets that are greater than 3mm are difficult to treat in rabbits.\(^1\) Quite commonly there is a tooth abscess when deeper pockets are present and this may lead to an extraction decision which can cause other complications with occlusion.\(^1\)

**Traumatic injury**: Symphyseal separation is a common traumatic injury found in rabbits occurring after a fall. Also, pulpitis and if left untreated pulp necrosis is often secondary to a tooth fracture caused by a fall or by improper trimming of incisors with nail clippers that can cause longitudinal fractures in these teeth.\(^1\) Vital Pulp Therapy may be required to prevent or treat these types of tooth injuries.\(^1\)

**Prevention of Dental Disease, Rabbit Husbandry and Diet**: Rabbits should be fed a diet high in fibrous roughage like timothy grass hay as well as fibrous vegetables.\(^3\) No more than one-third of the rabbit's diet should consist of pellets.\(^2\) Post-procedure nutritional support can be provided by a blenderized diet or a commercially prepared diet such as Oxbow Critical Care (Oxbow Animal Health, Murdoch NE; www.oxbowanimalhealth.com).\(^2\) Housing should always be provided in a safe place away from predators or a risk of falling.\(^4\) Often pet rabbits or other small animals are forgotten or neglected if housed away from the primary dwelling.\(^4\)

**RODENT DENTISTRY**

1. **Caviomorph rodents** such as Guinea Pigs and Chinchillas. Teeth have open apices, grow continually, elodont.\(^2\) They are true herbivores.\(^7\)

   Dental Formula: 2 (I: 1/1, C: 0/0, P: 1/1, M: 3/3) total= 20 teeth

2. **Murine rodents** such as rats, mice, hamsters and gerbils. Incisors are continually growing however the molars are brachydont and possess true anatomical roots.\(^2\) Murine rodents are omnivores.\(^7\)

   Dental Formula: 2 (I: 1/1, C: 0/0, P: 0/0, M: 3/3) total= 16 teeth

Anesthesia is generally required for proper intra-oral examination of rodents.

**Common dental issues are:**

Malocclusion causing incisor overgrowth. In caviomorphs the mandibular cheek teeth can overgrow and entrap the tongue below causing the animal to not be able to eat and leading to severe debilitation.\(^2,7\) In chinchillas and guinea pigs, malocclusion is secondary to cheek teeth overgrowth as in rabbits.\(^7\) Trauma, nutritional deficiencies and not enough roughage can lead to malocclusions as well as genetically inherited issues with the length of the jaws.\(^2\)

Tooth root abscess in all rodents, scurvy which is caused by a lack of vitamin C in guinea pigs, cheek pouch impaction in hamsters, caries can occur when diets high in refined carbohydrates are fed or tooth resorption which may be secondary to periodontal disease has been documented.\(^2\) Periodontal disease is likely in elodont teeth and somewhat common in brachydont teeth, it should be treated and prevented.\(^2\)

Similar care must be taken to prevent malocclusions secondary to tooth root elongation of cheek teeth, periodontal disease, tooth root abscess and other dental conditions by providing appropriate housing, chew objects, diet and supervision to rodents as in rabbits.

Rodents should be provided with timothy or similar grass hay and vegetables as roughage.\(^2\) No more than one-third of their diet should be from a pelleted source. Vitamin rich vegetables and supplementation of vitamin C should be fed to Guinea pigs.\(^2\) Rodents must be given chew objects such as wooden blocks to help them wear their incisors.\(^2\)

References are available upon request. pawsitivedentaled@hotmail.com
Goals of this presentation:

- Understand dental radiation safety practices
- Know different types of x-ray generators
- Ensure the sensor is “safe” in the patient’s mouth during the process
- Understand what is cone-cutting, elongation, foreshortening and other dental radiology terms
- Recognize how the animal's head position, sensor position, and beam positioning can each influence the outcome of the image using the “ABC” method
- Putting the “ABC” positioning method into practice—the six-steps to image acquisition.
- Learn the criteria that make an image diagnostic
- Create a goal to work-flow and time expectations for a full-mouth series based on the number of exposures
- Gain an understanding of "what causes what" so that we can quickly troubleshoot our images
- Strategize how to obtain a second view of a tooth for better interpretation possibilities
- Learn ways to utilize dental radiology software to gain efficiency of the process of full-mouth series
- Extrapolate the “ABC” method to obtain intra-operative or post-operative radiographs in lateral head position as needed.

Radiation Safety in the Dental Operatory

All of the same principles apply to dental radiology as to any radiology imaging in practice. Six-feet around the patient's head should be considered as the radius to stay out of when the tube head of the generator is emitting the radiation. All personnel should be out of the zone of radiation during that time. All individuals must move out of the six-foot radius behind the direction of the beam or behind a lead shield. All personnel performing dental radiographs and working in the dental operatory should wear their dosimetry badges consistently to monitor long term exposure. Another consideration is always reducing the focal distance to the patient to the length of the positioning indicating device or tube length whenever possible. It is completely unacceptable for someone to hold on to any part of the tube head or the sensor when the exposure is obtained, even with lead gloves. A lead shield or plate placed under the patient's head, on the side of the table or a larger shield on a floor stand can be utilized to avoid scattering radiation. Lead shielding absorbs radiation, whereas stainless steel tubs scatter radiation.

Dental X-Ray Generators

- Generators can either be mobile on a base with wheels, wall-mounted or hand-held.
- It is essential to understand how the settings on your dental x-ray generator functions and how to manipulate these settings to obtain the best exposure for that particular patient and teeth to be radiographed.
- Each x-ray generator may be a little different; however, modern machines simplify the process by maintaining a consistent Kilo-voltage (KV) or the Milli-Amperage (MA) and only require a minor adjustment in time of the exposure. Preset exposures may be available, allowing you to choose based on patient size and the area of the mouth to be imaged.
- Slight changes in the exposure time are necessary during the whole-mouth series when we are radiographing areas with more or less bone and soft tissues.

Dental Radiology Sensor Safety

Blocking the patient’s ability to feel pain or the sensation of the sensor in the mouth through general anesthesia and analgesia as well as regional dental blocks with local anesthesia are imperative to ensuring the sensor is safe.
Monitoring the anesthetic depth, reflexes, and reaction to any stimulus is key to ensuring the patient is at a plane of anesthesia which allows for safe placement of the sensor. An object can be used between the molars on one side of the mouth to be extra insurance that the patient cannot bite down on the sensor.

Remember this easy three-ingredient recipe: A+B+C= D

A (Animal's head position) +B (Beam-both vertical and horizontal) +C (Sensor/phosphor plate) =D (Diagnostic Dental Radiograph)

Six Easy Steps to Obtaining a Full-Mouth Series of Dental Radiographs

1. Place the patient in sternal recumbency for maxillary series, roll the patient into dorsal recumbency for the mandibular series. Always maintain the hard palate of the maxilla in a parallel position with the tabletop.
2. Place the dental sensor or phosphor plate in the correct location for the first image. Starting with the maxillary molars on the right side of the patient usually makes sense.
3. Dial-in the vertical angle of the beam.
   a. For dogs, we only need four different vertical angles: 70-degrees, 60-degrees, 45-degrees, and 20-degrees. In cats, we only use three angles 70-degrees, 60-degrees, and 40-degrees.
4. Rotate the beam in a horizontal direction per the instructions always relating to the midline of the head. The midline is 0-degrees horizontal rotation.
5. Hover the beam over the sensor or phosphor plate, just as if it was a helicopter coming in for a landing. With a size 2 plate, you can be within ½ to 1 inch of the patient's head. If you have a size-4 phosphor plate, you need to be at least one length of the plate away to enlarge the circle of projected radiation to saturate the phosphor plate and avoid cone-cutting.
6. Step out of the 6-foot radius ensuring all others are out of the radiation zone, push the exposure button. Usually, you must hold the exposure button for 2-3 seconds to expose correctly.

Note: By creating a standard for patient position and sensor position, the resulting x-ray beam position becomes standardized as well. Standardizing all of these things helps to minimize all of the variables that come into play. This method creates an objective and repeatable technique for obtaining dental radiographs that are easy to repeat with each patient. Just like making a great meal, ideally, each ingredient should be objectively measured every time!

Further Explanation of Steps One Through Four

Step 1: Patient Positioning: (A)

The positioning of the patient is critical when you are first learning to radiograph dental structures. If you always position the patient the same way when you are obtaining maxillary teeth or conversely mandibular teeth, then the angles that you use with the x-ray beam and placement of the digital sensor in the mouth, coincide with the patient's head position.

Placing the patient in ventral (sternal) recumbency is a natural position for all maxillary exposures in the canine and feline patients. Patients are often intubated in sternal and can remain in this position as the maxillary images are obtained. Have the patient lie on its chest with the head resting about 2-4 inches above the tabletop either placed on a solid object, ensuring the palate remains parallel to the tabletop. (I recommend a 250- 500mL plastic irrigation saline bottle that has squared sides, filled with water or sand), this works very well to rest the chin or head of most patients.

Dorsal recumbency is the preferred position for all mandibular exposures. When you are ready to move the patient to this dorsal position, carefully turn the patient on its back, ensuring that you do not twist the endotracheal tube or disconnect the monitoring leads. The palate should again be parallel to the tabletop surface. Roll up a small towel and place it comfortably under the patient's neck, which helps to ensure that the patient is in the correct position.

Note: The goal is to ensure the hard palate is as parallel to the tabletop surface as possible.
Step Two: Placement of the Digital Sensor in the patient’s mouth (C)

The placement of the digital sensor is consistent for each image. The flat surface of the sensor is directed towards the primary beam. The sensor should be placed in the mouth with the edge of the sensor at the cusp of the tooth crown(s). The cord from the sensor should always be directed rostral.

➢ Note: No need to try and make the sensor do any position it doesn't naturally do. The angles on the angle cheat-sheet, both vertical and horizontal expect the sensor or plate to lay in the mouth predictably every time. No need to make the sensor flat unless it naturally is when place in the location.

Step Three and Step Four: X-ray Beam Angles (B)

There are two directions in which we can move the tube-head of the generator.

1. Vertical angle: (Most units have degree-markings in a circle on the tube head). The angles run from 0 to 90-degrees. The angle at 90-degrees is perpendicular to the floor and at 0-degrees is level with the floor.
2. Horizontal rotation: This relates to the rotation of the tube head in a horizontal plane around the patient's head through the progression of obtaining all of the images. We can consider the rotation using the patient's midline of their head as a reference point. The midline is 0-degrees, 90-degrees horizontal is perpendicular to the midline.

Always dial in the vertical angle first and then rotate the beam in a horizontal plane to the desired horizontal rotation. We are always relating horizontal rotation to the midline of the patient's head.

➢ Note: Canine Patients at a minimum need a series of 10 to 18 exposures for a full mouth series, more if the patient is a larger dog or you are using a size #2 digital sensors for large teeth. Feline Patients at a minimum need a series of ten (10) exposures for a full mouth series.

What Makes an Image Diagnostic? (D)

There are some basic requirements for our images of dental structures to be diagnostic. For every image obtained, ask yourself these questions.

1. Did I get the tooth or teeth desired on this image?
2. Did I get 100% of the root structures of these teeth on the image?
3. Did I get at least 3mm of bone visible around each root on the image?
4. Did I get at least 3mm of crown structures beyond the horizontal margin of alveolar bone?
5. Is the image the correct length? Did I elongate or foreshorten the tooth structures?
6. Is the exposure correct? Is the image too dark or too light?

It can be essential to have the images of all of the crowns, especially if the crown appears to have some damage clinically; however, the main criteria that must be satisfied are in the questions above. Sometimes additional images may be required to obtain all of the above and the complete crown in larger teeth such as the canines of many dogs.

Dental Radiology Terms Defined

Elongation: Causing tooth and bone structures to become longer and thinner than they truly are. Decreasing the appearance of bone and tooth density.

Foreshortening: Causing the tooth and bone structures to become shorter and broader than they truly are, changing the appearance of bone and tooth density. Enamel folding over the bone is a sign of foreshortening.
Cone-cutting: Occurs when the beam and the sensor or plate are not in alignment when exposed to radiation; part of the plate does not get exposed, causing a white arch to appear instead of an image.

Distal: Directional term relating to the dental arch moving from the midline of the head in a caudal direction. The surface of a tooth or root that is farthest away from the central incisor in that arch.

Mesial: Directional term relating to the dental arch, moving from the midline of the head in a caudal direction. The surface of a tooth or root that is closest to the central incisor in that arch.

What Causes What?

- The vertical angle is directly related to how long or short the tooth appears in the image.
- The horizontal rotation of the beam is directly related to the projection of the tooth either palatal, lingual, caudal or rostral. Horizontal rotation can also cause the elongation or foreshortening of a tooth if it is too far distal or mesial to the tooth in question.
- If the patient's head is not palate parallel to the table, any tipping can cause the tooth or teeth to elongate or foreshorten as this causes a different relationship to the prescribed vertical angle.
- If the sensor or phosphor plate is not in the correct position, what you see inside the image directly relates to where you placed the sensor or plate. If you cut the root apex out of the picture, you most likely did not place the sensor in the ideal position within the mouth.
- All ingredients in the recipe for A+B+C=D are equally important and should be measured each time objectively; however, it is crucial to understand what causes what outcome.

➢ Note: Whenever you are trying to troubleshoot your images, ask yourself which ingredient was not appropriately measured, fix that one thing, and re-expose the area to be imaged. If you do not get D (Diagnostic Image), It is either A, B, or C that caused the issue.

Methods to Get an Additional View to Increase Interpretation Possibilities

S.L.O.B. Rule

Whenever we are obtaining exposures of teeth with multiple root apices, we need to be able to differentiate one root from another. The superimposition of root structures hinders our ability to make diagnostic decisions for that tooth.

There is a rule that helps us to obtain a radiograph of a tooth such as the maxillary fourth premolars #108 and #208 in the dog. Shifting the projected image such that the mesial-buccal root and the palatal root are no longer superimposed upon one another.

Same Lingual Opposite Buccal means that when we shift the primary beam from a 90-degree horizontal rotation, the palatal tooth root follows the shift of the primary beam. The palatal tooth root can be described as lingual since it is more toward the tongue, so that is why the word "lingual" is used in this rule.

The most common method is to take the primary beam and shift it HORIZONTALLY 20 degrees DISTAL at a horizontal rotation of 110-degrees. The resultant image shows the distal root where it is in a caudal location, the palatal root is in the middle, and the mesial/buccal root is rostral on the resulting image. Conversely, if the beam is shifted 20 degrees rostral at a horizontal rotation of 70-degrees, then the resultant image shows the palatal root in the most rostral position, the mesial/buccal root in the middle and the distal root is superimposed over the first molar position.

Rotated and Crowded teeth:

Rotate the horizontal beam direction at least 20-degrees either mesial or distal to project the desired teeth either in a rostral direction (110-degrees horizontal) or a mesial direction (70-degrees horizontal). Distal or mesial tube shifting causes the teeth to lean in direction. Projecting the tooth rostral or caudal allows the practitioner to develop an impression of the bone surrounding the teeth and their root structures from multiple directions. Changing the vertical angle would only serve to elongate or foreshorten the tooth in the image.
Efficient Work-Flow and Dental Radiology Software

All dental radiology programs keep work-flow for obtaining a full-mouth series in mind. Also, there is always the ability to obtain one or a few single images, such as when intra-operative or post-operative images are required. One should become very familiar with their software and all of its capabilities to not only efficiently obtain images such as the use of a template but also for enhancing dental radiographs with filters. Filters and tools can increase our ability to interpret images.

- Each image can be obtained efficiently in 60 seconds or less with a method such as the ABC method. We can perform 10 views in 10 minutes, 20 views in 20 minutes with the mindful practice of the techniques.
- Starting at the caudal molars in the 100-quadrant progressing around the arch to end up at the molars in the 200-quadrant, then rotating the patient dorsally to begin at the molars of the 400-quadrant, rotating around in a circle to end with the molars in the 300-quadrant may make the most sense.
- Keep the tube head close and make small movements; smooth is fast when it comes to being efficient and expedient.

What if You Prefer to Obtain the Dental Radiographs with the Patient’s Head in Lateral Recumbency?

1. Very merely, rotate the patient’s head so that the palate is precisely perpendicular to the tabletop, place a towel under the nose to ensure consistency.
2. Dial the vertical angle to the opposite angle that adds to 90-degrees.
   a. Example: 70-degrees in sternal or dorsal requires a 20-degree angle in lateral.
   b. Example: 45-degrees in sternal or dorsal requires a 45-degree angle in lateral.
   c. Example: 60-degrees in sternal or dorsal requires a 30-degree angle in lateral.
3. Horizontal angle now comes up and over the head just like the sun setting and rising.
4. Sensor positioning is the same as before relating to the teeth to be imaged.
Tooth Resorption on Blast!

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Tooth Resorption (TR) has been proven to occur in man and animal alike, even 13th and 14th century domesticated cats from the former Schleswig, Germany unearthed from 1971 to 1975 and examined, showed clinical and radiographic evidence of tooth resorption. However, even today in 2018, the etiology of this pathologic process remains a mystery and has yet to be fully studied and understood by veterinary dental professionals and researchers.

As veterinary professionals practicing dentistry, we are at the frontlines of discovering and treating this condition to alleviate any pain and infection that tooth resorption may cause our faithful furry patients. First and foremost, we need to understand the histological processes that occurs, what our chief methods of diagnosis are and what may be the appropriate treatment for these lesions.

Feline patients have taken the stage when it comes to research on tooth resorption; however, tooth resorption is very prevalent in humans, horses and dogs as well as many other species of mammals. During routine dental procedures, we are obtaining more thorough dental radiographic studies of these canine and feline patients, and we are discovering more and more evidence of tooth resorption.

- **Resorb**, by definition, means to break down and assimilate something. The tooth and the alveolar bone are two separate distinct structures held together via the periodontal ligament fibers. When the process of resorption occurs, it can be the act or process of breaking down and assimilating the tooth structure into the alveolar bone. Traditionally tooth resorption has been divided into two basic types in veterinary dentistry. Type I in which tooth destruction arise from the cementoenamel junction area and destroys structure either coronally or apically or both. Type I lesions radiographically retain mostly normal root structures with an intact periodontal ligament space. Type II resorption is where the root structures are remodeled into a cementum-bone type of tissue and the periodontal ligament space appears to be lost when evaluating the tooth on radiograph. Further classifications of tooth resorption are defined when it pertains to the destruction or changes in the crown and/or root structure with or without any assimilation of root structure and alveolar bone.

In dogs, the pathogenic nature of TR is not fully understood; however, humans and dogs may share some common risk factors such as periodontal and endodontic inflammation, which can be caused by many issues. Tooth resorption in dogs has also been referred to as idiopathic root resorption. It has also been postulated that cats and dogs share some common risk factors for the development of external replacement resorption. This is based on a tooth resorption study evaluating 109 healthy cats and the results of a 2007 study of 224 canine subjects that were presented to have dental procedures and/or periodontal treatment performed at the University of California-Davis William B. Pritchard Veterinary Medical Teaching Hospital.

On a cellular level there are giant multi-nucleated cells called odontoclasts that are actively destroying dental structures. These cells become active due to unknown reasons, but some theories include inflammation at the gingival margin due to periodontal disease, crowded teeth or aggressive chewing habits that may compromise the blood supply to the tooth roots. Other possibilities may be compression forces caused by space-occupying lesions such as dentigerous cysts or other masses that may be growing in size, gingival hyperplasia, endodontic disease that may occur due to crown fractures. Other causes could be abrasion or attrition and dental carious lesions. These odontoclastic cells may invade tissues around the periodontal ligament fibers at the cementum where external resorption occurs or can be active along the pulp canal causing internal resorption. It is a fine balance that occurs between cells that are depositing new tooth structure and those that are removing tooth structure. As the process continues, further destruction of supportive structures such as the periodontal ligament occurs. Then a
bone-cementum tissue is laid down to replace tooth root structure and the tooth becomes rigid in the socket and cannot move with masticatory forces, this lack of shock absorption may cause root fractures in affected teeth.

Abfraction, which is the occurrence of lateral forces causing stresses on the unsupported tooth structure at the cementoenamel junction (CEJ), is thought by some human dental professionals to be a possible cause of non-carious cervical lesions in people. It is unclear at this time if these types of forces have any bearing on tooth resorption in dogs or cats.

Of the 244 dogs in the UC Davis study, 120 or 53.6% were found to have some type of TR through careful clinical evaluations and full mouth radiographic studies. A significant amount of external replacement and external inflammatory resorption was discovered. Maxillary teeth were only slightly more commonly affected than mandibular teeth and advanced age also was linked to more frequent findings of tooth resorption in both cats and dogs in multiple studies over the last several years. An unexpected increase in evidence of TR amongst neutered male dogs vs. intact male dogs and a large breed dog predilection was also discovered. TR occurred most frequently in all pre-molars and the first mandibular molar in the dog, much less frequently in the mandibular incisors and the second and third mandibular molars and the first and second maxillary molars.

Numerous studies done on feline subjects have found a TR rate of anywhere between 28-67% with a disparity between randomly chosen feline subjects, those of specific breeds and those whom were presented to specifically have dental disease assessed and treated vs. random healthy feline subjects. A study published in 2008 regarding the incidence of tooth resorption in 109 felines found a very similar outcome of about 37.5% of cats being affected. A higher incidence was noted of cats presenting with concurrent dental disease; this group had a TR occurrence rate of around 67.0%. This may suggest that if there is concurrent dental disease there may be a higher incidental finding of TR in cats when evaluations are made with clinical findings and radiographs.

Clinical Findings of Tooth Resorption in Dogs

These lesions often go undetected until significant disease progression has occurred. In dogs TR is often found only on radiographs, where there will be significant type II resorption with the coronal structure of the tooth appearing completely unaffected. If there is a lesion exposing the pulp of the tooth, it is often found on the lingual surface at the cementoenamel junction. A shepherd hook explorer is useful to determine if there are any irregularities in the enamel surfaces and to probe the cementum to feel for any areas of stickiness that could indicate a defect. If one tooth is affected with TR, then often other teeth are affected as well. A complete set of intra-oral radiographs should be obtained if tooth resorption is discovered in a specific tooth to look for other teeth that may be affected as well.

In the case of external cervical root surface resorption, the crown of the tooth may take on a “pink” appearance because of the extensive loss of tooth substance; however, this should not be confused with internal resorption. This type is typically very destructive and leads to involvement of the pulp. The cause of external cervical root surface resorption is unknown.

Another clinical finding is the discovery of mild tooth mobility with the absence of periodontal disease or abnormal pocket depths. If a tooth is mobile in the alveolar bony socket it should be radiographed to determine the cause. Complete tooth root replacement resorption will often leave the crown portion and the most coronal aspect of the root almost floating in soft tissue attachment, which will cause the tooth mobility.

Occasionally, there will be the presence of hyperplastic gingiva or granulation tissue invading an area on the tooth crown where resorption has destroyed part of the tooth’s structure; this tissue can be either very inflammatory and easily disturbed, or very solid and full of connective fibers.
Radiographic Findings of Tooth Resorption in Dogs

The use of standard diagnostic-quality dental radiographs is the primary diagnostic tool that we have for evaluating common criteria to establish a diagnosis of tooth resorption in dogs and cats. The use of a classification system and descriptions of types of tooth resorption for human dental patients has proven to be helpful and applicable in over 96% of the 120 dogs affected with tooth resorption in the 2007 UC Davis study. Some form of tooth resorption was found to be occurring in 11.1% of the total number of teeth studied. These seven types of tooth resorption with specific descriptions of radiographic criteria as well as clinical findings, treatment and prognosis could be very helpful to the veterinary professional. Creating a diagnosis and treatment plan could provide the patient with the best possible treatment recommendations to help alleviate pain and infection in the oral cavity.

Classifications of Tooth Resorption in Dogs

Classifications below are based on the findings of the UC-Davis study on canine tooth resorption as well as the classification system used in human dentistry developed by Andreasen and Andreasen. The following are in the order of the most common occurrence in dogs as found in the 2007 study of the 224 canine subjects:

1. **External Replacement Resorption**: At 34.4% this is the most commonly occurring tooth resorption in dogs. The appearance of these tooth structures on radiograph reveal an indiscernible periodontal ligament space around each tooth root and remodeling of root structure into alveolar bone. In humans, these have a poor prognosis due to the progressive nature of the process of resorption and limited treatment options.

2. **External Inflammatory Resorption**: As the second most commonly found type of tooth resorption in dogs, 25.9% of the dogs in the study were found to have external inflammatory resorption. Inflammation such as periodontal disease and endodontic disease is a common occurrence in human and canine patients. It is thought to be the initiating factor in the development of this type of tooth resorption and addressing the underlying cause could be a primary treatment option for teeth affected with this type of resorption. Radiographic indications could include areas of periapical lucency around resorbing tooth roots which could be consistent with a combination of both periodontal and endodontic disease.

3. **External Cervical Root Surface Resorption**: This type of resorption was found in only 0.3% of the teeth affected by some type of tooth resorption in the 2007 study and seemed to affect neutered male dogs more often than any other sex predilection. Radiographs of these teeth show the progression of these lesions beginning at the cervical area of the tooth where cementum meets enamel causing destruction of tooth structure that often moves both apically and coronally. The tooth destruction can be extensive and is often confused with a type of internal tooth resorption.

4. **External Surface Resorption**: Only 0.2% (17 out of 8,478) of the teeth was affected with this type of resorption. Radiographically these lesions are not always apparent; clinically there are often no signs. If they are apparent on radiographs they will appear as shallow discontinuances of tooth structure on lateral edges of the root and only involve the cementum and dentin of the tooth. The periodontal ligament space and the lamina dura should still appear normal in these cases.

5. **Internal Inflammatory Resorption**: This was a very rare finding in dogs in the study group and only 0.1% of teeth with resorption had this type of resorption. Endodontic disease has been implicated in the forming of these oval-shaped enlargements, which are often located in the cervical area of the root canal. Treatment of the underlying endodontic disease could assist in resolving this type of tooth resorption in dogs.

6. **Internal Surface Resorption**: Mild trauma may be the initiating factor causing these oval shaped enlargements located in the apical third of the root canal. Active revascularization may occur which can be a self-limiting event and may not require treatment. This was found only in one dog out of the 120 with tooth resorption and it did not have concurrent periodontal or endodontic disease.
7. **Internal Replacement Resorption**: This was not found in any of the dogs in study. Located often at the coronal segment of a root fracture, it typically presents as an uneven expansion of tunnel-like areas near the root canal. This condition is not considered progressive and no specific treatment is indicated according to the authors of the 2007 study.

Of the seven types of tooth resorption defined by Andreasen and Andreasen, the most commonly occurring tooth resorption in canine patients is external replacement resorption which is very rare in people and carries a poor prognosis long term due to its increasing severity and lack of successful treatment options.

The American Veterinary Dental College (AVDC) established a classification system to categorize tooth resorption in veterinary patients. This system has proven very helpful in feline patients; however, in the evaluation of the 224 canine subjects it was most helpful in tooth resorptive processes involving external replacement resorption and external cervical root surface resorption, but was less helpful in describing certain radiographic configurations and specific locations of tooth destruction resulting in an actual lesion. This classification system was not applicable for any of the teeth affected with internal resorption and only 46.3% of the teeth affected with external inflammatory resorption.

These AVDC tooth resorption classifications are fully defined on the AVDC’s website under nomenclature.

As we realize the frequency of occurrence of tooth resorption in our canine patients, the classification of tooth resorption specifically for dogs may need to be re-addressed as these concerns arise in the future.

**Treatment Options**

Treatment options vary depending on the classification type and the extent of the disease for each tooth with resorption (See the above classification system for dogs and humans). Open pulp lesions are thought to be painful and progressive and may require extraction. Extractions are often difficult due to ankylosis which is the union of bone and tooth structure.

Conversely, subgingival lesions that are only identifiable on radiographs in people have been found to be asymptomatic. These lesions may also be similar in dogs and may not require any treatment at the time of diagnosis but should be carefully monitored in the future with follow up radiographs and clinical observations. These teeth may require extraction in the future if an open lesion occurs.

When treatment options are considered for feline patients not only the type of tooth resorption but the stage of tooth resorption should be considered when planning the appropriate type of extraction. Modified technique extractions such as crown amputations are only applicable in certain circumstances when the tooth roots have undergone replacement resorption and lack a periodontal ligament as well as normal root substance.

**Discussion**

The first step in helping our canine and feline patients that may be suffering from painful tooth resorption is our awareness of the commonality and progressive nature of this disease of the oral cavity and employing proper clinical, radiographic and histological procedures to thoroughly evaluate patients for tooth resorption. Recommendations for care should be based on the most current and available treatment options and always considering what is in the best interest of the patient.
Foundations of Emergency Care
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In the Beginning

Every technician starts as a baby tech. How does one go from deer in the headlights to the all coveted role of lead technician in emergency and critical care? Some say training, but no amount of training can give someone common sense, right? Wrong, you can train common sense. If you train common sense and the employee does not apply it, there needs to be human resource repercussions. Can you train speed? Yes, you can but you run into a dangerous paradox if they become too speedy and some mistakes happen due to the speedup. How do we create a work ethic or a passion for the “why” of everything we do? How do we create a culture of continual learning and growth? How do we get our team to apply the critical care thinking that is the hallmark of good emergency technicians?

Create the Culture to Foster Good Foundations

We have to rail against those who are bent on retaining knowledge and position. We have to ensure that no questions are treated as stupid. We have to give reasoning behind the how and why of what we do. We cannot lean on the old saying, “it is how we have always done it.” We have to encourage those that question our methods. If our methods are truly the best for our patients and protocols, they will stand up to the questioning. We have to promote team members that foster growth with other team members. Do you have a team member who spends time befriending, mentoring or training new staff? It is far less expensive to thank them with a gift card or a raise than to have to hire a new team member when the first does not work out or does not make it past the hazing period.

Don’t allow a hazing period. Period. At my first volunteer emergency position, I was told that a particular supervisor would learn my name after I had lasted six months. That comment well nigh guaranteed that I moved on to a new clinic when offered a position. Create a supportive environment where new employees are mentored instead of shadow shifted and then allowed to fend for themselves.

Create training programs that cover all required skills and knowledge. Be sure that the training programs have clearly delineated paths upward in terms of knowledge and status. Have leaders participate in the training levels to set the standard and create the culture.

Create learning opportunities such as lunch and learns. Encourage the whole team to contribute as nothing pushes home knowledge of a topic like having to present on the topic. Create a safe space for the team to present the first time. This may involve smaller group presentations or more than one presenter or dividing topics to make them more manageable. Those with a fear of public speaking can be encouraged instead to provide training homework or review sheets.

Encourage the team to develop required information that is always kept on them in the form of a “dumb book” or “nerd book”. This not only encourages them to identify the most critical information but also writing the information can help them retain it as they learn.

Critical Thinking for the Masses

Nothing helps with critical thinking more than presenting cases in a group. As you begin associating disease process with their respective cases, the symptoms and associated physiological changes will become more ingrained. While technicians are not called upon to diagnose, we have all had that case where our astute physiological observation made a difference in the doctor’s assessment and treatment of the case.
I will always remember the case where a straight forward single bite wound from the housemate became a treated snake bite as I found echinocytes on a quick blood smear check. We were able to get a jump on the golden period of treatment because of that fortuitous find.

The quickest way to stop critical thinking is to allow a team member to disregard information brought to their attention. For example, a TPR reveals an abnormal temperature or a patient with altered mentation. This may be old news or perhaps expected news or even a presenting symptom. If the team member receiving the information treats it with gravity even if old information, that new staff member is encouraged to report more critical information. If there is something about that information that may be expected or insignificant use it as a teaching experience, but be cautious to praise the find regardless.

Does your hospital round patients? Does your team round patients without doctors involved? One of the best ways to build understanding of disease process and the importance of astute nursing is by having joint rounds sessions. The doctors present the physiological side of the case and the technical staff provides the perfect foil for the case by discussing the important status and nursing care changes. We can discuss urine output requirements all day long, but until we discuss how the kidneys are not producing adequate urine or how the bladder is affected by a blockage in a particular patient, technicians cannot attach the appropriate level of importance to the information. It is significantly easier to watch disease process and it's affect on a specific patient and assimilate that knowledge than to memorize the pathophysiology of a disease.

**Recording Information**

One of the hallmarks of a good emergency technician is the ability to inform doctors, residents, other technicians and owners about the status of their patients. Mediocre technical staff see medical records as a necessary evil, whereas, great technicians see them as a lifeline. Great technicians know that any changes in physiological status could indicate a trend or could indicate a worsening of condition. Are your medical notes complete, dated, initialed and brought to the right person’s attention when necessary? Sometimes it is busy, but medical notes are absolutely a part of the treatment.

**Prioritization**

Priorities are difficult. Do you prioritize by the doctor, by the flow, by the case needs, by the patient needs, by the staffing or by the client imperatives? If you walk in the lobby and ask your clients the greatest need is the ear laceration that bled all over the car interior. We know that the 8-year-old Great Dane that has a distended abdomen is the truly emergent case in our lobby. The answer is to prioritize by patient needs always. In managing in house patients this is true as well. You may have four 2 AM treatments, but they should be done in order of critical status.

Having a lead technician on the floor to handle incoming case assignment (based on technical needs of the case and matching that with technical abilities) and to determine order of go for any procedures or workups is imperative. This prevents a greedy doctor from hogging all of the technicians and keeps the flow focused on the most critical cases instead of those designated critical by the doctor assigned to them.

**Managing Speed of Work**

We all have that one assistant or technician who is slower than molasses. How do we work on an adequate pace without risking mistakes from rushing? We need to start by looking at what is taking so long first. Is that technician getting drug into a full life story by a client? In that case, we need to work with them through role playing and scripts to give them an appropriate way to steer and curb the conversation. Is it a lack of planning? Perhaps they need a surgery checklist to ensure that they can
quickly prep and get a patient induced. Is it inefficient skills? Perhaps they need an IV catheter workshop.

Next, we have to assess how much time they are currently spending on any tasks or treatments that they are doing. Once you establish current time spent. Assess if there is an opportunity to time them without increasing mistakes. Perhaps they don’t go to the cage with all essentials and one treatment takes multiple trips. Perhaps they are spending too much cuddle time are there any fear free techniques that could shorten their time spent. At this point give them a timer and a time just short of their current time spend. Keep shaving time off of their goal each week to speed them up.

Some team members refuse to put any speed into true emergency situations. For example, a CPR code is called and that team member saunters over to the treatment area. For situations like this we use drills to speed the team. A favorite new drill for my emergency team is “hot lava” they have to make it to safety on a counter when someone calls out lava. After we have the result we want in terms of reaction, we change the shout to “code” to elicit a similar speed response.
Recognizing and Responding to Emergencies
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Recognizing emergencies begins before they hit the door. Often it begins before the client even calls. It begins in recognizing the potential for emergencies based on the time of year, the holiday or even local events. It begins when we recognize that in summer fish hooks are caught in lips and dogs are left in hot cars. In winter, poinsettias make our patients sick and tinsel is like cat nip for cats. As we gain experience in our technical roles, we will start recognizing patterns that will help us interpret vague symptoms and recognize emergencies from afar. We utilize our medical knowledge to triage or order the severity of patients coming into our hospitals.

_Triage is a French term for the assignment of degrees of urgency to wounds or illnesses to decide the order of treatment of a large number of patients or casualties._

The phone call

If we are incredibly lucky we will have some idea of what is coming based on incoming phone calls. We may even have the ability to give instructions for care en route. More often our clients may not call before coming in with an emergency. On those occasions when clients do call ahead, we need to have prepared and informed reception staff. Often phone triage is left to a receptionist that has none of the training necessary to recognize emergency symptoms. Either receptionists need to be trained in phone triage or technicians and doctors need to be available to do phone triage on every non-appointment phone call coming in.

Rules for phone triage

- Get a full signalment (including species, breed, age, sex, spay/neuter status, approximate weight, past pertinent health history)
- Get contact information (at least a good phone number to contact the client)
- Anything ingested is a toxin until proven otherwise (there are many things that sound benign but are not, legally a doctor must prescribe inducing emesis or not treating an inappropriate ingestion)
- Ensure that clients bring the packaging of any ingested items
- Advice on ingestions is only as accurate as our knowledge of the ingestion (how much, of what, active ingredients, when, for what size animal, are we sure?)
- If the client thinks it is an emergency, it is an emergency (we don’t need to stop everything for a non-emergency situation, but we do need to give our clients the attention their worry warrants)
- Even if something has gone on for days, worsening of symptoms constitutes an emergency
- Hallmark signs for an emergency should not be ignored

See the VSPN notebook for a great breakdown of common phone emergencies

The arrival

Timely announcement and cataloging of incoming emergencies is critical in our response. True emergency clinics should have a crash area set up at all times. Other practices should strive to set up response areas for any incoming emergencies as they are announced. Our reception team is busy, but they need to be aware of the timeliness of emergency setup and response. New reception teams should be briefed on the difference five minutes of response time can make in the treatment of an emergency.
Emergency response area setup

-IVCs appropriately sized or a variety if size is unknown
-Catheter tape setups
-IV setup
-Monitoring equipment
-Emetics if appropriate
-Crash cart
-Oxygen (anesthesia machine or Ambu bag)
-Intubation tubes if not in crash cart
-Bair hugger or warming equipment
-A gurney for transport from the car

Discuss a game plan for the arrival. If the phone history leaves the impression that the pet is unstable it may need to be rushed to the treatment area. Technicians should free themselves from other duties for the arrival and be on standby. Our reception teams should be versed in obtaining emergency treatment authorization. They need to discuss the pet’s likely needs in terms of supportive care, the potential costs associated (even if in vague terms), code status and next steps in terms of treatment. They can give the clients a reasonable expectation of stabilization time before the doctor is free to discuss their pet's status. While they cannot legally discuss treatment plan without a doctor's orders, they can discuss what general stabilization plans can potentially include. This all needs to happen simultaneously while technicians are assessing the pet and performing medical interventions with the doctor.

Assessing our unstable patient

How do we know if a presenting patient is unstable? Our phone history may give us a clue. Patients with altered respiration or mentation should be considered unstable and critical. Patients with altered vital signs should be assessed by a veterinarian and considered unstable until assessed. Intake clip boards should include a cheat sheet (on the clipboard itself) of vital signs broken by species and patient size so that reception teams and technical staff have a reference for vital signs (including blood pressure) in emergency situations. Included below is a link to a client-friendly explanation of vital signs great for veterinary teams and client use.


We start with an initial assessment ABC meaning airway, breathing and circulation. Immediately upon presentation we will check the respiratory rate. Increased respiratory rate suggests tissue hypoxia, either due to poor blood oxygenation from pulmonary disease or cardiac disease, or poor circulation of oxygenated blood. We will check mucous membrane color. If the gums are pale, this could represent shock or anemia. If the gums are dark red this could be a sign of hyperthermia or sepsis. We will also check the heart rate. Tachycardia (high heart rate) often suggests shock or hypoxia. Bradycardia (low heart rate) can be a sign of heart disease, hyperkalemia, or hypothermia. This should be done alongside the pulse rate to assess pulse quality and synchronicity.

We will have to intubate and start breathing for the patient if in respiratory arrest. We will need to start chest compressions if no heart beat is present. We should be practicing our emergency response and our CPR in between emergencies to create a smooth and quick protocol. We want to smoothly assign responsibilities as we start a CPR response. We should be assessing how many team members we will have available for responses at different times in our day. We may need to educate our veterinary assistants and veterinary receptionists on parts of CPR to ensure timely and appropriate response.
We will need someone to intubate and then manually breathe for the patient, someone to perform chest compressions and a third person to place IVCs and administer medications. A fourth person will act as liaison to the owners and chart times and treatments. Often the receptionist is relied upon to chart the CPR attempt and take information to the owners.

In many practices, I hear the argument that they do not see enough emergencies to warrant CPR drills and training. In reality, there are many clinics that do not see enough emergencies to stay current on their CPR drills or their CPR training. It is these clinics that need drills most for those seldom seen emergencies. Not seeing emergencies often is not an excuse for being out of practice. If you brought a pet to a clinic and the emergency response was lacking and disorganized, how would you feel? Would you understand that they do not see many emergencies or would you be hurt and frustrated?

As we move past initial assessment a useful mnemonic for assessing our emergency patients is A CRASH PLAN. This builds upon the ABC mnemonic and gives us a meaningful order in which to assess our critical patients. Once we have our critical patient past the danger of a respiratory or circulatory arrest, we should also be gathering a complete history from the owners.

A - Airway
C - Circulation
R - Respiration
A - Abdomen
S - Spine
H - Head
P - Pelvis
L - Limbs
A - Arteries and Veins
N - Nerves


In this critical period we want to ensure that the most experienced member of our team is on hand to monitor vital signs including respiratory effort, mucus membrane color, CRT, respiratory rate, heart rate, pulses, blood pressure, EKG and potentially end tidal CO2. The technician will be alerting the doctor to status changes and supplying oxygen, supportive fluids, colloids, medications and potentially vasopressors to increase blood pressure as ordered by the doctor.

This technician will be watching for changes in the patient's shock status. Shock is defined as ineffective delivery of oxygen to the tissues. Prolonged shock can lead to irreversible organ failures. Patients in shock can be in compensatory or decompensatory shock.

In early or compensatory shock the patient’s body makes an effort to improve circulation and oxygen delivery to the tissues. Clinical signs associated maybe include a higher heart rate, pale mucous membranes (as peripheral blood vessels constrict to move blood to more vital organs), increased respiratory rate and bounding pulses.

In late stage shock the body has utilized all resources and compensatory mechanisms can no longer be maintained. Clinical signs may be weak or thready pulses with an increased or normal heart rate, cold extremities and dull mentation from lack of oxygen.
The technician will also be watching for instability in the patient’s respiratory pattern or work of breathing. A dyspnic patient having difficulty breathing should be treated immediately with oxygen (possibly sedation and sometimes intubation). Respiratory patterns associated with dyspnea include prolonged inspiration (often associated with upper airway obstructions), prolonged expiration (usually associated with lower airway obstruction such as asthma or smoke inhalations), shallow breathing or orthopnea (associated with pneumothorax, diaphragmatic hernia, pleural effusion or hemothorax) and a mixed inspiratory and expiratory effort (associated with pulmonary edema or pneumonia). We need to choose our method of oxygen delivery carefully in presenting patients. Oxygen cages will impede our ability to monitor and care for our patients. We must remember that every time the cage is opened our oxygen concentration drops to that of room air and no longer benefits our dyspnic patient. Oxygen masks can stress our patients and potentially expose them to higher concentrations of their own expired CO2. Upper airway obstructions may limit our ability to intubate the patient. Patient fragility or fractiousness may limit our ability to place a nasal canula, nasal catheter or a face mask. Every situation must be assessed to choose the best option for oxygen delivery.

Assessing our stable patient

If our patient presents with no altered vital signs we know that we are dealing with a stable patient. In this case we want to move into obtaining a history and initial assessment in order to treat and head off any problem that could challenge the patient’s stability. We may have a solid phone history on the patient but often the car ride or perhaps the face to face discussion can jog the owner’s memory for additional information. Even in the case of a clear cut diagnosis, we want to get a full history as it may change our treatment options. A mnemonic borrowed from human medicine to assist us in gathering a complete history is a SAMPLE history.

S-Symptoms and signs
A-Allergies
M-Medications
P-Past medical history, injuries, illnesses
L-Last oral intake
E-Events leading up to the injury and/or illness

Plan of attack

Often our lives do not stop when emergencies happen. There may still be animals in surgery or patients coming in for appointments. We may be able to have some things wait but for others life goes on. We need to plan for these situations.
- Know how, who and what to delegate. I say who and what because you want to ensure that you are delegating tasks within your coworker’s abilities and to the comfort of your clients and doctors.
- Establish effective ways of speeding up a coworker whether it be an assistant, a technician or a doctor.
- Establish effective ways and a set of guidelines for interrupting an appointment for an emergency.
- If you have a way of visualizing what procedures need to happen and the “order of go” such as a white board, you can often keep your team informed when you are out of earshot or dealing with a critical patient.

Why discuss snake bites?

Snake bites are a fascinating emergency. The multiple ways that venom assaults a victim’s body are varied and difficult to mitigate. The treatment is complicated, costly and patient specific. The immune process is different from the response to any other toxin out there.

Rattlesnakes are throughout California to the point that you might be as likely to find a snake in your backyard as in the back woods.

Rattlesnakes can be identified by the “nostril” looking pit or sensory area on either side of the head. Unfortunately, this involves being far too close to the snake for identification and should be relied on only in dead snakes. Most rattlesnakes also have a triangular head. This can be imitated by other harmless snakes and may lead to incorrect identification. Imposter snakes may also behave like rattlesnakes when confronted, hissing and vibrating their tails.

For more identification information follow these links:

http://www.californiaherps.com/identification/snakesid/rattlesnakes.html

Rattlesnake bites are incredibly rare when compared to other outdoor emergency situations and rarer still without provocation.

April to October are the months of snake bite season near San Diego according to the California Department of Fish and Wildlife.

How are bites delivered?

- Rattlesnakes fangs are hollow for delivering venom.
- They point forward when striking prey.
- They can be ½-1 inch in length.
- If broken they are replaced and they are shed and replaced monthly.
- The snake controls how much venom is injected.
- Venom is emitted from ducts behind and beneath the snake’s eyes.
- Young rattlesnakes can emit venom up to three times as strong as adults.
- Snakes can strike up to ½ the distance of their body.

First aid for snake bites is a lesson in what not to do

- do not ice the bite
- do not incise the bite
- do not use a tourniquet
- do not use hot packs
- do not use suction on the bite
- do come to the clinic ASAP, time spent on first aid is time that could be spent in treatment
- do immobilize the animal
- do not put yourself in jeopardy obtaining the snake. Remember that even dead snakes can bite (reflexes stay active)!
How bad will the bite be?
Bite severity depends on
- depth of bite
- number of bites
- location of bites
- the amount of venom injected
- size, species and age of the pet
- pet’s sensitivity to venom
- microbial activity in the snake’s mouth
- initial and subsequent treatment

Is it a snake bite?

Sometimes it is hard to discern if there has been an actual snake bite. Dogs and cats sometimes present with non-specific wounds or non-specific swelling. Unless the bite is witnessed, accompanying symptoms have to be taken into account to establish the true nature of the wounds.

It is essential to establish if the wound or swelling is a bite with an envenomation, a bite without envenomation (dry bites constitute 25% of all bites), a bite from a nonvenomous snake, or a non-snake related wound or swelling.

The presence of one or more of the following: bite wounds, rapid onset swelling, pain and edema point to the presence of a snake bite.

Any of the following point to an envenomated snake bite:

- hematological signs
- pain
- impaired vision
- cyanosis
- hemolytic anemia
- coagulopathies (60%)  
- systemic shock
- tissue necrosis  
- neuromuscular toxicity
- renal injury
- weakness
- hypotension
- thrombocytopenia (30%)  
- petechiation
- increased salivation
- echinocytosis (80%+)

What happens to our patient during and after a snake bite?

During a snake bite, bacteria is transferred to the bite wound and the bitten tissue is damaged. Phospholipase A2 in venom creates lysolecithin which causes RBCs to form echinocytes and eventually spherocytes. Spherocytes then may hemolyze or break. Enzymes in the venom cause necrosis by making capillary membranes permeable and allowing electrolytes, RBCs and albumin to flood the site. Swelling from this permeability and from damaged blood vessels can be dangerous to the long term viability of the tissues and can be especially dangerous near the face or airway as it can endanger breathing. This fluid loss to the tissues can reduce total circulating blood volume and cause hypotension for the patient. This in turn can lead to systemic shock. Vasodilation can lead to pulmonary pooling and then to pulmonary edema and respiratory distress.
not corrected, low pressures can impede renal filtration and cause lasting kidney damage. Bradykinin and prostaglandin in the venom cause increased inflammation. Thrombin in the bitten pet converts fibrinogen to fibrin and increases anti-coagulation. Platelet deficiencies then arise from platelet consumption at the bites. Thromboxane A2 in the venom then causes platelet aggregation and activates additional new platelets.

**Treatment of snakebites**

CroFab and antivenin are two specific snakebite treatments that are the most effective form of treatment for envenomations. Due to the incredibly high cost of CroFab and antivenin, it is unlikely to have them on hand unless in a high bite area. They are sometimes available from local hospitals and hospital pharmacies. They can also be obtained from suppliers, but often not in the optimum treatment time (the first four hours). Call the National Poison Center at 800-222-1222 for locations carrying antivenin. Call the Animal Poison Control Center at 800-213-6680 for assistance in formulating your bite plan.

**What is CroFab?**

-CroFab is a newer antivenin approved for use in 2000. CroFab is prepared from the blood of sheep immunized with Western and Eastern Diamondback, Mojave rattlesnake and water moccasin venom. It is lyophized into immunoglobulin fragments. Unlike Antivenin which only has 15-25% effective antibody per vial. CroFab has been compared to the efficacy of 5 vials of traditional Antivenin.

-Average treatment dose is 1.25 vials for dogs per Micheal Peterson’s study of 120 dogs. It does clear the body faster so it is recommended to divide the dose and administer the second portion 2-4 hours after the first. There has been no serum sickness seen with CroFab treatment.

**What is antivenin? (Crotalidae Polyvalent [Equine])**

-Healthy horses are vaccinated with venom from Eastern and Western Diamondback, Central and South American rattlesnakes and fer-de-lance snakes. Globulins are obtained from the blood serum and preserved with phenol and a mercury derivative.

-Antivenin is currently manufactured by Boehringer Ingelheim, Vetmedica and VetOne

**Treatment plans without antivenin**

Treatment plans without Antivenin or CroFab can involve

-initial assessment involving exam, blood work (serum chemistry, urinalysis, platelet count, complete blood count, blood smear evaluation (for echinocytes) and non-invasive blood pressure

-Clipping and cleaning the wound

-IV catheter placement (a long line catheter for subsequent blood work checks is convenient)

-Pain management

-Antibiotics

-IV fluids, blood products and colloids as necessary

-PT and APTT coagulation testing (favored over ACT testing for increased sensitivity)

Contraindicated medications would include

-corticosteroid treatment is proposed to detrimentally inhibit the victim’s immune response, they are indicated in the event of an AV reaction

-antihistamines have no proven benefit save in the treatment of AV reactions and venom is not known to effect the H1 receptors to require antihistamine treatment
-NSAID use is contraindicated as it may potentiate coagulation issues and may affect kidney health.
- Morphine may cause a histamine response and because of that may be contraindicated.
- Antibiotics should be used with caution in snake bites because of antibacterial qualities in venom and recent studies showing a higher infection rate in animals treated with antibiotics.

**Treatment plans with AV or CroFab**

Recommended treatment plans with AV involve

- initial assessment involving exam, blood work (serum chemistry, urinalysis, platelet count, complete blood count, blood smear evaluation (for echinocytes) and non-invasive blood pressure
- Clipping and cleaning the wound
- IV catheter placement (a long line catheter for subsequent blood work checks is convenient)
- Pain management as necessary
- Antibiotics
- IV fluids, blood products and colloids as necessary
- Antivenin or CroFab as necessary to control pain and coagulopathy

**Does this bite warrant Antivenin?**

- Patients with shock, coagulopathy, or worsening puncture wounds warrant Antivenin use.
- Some patients require more than one vial of antivenin for treatment. In fact, smaller patients may need more antivenin due to the high volume of venom per kilogram. Age and species of the snake, location of the bite, sensitivity to the bite and amount of venom injected influence the necessary dosing. Often client finances will heavily influence the number of doses available to a patient. As variables are often unclear, most treatment plans involve administering a single vial and repeating as necessary if financially feasible.
- Average dosage for dogs and cats is one to two vials.
- The “golden period” refers to the first four hours post bite time when Antivenin administration is most effective.
- Does it make sense to administer AV post “golden period”? It will aid in treatment as long as there are still venom components in the blood stream. Venom takes days to weeks to clear from the blood stream.
- AV does not reverse tissue necrosis. It can help with treatment of coagulopathies and pain.

**Antivenin Reconstitution**

- Warm antivenin prior to mixing to aid in complete dissolution
- Add the diluent slowly while rotating vial
- Avoid adding air which can create expensive unusable foam or break down proteins, instead add saline to completely fill the vial and submerge the protein
- Use a rocker to aid in dissolution (it takes 30 minutes to achieve full dissolution)
- Extract solution as it becomes mixed to add to your diluted fluid bag. Adding more saline from your fluid bag as you go
- Rinse the vial 4-5 times to aid in as much as 30 % protein recovery

**Antivenin administration**

Antivenin once reconstituted is added to 100-250 mls of saline and administered over 30-120 minutes. All antivenin administrations should be monitored for reaction at 5, 10, 15, 30, 60 and 120 minutes. Per Dr. Brady the consulting veterinarian at Boehringer Ingelheim (the veterinary manufacturer for Antivenin), using any type of filter for administration is unnecessary. The ideal treatment model is to ramp the fluid administration rate in the initial 15 minutes to observe for reaction.
IMPORTANT: Skin testing for allergic reactions was shown inaccurate in human studies and has proven even more difficult to evaluate in pets.

**What does a AV or Crofab reaction look like?**

- nausea
- hyperemia of the inner pinna (engorgement; an excess of blood in a part)
- fluffing of the tail
- pruritus (intense itching)

Of Note: of 129 dogs in Michael Peterson’s study on CroFab no adverse reactions were seen. 4

2 http://www.cobras.org/snakebite.html
3 http://www.drugs.com/vet/antivenin.html
Introduction
One of the most important roles of the credentialed veterinary technician is to teach clients how to best care for the pets they own. In progressive veterinary practices, technicians are often utilized as the first professionals to communicate with clients during an appointment where they will evaluate the current husbandry of the patient and make recommendations. This presentation will provide technicians with the foundations they need to best understand rabbit and guinea pig care and then teach clients about their unique husbandry requirements.

Basic Anatomy
Rabbit and guinea pig eyes are situated on either said of the head, rather than in the front. This allows them, as a prey species, to be able to have a wide visual field to watch for predators while eating. This also prohibits them from being able to see directly under the nose, which is why they have adapted to select food based on smell and the very tactile information they receive from the vibrissae around the nose and lips. This also explains why, particularly rabbits, do not tolerate being touched around the nose and mouth.

Rabbits and guinea pigs have aradicular hysodont teeth, often referred to as elodont. This means that they have open roots that are continuously growing. The incisors have adapted to be able to cut through rough vegetation with the lower incisors occluding just behind the uppers to form a sharp cutting edge. Rabbit incisors grow approximately 2mm per week. Premolars and molars form a tight row that grinds the food with jaw movements of up to 120 per minute. Without proper roughage in the diet to grind these teeth, they will have crown overgrowth as well as root elongation.

The stomach of rabbits and guinea pigs comprises about 15% of the volume of gastrointestinal tract. The postprandial pH of the rabbit can fall to 1-2, and guinea pigs approximately 3, both very acidic. This effectively sterilizes ingesta before it passes into the GI tract. Digestion and absorption of nutrients in the stomach and small intestine are similar to that of other monogastric animals, however this all changes in the cecum. In rabbits, at the junction of the ileum, cecum, and proximal colon, food is separated with the large particles of indigestible fiber sent distally along the colon and small fermentable particles and fluid sent proximally into the cecum where bacterial fermentation takes place. The rabbit cecum holds up to 40% of the GI content, while guinea pig cecum's hold up to 65%. In rabbits, the large, indigestible fiber is rapidly eliminated as hard, dry fecal pellets while the particles in the cecum undergoing fermentation release volatile fatty acid and synthesizes proteins and vitamins. This product is expelled generally in crepuscular hours as a soft, mucus covered clump and consumed directly from the anus. This re-ingestion of cecotropes is a vital source of nutrients. This process in rabbits is called cecotrophy; the consuming of cecotropes, which differ from feces in their nutritional richness. Guinea pigs differ in that they do not produce cecotropes, but are coprophagic; ingesting their feces directly from the anus several times a day. The nutritional contribution of this process in guinea pigs is not fully characterized, however it is an important function and when coprophagy is prevented in guinea pigs, they lose weight, digest less fiber, and excrete more minerals.

Nutrition
Fiber is the critical component of rabbit and guinea pig diets. Indigestible fiber (that passes past the cecum to be excreted in rabbits) is important for stimulating gut motility, providing foraging enrichment to prevent boredom behavioral issues such as fur or carpet/plastic chewing, providing proper dental wear and exercise, and to stimulate the appetite and ingestion of cecotropes. In the wild, rabbits and guinea pigs will strip and eat bark, chew roots, and consume dried fibrous vegetation. Captive adult rabbits and guinea pigs have a higher requirement of indigestible fiber to prevent motility issues and prevent obesity. This should be provided in the form of ad lib grass hay. Commercially, this is sold in several varieties such as timothy, orchard, brome, oat, and meadow. A combination of these hays should make up 70-80% of a captive rabbit’s diet. Guinea pigs digest fiber more efficiently than rabbits and can consume less grass hay, however it should still make up 40-50% of their diet.
Alfalfa, clover, and peanut hay are examples of legume hay. These types are very palatable for rabbits and guinea pigs. Legume hay has approximately 21.2% protein, while grass hay averages 10.8%. This is significant when choosing hay for a juvenile or geriatric pet with higher protein requirements. Legume hay also has about 3 times as much calcium as grass hay. This is important because rabbits and guinea pigs excrete calcium primarily from their urinary system, almost 60%, as opposed to other species which are approximately 2%. High dietary calcium may predispose them to bladder stones and calculi. Grass hay is also higher in fiber than legume hay, therefore regardless of the age of the pet, it is not recommended to feed exclusively legume hay. Ideally, rabbits and guinea pigs should eat mostly a combination of grass hay with supplemental addition of legume hay in limited quantities.

**Fresh vegetables**

Green leafy vegetables and edible plants provide micronutrients and important enrichment. Most have high water content while having low calories, making them a good addition. While there is some fiber in many greens, the content is insufficient to meet their needs and should not be used as a substitute for hay. Suitable vegetables include collard, dandelion, carrot/beet/broccoli tops, herbs, lettuce, chicory, chard, endive, and others. Approximately 2 cups per 5 pounds a day is appropriate. Fruit and carrots should be fed very sparingly if at all because of the high sugar content and potential for hindgut dysbiosis due to carbohydrate overload.

**Pellets**

Digestible fiber that is smaller in size and digested in the cecum is vital to provide a substrate for cecal microflora, provide optimal cecal pH and volatile fatty acid production, prevent proliferation of pathogenic bacteria in the cecum, and to increase fiber content of cecotropes. Commercially made extruded or pelleted diets are made primarily of hay, but also include minerals, vitamins, carbohydrates, and other fillers/products. Some are made of grass hay, others primarily of alfalfa hay. Mixes made of "mixed rations" that contain flaked or rolled oats, dried fruits, corn, or other carbohydrate rich foods should be avoided. Ideally, Rabbits and guinea pigs should eat a high percentage of grass hay plus a wide variety of fresh green leafy vegetables with grass-based pellets fed in limited quantity based on body weight to prevent obesity.

**Vitamin C**

Guinea pigs lack l-gulonolactone oxidase, an enzyme involved in the synthesis of ascorbic acid from glucose. This means they, like humans, require a supplemental source of vitamin C or they will be susceptible to scurvy. Healthy adults require 10mg/kg/day. This can be provided by choosing vegetables and limited fruit that are high in vitamin C such as kale, red peppers, or strawberries. Oral supplements can also be used with preparations that come in tablet/treat form or that can be used in the drinking water. Many guinea pig pellets are made with stabilized vitamin C and can be used as directed.

**Water**

Rabbits and guinea pigs drink 10-12% of their body weight in water daily. Providing a fresh, clean water source is mandatory for optimal gastrointestinal health, as dehydration is a major cause of ileus. There are pros and cons to offering water via a sipper bottle vs. a deep, heavy crock. Crock is easier for them to drink more quantity faster than licking a sipper bottle, which may encourage them to drink more. However, rabbits like to push and spill dishes of water, and guinea pigs like to urinate and defecate in them. Also, rabbits with large dewlaps are susceptible to moist dermatitis when the dewlap hangs in the water dish. Sipper bottles may be cleaner, however, it takes a lot of effort for the animals to obtain small amounts of water. Sipper bottles can leak or become blocked, leaving the animals with no water available until owners notice. They also are difficult to properly clean. One option would be to offer both.

**Housing: Rabbits**

Rabbits require an abundant amount of time and space to run and jump. They are crepuscular, being most active in the early morning, or late afternoon. Having an enclosure is a good idea to protect them from possible dangers in the home while not being monitored, but it’s important that clients are made aware that they should not be considered caged pets. Dangers in the home include eating potentially toxic plants, lead paint, or chewing on electrical cords. Puppy gates can be used to prevent access to high risk areas when rabbits are not in an enclosure. Rabbits will seek out a corner or unique area to
urinate and litter boxes should be placed in these areas around the house. Use of feline litter is
dangerous and only recycled paper products and/or hay should be used. While rabbits will mark territory
by leaving dry fecal pellets around, they are very neat about urinating in a litter box when provided. If an
enclosure is required, it should be large enough to accommodate a litter box and something soft such as
good ventilation. A natural prey response rabbits have is to bolt and hide when frightened, generally
underground. In captivity, rabbits will appreciate a place to hide, such as an overturned cardboard box to
be made available both in the enclose and when they are out.

Rabbits and guinea pigs, like cats, are fastidious groomers and bathing is not necessary. Brushing
however, is encouraged. Unlike cats, rabbits cannot vomit hair balls and the less hair they ingest while
self-grooming, the less likelihood hair will slow down the GI tract. High fiber in the diet will also help
prevent this. They also use their hind legs to clean their own ears, although lop-eared rabbits can have
difficulty with this. Also, as rabbits age, arthritis can make this difficult and cleaning the ears for them may
be necessary. Toe nails should be groomed as needed, approximately every 3 months, depending on
exercise and substrate.

Most people are not aware that rabbits are very social animals and enjoy playing. They like throwing toys,
scratching hay pads and mats, digging, and interacting. Ideally, rabbits should be housed in groups of at
least two. This provides them with companionship and enrichment in their lives. Rabbits will groom each
other, cuddle, and play. Providing them with ways to entertain themselves is an important part of rabbit
ownership. They can have bunny agility courses, shelves/climbing furniture, and a myriad of foraging and
enrichment toys to keep them happy.

If a rabbit is going to be housed outdoors, it is important to prevent escape by digging, jumping or
climbing. Rabbits are capable of digging deep, long burrows and sunken fencing will be required. Also,
predator-proofing is required. Raccoons, foxes, coyotes, large raptors, and other hunting species will all
attempt to prey on rabbits. In warm climates, protecting rabbits from flies and mosquitos with netting is
ideal, however may be difficult outside. Owners should be aware of the diseases insects can spread such as
myxomatosis, as well as the risk of myiasis. And, while rabbits tolerate cold better than heat, they must
have shelter, warm bedding, and potentially a heat source in cold winters when temperatures go below
40°F. Both rabbits and guinea pigs are highly susceptible to heat stroke in temperatures above 80°F. Use
of marble or other stone blocks for them to lay on, air conditioning, fans, or even ice blocks are important
to have available on hot days.

Housing: Guinea Pigs
Unlike rabbits, guinea pigs do well in cages for most of the day, however many do enjoy time to roam
and explore outside of their environment. A well socialized, confident guinea pig will enjoy time with human
companionship and exercise/play time. Supervision while outside of the enclosure is very important;
guinea pigs are small enough to get lost, stuck/trapped, and like rabbits they will chew and eat plants and
wood that could be dangerous. Guinea pigs do best in groups, especially when introduced young.
Neutering males will often help prevent dominance fighting in adulthood. Enrichment for guinea pigs can
include same species companionship, foraging games with favorite foods, agility games, low level
climbing areas, and more.

Most are very shy and may prefer their enclosure, which is why it’s very important to provide them with
ample room to run, play, and relax. Enclosures should be large, with enough space to have at least one
hiding box, food and water bowls, and space to run. Like rabbits, guinea pigs should never be kept on
wire flooring. They need soft bedding/substrate such as Carefresh® recycled paper bedding, old towels,
and lots of grass hay with a wire caged siding and roof. Scented shredded wood such as pine or cedar
should be avoided and can cause respiratory problems as well as cause abrasions on the eyes and
mouth from the sharp edges. Guinea pigs enjoy fleece blankets and sacks to lay inside and on top of.
Hiding boxes can be varied with cardboard tubes or boxes, plastic huts, mounds of loose hay, or other
creative areas for them to be able to have privacy. They will spend most of their day relaxing in privacy and not having a hiding area can be very stressful for them. Water should be offered from a low, heavy bowl as well as a sipper bottle. Owners should be aware that guinea pigs often defecate in water dishes and often inject chewed food into sipper bottles, so frequent cleaning is important.
Avian and exotic veterinary practices face many unique financial challenges that dog and cat hospitals do not. Some of these challenges are impossible to control, however there are ways in which to creatively increase profit by utilizing staff, in particular veterinary technicians. Properly educated veterinary technicians are an untapped resource in most veterinary hospitals. Despite the many frustrating limitations facing avian and exotic practices, educating and utilizing technicians to their fullest capacity can help these businesses not only survive, but be profitable.

Challenges
The first step toward utilizing technicians well is to understand some of the challenges that are unique to exotic pet practice.

- Avian and exotic exams require more time than dog and cat exams. There are complex histories to obtain and often physical challenges for the exams such as requiring sedation.
- Clients often to not value the bond they have with exotic pets as much as they do their dogs and cats, which diminishes compliance.
- Many exotic pets are purchased for children rather than for the whole family, so the adults who make decisions and pay the bills have little connection with the pet.
- Some exotic pets do not have very long lifespans making a financial investment less logical.
- These pets do not usually require vaccines or flea/tick/heartworm prevention, so it is common for exotics to present for the first time at a vet when they are very sick.
- Exotic vets often are unable to perform the tests that most dog and cats vets earn their highest profits from due to the size of the animal.
- Profit-building services such as preventative dentistry, microchipping, vaccines, heartworm/flea prevention and grooming are not a big part of the avian and exotic practice.

In order to combat these problems, exotic pet practices need to find ambitious, intelligent people who are interested in more than just being a technician. Avian and exotic technicians need to want to teach, enjoy client communications, and have a lot of patience. Most licensed technicians already have a general medical background but need to be trained in avian and exotic medicine. Working with exotics requires techs to have the ability to understand the huge variety of medical conditions involved with exotics. This process will take time and some financial backing for continuing education, but is worth the investment.

The following are examples of how properly trained technicians can improve profitability and client service.

CREATE A POST-PURCHASE EXAM SERIES FOR PET BIRDS
The goal of an avian post-purchase series, much like a puppy or kitten series, is to educate new bird owners over the course of several weeks. Many bird owners are new to birds, and there is far more information to give them than can be done in a 20-30 minute time limit. Having clients bring their bird back a few times will not only create a better veterinary-client-pet bond, but will help decrease fear and stress of the bird associated with traveling and restraint. By putting technicians in charge of the educational follow-up consultations, veterinarians will be free to see other appointments. The clinic will profit by charging for each follow-up instead of answering common questions over the phone or email for free.

Here is one idea of how a post-purchase series could be organized:

Visit 1: Exam, Testing, and Nutrition
This visit introduces the client to the veterinary team. They will fill out paperwork and history forms. The veterinarian will perform a physical exam and recommend any preventative or diagnostic testing. Once samples are acquired, the technician will complete the visit to discuss all of the aspects of nutrition that are pertinent to that individual bird such as feeding pellets vs seeds vs human foods vs treats, vitamin/mineral supplementation, and ideas for dietary enrichment
The technician should give the client any nutrition handouts and make recommendations for purchasing nutrition related products. The client should be given homework, such as a project regarding foraging and improving nutrition. The next appointment is scheduled with a technician in two to three weeks.

Visit 2: Husbandry and Behavior
The purpose of this visit is to review the homework given during the prior visit and answer any questions. Weigh the bird to ensure there have been no changes during a potential diet transition. The technician then proceeds to discuss specific husbandry requirements and behavior traits common to the specific species. This discussion will include but not be limited to:

- Suitability of cage sizes, shapes, and locations in the home
- Importance of a variety of cages/play stands
- Cage furniture, perches, toys
- Foraging and the importance of incorporating it into the birds life
- Exercise
- Harness training/getting birds outside
- Lighting and air quality
- Grooming needs- pros and cons of trimming wings
- Screaming, biting, territorial behavior, and the benefits of positive reinforcement training
- Sexual maturity and what to expect
- Inform the client of the hospitals behavior resources should the need arise

The technician should again give any pertinent handouts and a homework assignment regarding the visit: redesigning cage, foraging exercises, positive reinforcement exercises, training, etc. The technician should also make recommendations for products the client should purchase, such as books or DVD’s regarding behavior and training. Set up final visit with a technician in two to four weeks.

Visit 3: Common Health Issues and Prevention
The technician should again answer any questions or concerns that may have come up in the previous weeks and review homework. Re-weigh the bird. Practice some behavior tricks, hopefully from homework. The technician then proceeds to discuss common health concerns with the particular species involved.

- Reproductive concerns and how to prevent egg laying/egg binding.
- Disease transmission, quarantine recommendations for additional birds, and the dangers of boarding/grooming facilities
- Hygiene and how to clean the cage
- Signs of illness
- When is it an emergency vs. a scheduled appointment
- How to set up an emergency kit and plan ahead for potential disasters
- Household dangers
- Veterinary pet insurance, the availability of programs such as CareCredit® and how to financially prepare for unanticipated problems/costs.
- Preparing family members for changes in the pet and family dynamics whether related to normal development, disease, injury, “sibling rivalry”, death, moving, a new baby or additional pets, and more.

This appointment concludes the post-purchase series. An avian “wellness” folder should have all records and pertinent information, perhaps a photo of the tech with the bird!

This post purchase series is one way of creating an informed and educated client, enabling the technicians to get involved on a much more personal level, and profit-building that is beneficial to everyone. These clients have learned more about their bird and the services the hospital offers, bought more products (that are justifiably helpful to their pet), and have a close bond with the staff.
USE TECHNICIANS TO REVIEW COST ESTIMATES
Technicians can increase revenue not only for wellness exams, but also for problems. Consider having technicians review all treatment plans and estimates with clients. This will allow the doctor to move on to other things, while separating them from the financial aspect of medicine. Having an exotics-trained technician review the cost estimates rather than a receptionist, will provide better explanations of the recommended services and increase compliance. It will also give the client another valuable staff member to bond with.

TECHNICAL PROCEDURES
Technical procedures should be performed by technicians. Often veterinarians are nervous about technicians working on exotic animals due to their delicate nature. It does take time and experience to become adept at working with exotics, and CE opportunities with wetlabs will help with training. Procedures technicians should be doing independently include phlebotomy, cytological collection, radiology, wound debridement/care, bandaging and splinting, calculating drug dosages, dental procedures (ferrets, rabbits, and rodents depending on the nature of the problem), grooming, enemas, and more. Technicians are only limited by their training and the hospital’s protocols.

BEHAVIOR CONSULTATIONS
Once the veterinarian diagnoses a behavior problem, instead of the doctor speeding through a list of books and websites for the owner to read, they should recommend an appointment with a technician who has had the proper training. These could include home visits, or a series of consultations depending on the problem. Behavior problems are not limited to birds with feather-picking, screaming, and biting. Any time clients have questions about reproduction, changes in behavior, or even simple questions about what is normal for their pet, rather than doctors and technicians spending time on the telephone for free, a consultation should be scheduled. Fighting rabbits and rodents, rabbits chewing on cords, and ferrets urinating inappropriately are all potential behavior problems that technicians can help clients with. There are many continuing education opportunities available to technicians to become adept and even specialize in behavior. Clients will appreciate the knowledge they will gain and they will value it more if they pay for that information.

LABORATORY SKILLS
Reference laboratories charge significant fees for testing that can and should be done in the hospital. Many of these tests are easy to perform, do not take a lot of time, and are often misread or suffer changes by the time they get to the laboratory. By training technicians to do simple in-house testing, the hospital will save money, and get faster and more reliable results. Tests that technicians can be trained to perform include:
- Fecal examination
- CBC estimates and morphology evaluation
- Skin cytology
- Crop/cloacal cytology
- Mite checks
- Urine analysis

FEE-BASED NURSE APPOINTMENTS
There are frequently instances where doctors recommend that clients schedule a technician appointment for a service such as an injection, suture removal, or checking a bandage at no additional charge other than the treatment. Often these courtesies are appreciated by the client and build compliance and trust. However it is also an example of a time when clients can have many questions and concerns that require staff time, often hoping the doctor will “take a quick peek”. Most clients will respect the technician’s time better and recognize that they have received superior attention and care if they get to sit down with the technician and have their concerns addressed. They will pay a small fee for this professional effort. Consider differentiating when a technician appointment should be paid for, or given away as a courtesy.

Examples of when not to charge additional:
Assuming the client does not have several questions: injection series (Doxycycline, ivermectin, leuprolide acetate, etc.), grooming, suture removals, hospital discharge instructions.

**Examples of when to charge additional:**
Broken blood feathers, teaching clients (grooming, injections, subcutaneous fluids, handling, restraint, physical therapy, syringe-feeding), e-collar fitting, behavior consultations, husbandry consultations, and more!

**PUBLICITY AND SOCIAL MEDIA**
There are many ways that hospitals can get their names and services publicized while educating pet owners. Local pet stores, schools, expos, and organizations (clubs) servicing exotic pets usually love the opportunity to have technicians teach seminars on basic animal care. These seminars can be given at the hospital, a pet store, or a location chosen by the organization. Technicians could put together talks about nutrition and husbandry, normal and abnormal behavior, common illnesses, etc. These events can be sponsored inexpensively and are perfect venues to encourage pet owners to have wellness exams, as well as let them know where to go when their pet gets sick. This could be enhanced by appointing technicians in charge of various social media outlets. Facebook, Twitter, Instagram, or blogging can all be fun interactive ways to get information to clients while garnering their interest in the practice.

**PET SITTING AND BOARDING**
Technicians are often asked to pet-sit for client pets, either in the client’s home, at the hospital, or at the technician’s home. Hospitals should develop a protocol for offering these services to clients. By implementing pet-sitting as a hospital service, clients will feel secure the technicians can be trusted, and should a problem arise, the pet will be well cared for. Since the facility’s reputation is associated with a technician employed at the hospital, the hospital should develop a policy regarding technicians who pet-sit that could include the mandatory length of employment of the technician, compensation, emergency protocols, and boarding monitoring sheets.

If the hospital has a boarding space, the technicians can be utilized to create an environment which will make clients feel comfortable leaving their pets in their care. Consider giving small foraging toys or healthy treats to the pets when they are discharged, along with a “report card” which should include appetite, weight, and behavior while boarding. Owners love getting photos of their pets while they are away. These specialized services have tremendous value to people who want the best for their pets. Most people will be happy to pay for high quality care.

**CLIENT COMMUNICATION**
Technicians can also be used to write newsletters, handouts, and website material for the hospital. Literature is vital for client communications, and monthly newsletters that are emailed or mailed to clients achieve several valuable profit-building purposes. Topics should be informative, while reminding the owner that they should be coming to the hospital for various reasons. Some suggested titles are: “Importance of Post- Purchase Testing”, “Common Zoonotic Diseases”, “Why Yearly Exams Are So Important”, “Proper Quarantining for New Pets”, “Common Signs of Illness”, and “A First Aid Kit for Your Pet.” These topics are meant to inform clients and potential clients while helping them to understand the importance and the value of what avian/exotic veterinary hospitals do.

**HOUSECALLS/PICK-UPS**
Many clients are nervous about bringing their exotic pets out in foul weather, or are uncomfortable with catching their pet and transporting them. Technicians can make housecalls for grooming, to help place pets in carriers and transport them to and from the hospital, and for behavior consultations.

These ideas are just a few ways that veterinary technicians can increase profitability in the avian/exotic practice. Credentialed technicians are highly trained, qualified professionals who are capable of making the difference these unique practices require in order to be successful and profitable.
Nursing Management of Rabbit and Parrot Emergencies
Lorelei D’Avolio, LVT, VTS (Exotics), CVPM

Introduction
Some of the most common patients currently seen in exotic pet practice include parrots and rabbits. Among these species, there are some trends in what constitutes a real emergency. Technicians are often the first ones evaluating these cases, and they should be prepared to not only identify what the emergency is, but be able to communicate the urgency to the client and doctor. Technicians should be able to prepare possible therapies and treatments, handle fragile animals properly, and anticipate the needs of both the doctor and the patient. Understanding what may be causing the emergency is vital to being able to achieve these nursing skills.

Identifying a True Emergency
While clients may be confused and scared about their pets’ condition, some of the key indicators that these pets should be seen right away include: trauma, bleeding, respiratory distress, anorexia, seizures, or reproductive distress. When these patients arrive, technicians should be cautious. There is hardly ever an instance where one of these animals should be rushed to a treatment room with traditional emergency care initiated, such as IV catheter placement, blood pressure assessment, CPR, etc. As prey species, they need to be treated differently than a dog, cat, or ferret to prevent a stress response that can result in death. Technicians should be calm, gentle, and quickly assess the history to identify the nature of emergency, often not touching the animal until they have a general idea of what is wrong and can get the animal to a quiet place where heat and oxygen are available.

PARROTS
Respiratory distress
Triage and history are crucial to determine the possible cause of distress. In birds, difficulty breathing can be caused by several factors including upper or lower airway disease caused by fungal or bacterial infection, space occupying masses such as reproductive cysts/cancers/eggs or liver enlargement, inhalant toxicities such as PTFE (non-stick cookware), air fresheners, or smoke, aspiration, or heart disease. Obtaining a simple and fast history from clients often reveals the potential cause, or at least rules out obvious non-factors. For example, if it is a young, male bird that was just purchased from a pet store, the chances are the respiratory distress is not originating from reproductive disease or a primary coelomic mass and more likely something infectious or environmental.

Sometimes handling alone of these panicked patients can cause death. These birds should be placed in a warm (80-85 degree f) enclosure with humidified 50% oxygen. Some birds may not respond to oxygen, which could indicate a tracheal obstruction requiring an air-sac canula be placed. Others will noticeably calm down, potentially allowing for a limited exam, SPO2 monitoring, weight, supportive care needs, and diagnostics.

Trauma
A traumatic injury can cause many symptoms and it can be tempting to attempt to address the tissue wound as the first priority. However, unless the bird is actively hemorrhaging, stabilizing the patient must be the primary intent. Some common accidents that happen to birds in captivity include flying into mirrors/windows/ceiling fans/hot water, bites or scratches from other animals including other birds, being stepped on or crushed in a door. Technicians should try to obtain information such as when the accident happened, if the owner tried any “first-aid” at home, and if there are any concurrent medical conditions the owner knows of. Sometimes owners think the bird hit a window and that caused it to collapse to the ground, but a thorough history may show that the bird had been acting strange for several days and hitting the window was secondary to weakness.

If the patient is not bleeding, technicians should prepare an enclosure that is safe and comfortable for an injured bird. Consider a dark padded enclosure, low or no perches, a variety of easily attainable food items, and heat support. Analgesics should be a primary consideration as well as the potential to treat for...
shock if required. Analgesics could include butorphanol, meloxicam, lidocaine, gabapentin, or LLLT (low level laser therapy). A prepared technician will be ready for obtaining radiographs, cleaning wounds, giving fluids, splinting/bandaging, and persistent monitoring of behavior to address changes as they occur. General anesthesia may be required for many of the repairs needed in traumatic cases, and technicians should have all equipment ready to use.

**Bleeding**

There are many causes of bleeding in birds, and while not all are traumatic, all should be treated immediately. Some common causes for bleeding in pet birds are broken blood feathers, chipped beak tips, at-home-nail-trims, wounds (self-inflicted or other), trauma, or from the vent which can include lead poisoning (hematuria/hematochezia), reproductive, or papillomatosis.

Unlike other emergencies, bleeding should be addressed quickly, especially if originating from obvious tissue instead. Often owners know what caused the bleeding such as in the case of trauma. If the bleeding is in the droppings, owners should be asked about the potential for chewing on lead items such as old painted windowsills or furniture, antiques, stained glass, old bells, zippers, or jewelry.

Technicians should be prepared with appropriate methods for cautery. This could include applying manual pressure, or preparing electrocautery/penlight, quick stop, or other hemostatic agents. They should be ready to clean wounds, administer prescribed analgesics, antibiotics, and supportive care. Technicians can also be instrumental in helping owners prevent future incidents by educating them about safety for birds in the home. They should take the time to investigate and come up with a plan that the owner can put into effect to avoid further accidents.

**Anorexia**

Anorexia is an emergency for birds, particularly if it has been going on for more than a few days. These patients require technicians to triage by obtaining more of a history than with some of the other emergency presentations. Information such as age, last time witnessed eating, what food is offered, quality of droppings, other bird or pets in home, and other signs of illness should all be obtained. Some of these birds may present severely weak and unable to perch, others may have fluffed feathers and looked depressed. If the bird is not dyspneic and allows gentle handling, technicians should assess baseline parameters such as a BCS, HR/RR, weight, and hydration status.

Almost anything can cause anorexia in birds. Etiology could be infectious, metabolic, environmental, or neoplastic. Therefore, diagnostics will be mandatory. Technicians will be required to have exceptional skills to gently assist with restraint, phlebotomy, sedation, and radiology. Nursing care of these birds will include creating a comfortable enclosure with padding, heat and humidity support, a variety of favorite foods, and limited low perches. Technical skills may also include placing IV or IO catheters, administering fluids, gavage feeding, and diligent patient monitoring.

**Reproductive**

Female birds may present on emergency in a variety of reproductive emergencies. They may appear to be in respiratory distress, or be bleeding, or have a prolapse, or even a combination of these problems. Sometimes clients may not even know the gender of their bird, or they may not understand that a parrot living alone could lay eggs. When triaging these birds, technicians need to obtain the vital history needed to determine if reproductive disease may be causing the problem the bird is having.

Often, these birds will have an egg distal enough in the oviduct to be able to be palpated in the coelom. If the bird is not stable for anesthesia or sedation, taking a radiograph of a bird either with a lateral beam or DV while standing can help give information about egg size and placement. These birds need to be set up in a warm, humid environment that is quiet, dark, and comfortable. Often vets may prescribe vitamin D3, calcium, vent lubrication, analgesics, and fluids to help support the bird to potentially lay the egg without assistance.
RABBITS

Anorexia/GI Stasis
When rabbits don’t eat, even if only for 12 hours, profound changes can occur in their gastrointestinal tract that warrant an emergency visit to the vet. Technicians should be specific during the triage to determine age, gender, usual diet including quantities and specifics about what makes up the pellets, defecation, other pets in the home, spay/neuter status, history of stasis or other illness, and chewing habits in the home. Most of these rabbits are stable enough to obtain vital signs: TPR, abdominal palpation, pain score, and dental assessment. Rabbits who are hypothermic (under 100 degrees f) should be treated with slow warming quickly. A tense, bloated abdomen is indicative of painful gas and veterinarians should be alerted to the likelihood of pain. Dental abnormalities, including the cheek teeth, should be described. This information can be easily taken by a technician in a 5 minute triage and will help the veterinarian pinpoint their diagnostic and treatment plan.

There are many potential causes for anorexia in rabbits. Pain, parasites (external, intestinal, or in the blood), poor diet, concurrent illness (reproductive, dental, urinary, or respiratory disease), primary dehydration, trauma, age/arthritis, or lead poisoning/toxicity are all common etiologies. Until the instigating cause is determined, technicians need to be proactive in their nursing care. Some important considerations when rabbits aren’t eating are to prevent ileus and gut stasis. This can be through encouraging exercise or administering prescribed prokinetics. Preventing or treating pain is vital if there is gas building, and rehydrating the rabbit with crystalloids is usually an initial step. A rabbit’s temperature should be between 100° and 103° f, and technicians should manage hyper or hypothermia. Setting these rabbits up in an enclosure that is quiet, appropriately temperature controlled, not too bright, soft, and maybe with a hiding spot are important parts of keeping the rabbit calm. When these primary concerns are addressed, it may be safe to begin the diagnostic work-up.

Seizures/Neurologic Disease
When triaging these rabbits, technicians should determine if the pet is “stable”: can it stand or is it spinning uncontrollably or obtunded. Immediate questions for the owner should include the duration of the symptoms, if there is a history of possible toxin exposure such as eating paint or use of parasite control at home, age of the rabbit, whether or not the pet has been able to eat, and if there are other rabbits or pets in the home. Technicians should take a TPR (temperature, pulse rate, and respiration rate), look for signs of trauma, and evaluate the ears.

Causes of neurologic emergencies in rabbits include Encephalitozoon cuniculi, a microsporidial parasite classified in the kingdom Fungi that can damage brain tissue causing severe vestibular disease and seizures. A severe ear mite infestation or ear infection can cause similar symptoms as well as torticollis and spinning. Toxicities such as the use of Fipronil (Frontline ™) or lead poisoning can also present with neurologic signs. Other potential causes include spinal trauma, brain tumors, or stroke.

In an emergency situation while waiting for diagnostics, these rabbits should be cared for by treating the symptoms. Hospital caging should be padded well and free from things that may cause stress or trauma such as leaving hay with a spinning rabbit that may cause corneal ulcers. These rabbits are at high risk for developing gastro-intestinal stasis if they don’t eat and become dehydrated. Technicians need to support them with fluids, orally and parenterally, prokinetics, assistance feeding a variety of high fiber palatable foods, analgesics when warranted, and potentially anti-vertigo drugs such as meclizine.

Respiratory
Because rabbits are obligate nasal breathers, respiratory distress is a real emergency. If at triage, technicians see a rabbit breathing through the mouth, putting it in a quiet, calm, darkened enclosure with oxygen may be immediately warranted. Technicians should look to see if there is nasal discharge, as well as sticky matting on the front paws from wiping the nose. Questions about appetite, defecation, and duration of distress are all important to gather. If the rabbit is stable, checking for a TPR and assessing the color of the mucus membranes will be helpful.

As well as providing supplemental oxygen, cleaning the nose is important, although care should be taken as rabbits generally become very stressed when people clean tor touch their noses. Stress while having
trouble breathing can easily cause respiratory arrest in rabbits, so any diagnostics should be taken with extreme slow, gentle care. Do not keep these rabbits near loud animals or other noises.

**Heatstroke**
Rabbits do not sweat or pant, making them highly susceptible to heat related fatalities. Signs of heat stroke include red ears, open-mouth breathing, nostril flaring, salivating, recumbency, and convulsing. The history may be the most important part of the triage for these rabbits. Owner should be questioned about the environment at home. If the rabbit was kept in an area that was over 80°C without fresh, circulating air or a cooling stone, heatstroke should be suspected. Also a lack of a water source, lack of shade, and an unkempt hair coat may be clues. If all of these signs and historical details are evident, and the temperature is over 104°F, the rabbit most likely is suffering from heat stroke.

Like with other species, cooling should be immediate, but not shocking. Cool wet towels applied to the body, subcutaneous or intravenous fluids at room temperature, applying alcohol to the ear pinna, and syringe feeding cool water (if swallowing) are all techniques technicians can use.

Technicians should also be sure to check for fly strike in these patients, as it is common in hot weather for rabbits to succumb to myiasis or maggots infesting a wound. This can be terribly painful and create tremendous stress for rabbits. Technicians should be diligent in examining the scrotal region and vaginal folds as well as the ears and dewlap.

**Trauma**
Rabbits that present with trauma could be completely stable, or in shock. Technicians should gather information about the nature and timing of the trauma from the client. The history should include any known changes in appetite or defecation, and technicians should obtain a TPR, blood pressure, as well as evaluate the mucus membranes for color and capillary refill time to evaluate shock. Any rabbit suspected of shock should immediately be brought to the attention of a veterinarian to decide the protocol. Rabbits may require IV fluids, analgesics, oxygen and its temperature needs should be addressed. All of this must be done using caution not to increase the stress reaction the rabbit may already be feeling.

**Diarrhea**
True diarrhea in rabbits is an emergency. Technicians should determine that it is truly watery stool, not just soft or feces or cecatropes that the owner is seeing. Diarrhea can be caused by dysbiosis, intestinal parasites, or primary intestinal disease. These rabbits should have a PCV checked and be started on IV fluids as soon as possible.
SPAYING AND NEUTERING OF BIRDS
Lorelei D’Avolio, LVT, VTS-Clinical Practice (Exotics), CVPM

INTRODUCTION
Exotic pet veterinary technicians must be prepared to discuss all aspects of reproductive health with clients, as well as to then be able to competently execute the anesthetic and nursing care required for the procedure. While most clients understand the importance of spaying and neutering dogs and cats, most do not consider this procedure for their pet birds. Technicians should be prepared to educate clients about the importance of this procedure in birds. Gender determination is often impossible with psittacines without a DNA gender test. Some species such as budgerigars, cockatiels, or eclectus parrots have sexual dimorphism, however most do not. Clients should be encouraged to determine the gender in order to be prepared for reproductive behaviors. This is not a presentation on anesthesia, but it will cover some key points specific to birds.

Facts about the Ovary and Oviduct
The female birds (hens) ovary develops into a functioning reproductive organ from age 1 year to 7 years depending on the lifespan of the bird. During the species breeding season the ovary will be developed with engorged follicles, and then shrink during the non-breeding season. It is located caudal to the lung on the left side and slightly caudolateral to the left adrenal gland. Birds only have one ovary and oviduct on their left side, with the right side being vestigial or non-existent in most species. The inner part of the ovary, the medulla, becomes highly vascularized and irregularly shaped as it matures. These vessels, along with nerves, supply the ovary, as well as the smooth muscle and interstitial supportive cells. The ovarian vessels, called the ovario-oviductal artery, are very short segments coming off of the abdominal sections of the aorta and vena cava. This complex, highly vascular anatomy is part of what makes removing the ovary of birds difficult and at high risk for significant bleeding.

The ovary and reproductive cycle of birds has evolved to ensure that ovum are fertilized sequentially and travel down the oviduct where they are shelled for external incubation. The follicles enlarge at differing intervals so that they do not mature at the same time, allowing for a period of rest between eggs that varies from 1 day to several depending on species.

Once a follicle is released from the ovary, it passes into the infundibulum. This is where fertilization takes place, if sperm is present. This is also where albumin is first secreted to surround the yolk (follicle). The next region of the oviduct is the magnum, where the majority of the albumin is added. Next is the isthmus where the inner and outer shell membranes are formed and calcification begins. The uterus, often called the shell gland, is next. Here, water and electrolytes are added to the egg, and calcification proceeds. The uterus has the ability to extract large amounts of calcium from the blood stream which requires the release of calcium stored in the long-bones. The egg is then ready to be expelled through the cloaca and out the vent.

Removal of the oviduct in parrots is a difficult procedure, and complete removal of the ovary is currently not considered possible. This is primarily because of the previously described ovario-oviductal artery which feeds the ovary and is very short and difficult to locate and clamp. It is hypothesized that removal of the oviduct alone is sufficient to turn off hormonal feedback to the ovary and stop the formation of follicles. Therefore, the most common procedure to stop a parrot from laying eggs is a salpingohysterectomy, or removal of the oviduct.

Due to the risks and complex nature of spaying birds, most avian vets agree that it is not recommended to routinely spay birds for preventive reasons. But, birds do suffer common reproductive disorders, and technicians should strive to educate clients on measures to prevent reproductive behaviors that can lead to problems. Discussing and addressing these factors with clients may help minimize the most frequently seen reproductive disorders.

- The most important environmental cue for reproduction in captivity is the photoperiod. Long days stimulate the release of luteinizing-releasing hormone, promoting gonadotropin secretion. The most stimulation occurs with 12-14 hours of light. Technicians should encourage
clients to give their birds a minimum of 12 hours of quiet, covered, dark to not be stimulated.

- Many species of pet birds, such as the zebra finch, breed immediately after rainfall regardless of the time of year. Rain also indicates bountiful food supplies. These species may have less reproductive activity in a more arid captive environment.

- Presence of a mate will almost always stimulate reproduction. While technicians should advocate for birds to have flock-mates, having a mated male should be avoided in birds with reproductive concerns.

- Vocalization of a male to a female is very stimulating, such as with canaries. Even if not in the same enclosure, the signing can instigate egg-laying.

- Providing nesting material such as paper to shred, soft bedding, or a box that simulates a nest cavity are reproductive stimulants. With the exception of Quaker parrots, most parrots do not live in nests unless it is their reproductive season and they are breeding. Clients should be advised not to give these birds anything that mimics a nest, including those they find around the house such as a cabinet or dresser drawer.

- Allopreening or petting of the back and rump can mimic copulatory acts and stimulate reproductive activity. Owners should be warned not to stimulate their birds in this way.

- Birds often regurgitate or masturbate on toys or objects in the cage, increasing reproductive stimulation. While clients often feel badly about removing a perceived favorite toy, if they understand that those toys could endanger the health of the bird, they may comply.

- Birds that have appropriate nutrition, including balanced calcium, phosphorous, and magnesium in their diets, have a better chance of being able to lay eggs without problems. Technicians should work towards always educating clients about the best ways to get good nutrition into their birds. This may include a recommended pelleted or extruded commercially available diet, combined with a healthy variety of vegetables, fruits, and grains.

Facts About the Male Testes
The testicles of birds lie deep in the coelomic cavity caudal to the lung and ventral to the kidneys. Male birds have two gonads which lie directly on the iliac vessels where they join to become the caudal vena cava. They are attached to these veins by a short ligament. There is also significant vasculature passing through this ligament providing blood to the testicle. The size of the testicles and the associated vasculature vary with species and the seasonal reproductive cycle. In sexually active birds the testicles are quite large and vascular making castration extremely challenging and risky due to the potential for hemorrhage.

Removal of the testes in parrots is considered extremely high risk with high mortality, and is reserved as a last option to save a life rather than as a means to control undesirable behavior. Removal of the testes requires an experienced and skilled surgeon, highly specialized equipment, and extreme caution to prevent damage to the vena cava, adrenal gland, kidneys, or ureters. The procedure tends to be slightly easier in columbiformes or galiformes due to their comparatively large coelomic cavity and slightly varying anatomy. Another frustrating factor is that if any testicular tissue is left intact, the tissue can regrow. This persistent tissue can behave exactly as a normal testicle would, rendering the entire procedure pointless unless all cells of the testicle were successfully removed.

Surgery
Some of the most common reproductive disorders that would warrant a spay or neuter surgery include the following:

- Chronic egg laying can deplete a bird of vital nutrients, making them lethargic, prone to pathologic long-bone fractures, suffer hernias, and unable to form normal eggs. Common species that are prone to this include cockatiels, budgies, finches, lovebirds, and Aratinga conures.

- Egg binding/dystocia commonly occurs when birds are calcium deficient and unable to either form an egg properly or have the muscle contractility required to evacuate it. Signs include lethargy, inability to perch, and coelomic distension. Often these birds will be straining, bleeding from the vent, and dyspneic.

- Ovarian cysts occur most often in budgerigars and cockatiels. While they are benign,
they can grow quite large or have more than one cyst present causing severe coelomic distension and respiratory distress.

- Prolapses of the cloaca can occur in males or females. Females generally prolapse due to calcium deficiency, physiologic hyperplasia, dystocia, or abnormal egg laying. Males, particularly cockatoos, prolapse from chronic masturbation or straining due to hormonal stimulation.
- Gonadal neoplasia is most common in galliformes and budgerigars. Signs include change in secondary sex characteristics such as cere color, coelomic distension, and unilateral leg lameness.
- Behavioral reasons: This “disorder” needs to be considered cautiously. Females and males may both have undesirable reproductive behaviors such as screaming, biting, fighting, “crowing” in the morning (roosters), or destructiveness in the home. Most of these behaviors however, are in fact not abnormal or a disorder, but rather normal bird behaviors. Before considering risking the life of a bird, owners should be counseled on what are normal and abnormal behaviors, and how they may be able to modify the undesirable behaviors without considering a surgical procedure that may not only be ineffective, but that their pet may not survive.

The success of a long and complicated surgery is often contingent on the nursing care provided. Presurgical testing and supportive care is recommended for both genders. These birds are often already suffering from egg binding, anemia, pain, nutritional distress, or dehydration. Providing them with a quiet, comfortable enclosure in the practice with supplemental heat, away from loud noises is important. If their physiological needs are not met, their anesthesia will be more risky. Technicians should be vigilant to advocate for proper pain management, which should include use of opioids (generally butorphanol 1-2mg/kg q4-6h is preferred due to the prevalence of kappa receptors in birds), NSAID's (meloxicam 1-2mg/kg Q12-24h), dissociative agents (such as ketamine 5-30mg/kg depending on concurrent sedatives given), and local analgesia (lidocaine 1-2mg/kg).

Once sedated, birds undergoing these procedures should have an IV (intravenous) or IO (intraosseous) catheter placed. IV catheters can be placed in the basilic vein of the avian wing, the medial metatarsal vein of the leg, or the jugular vein. For patients whom using a vein is not an option, such as a bird with tiny vessels or vessels already used for phlebotomy, IO catheters are also fast and easy options. IO catheters can be placed in the distal ulna or the proximal tibiotarsus, making sure to use sterile and aseptic technique.

Intubation is easily achieved in birds who have no epiglottis and a large tracheal opening situated towards the back of the tongue. Use of uncuffed endotracheal tubes is important in birds due to their complete cartilaginous tracheal rings. Use of a cuff could cause severe pressure necrosis on the trachea that cannot expand, and these lesions can swell after surgery causing asphyxiation. Technicians should be prepared with a variety of tubes including Cole® tubes with a graduated tip to prevent gas leaking from the glottis. Small birds such as cockatiels and budgies do well with 1.0-1.5mm tubes, or use of an 18g (or smaller) IV catheter used as an ET tube. When using these small tubes, technicians need to be vigilant in monitoring that the tubes do not kink and do not become clogged with mucus.

Technicians should be prepared with emergency drugs calculated or even pre-drawn up, as well as blood products and colloids ready to inject. There is often no time once a bird stops breathing or starts bleeding, to start drawing up medications. A temperature probe could be placed in the vent or crop to monitor trends in temperature change, with various heat sources available such as radiant heat or forced hot air blankets. Surgical monitoring should include SPO2 and CO2 monitors, indirect blood pressure, and ECG. These parameters should be closely observed for changes. Birds may become bradycardic, develop arrhythmias, get a mucus plug in their ET tube, and monitoring devices will aid in fast identification of distress. Some of the most difficult challenges for technicians involve getting these monitoring devices set up and secured so they are effective throughout the procedure. Using tape to secure the ET tube and the BP Doppler/cuff will help. Having everything prepared, cut, and set up before the patient is sedated will help minimize anesthetic time.
Once birds are induced and on isoflurane or sevoflurane as their maintenance anesthetic, technicians will need to remove feathers and position the bird appropriately, securing all appendages. Care must be taken to moisten fingers or use hemostats to remove feathers, and not allow them to blow around the surgery room. Technicians should use aseptic technique to scrub the surgical site after pre-warming the chlorhexidine scrub and saline. It is crucial to avoid cold substances that will drop the body temperature. Use of alcohol should be avoided for this reason, as well as because of the risks of fire when using electrocautery. All of these tasks need to be done in a timely fashion, as anesthetic duration can become a serious problem. The goal should be to have everything ready and have practiced extensively in order to achieve the fastest induction and prep time possible, allowing the doctor to have as much time as possible for the actual procedure. Once the coelomic cavity and abdominal air sacs are open, there will be changes in pressure and spontaneous respiration. Technicians should be prepared to manually ventilate the patient every 6-10 seconds, or have the bird hooked up to a mechanical ventilator. Reflexes technicians can use to monitor depth of anesthesia are the palpebral, toe pinch, and wing twitch, which should be abolished at a safe anesthetic plane. Loss of corneal reflex may indicate that the bird is too deep under anesthesia.

Recovering birds from spaying or neutering is a critical time, and sadly many birds perish during this delicate period. Technicians should be careful to not right the bird or move it abruptly to prevent dramatic blood pressure changes. Manual ventilation should be continued with oxygen until the bird shows signs of spontaneous respiration. Temperature monitoring during this period is crucial, and keeping them on a constant rate infusion of crystalloids is recommended. When the bird is able to swallow and reflexes have returned, it can be safely extubated. Technicians should swab the glottis and oral cavity as mucus can accumulate during the procedure. Maintaining a quiet, warm, and calm atmosphere will assist with recovery.

Technicians should also be part of the team to discharge the patient, often instructing clients on home care and medicating. They should be prepared to discuss not only techniques to administer prescribed medications, but also to monitor the bird for signs of chewing the incision or having a bad recovery at home. Additionally, they should be ready to talk about what to expect with regards to further reproductive behavior at home. They should help explain that the ovary is still present in the bird and that they should still provide 12 hours of quiet night time darkness and avoid sexual stimulants. Birds with an ovary and no oviduct can still produce follicles that may drop yolks into the coelomic cavity causing coelomitis, hernia, and other complications. This is most common in pigeons and chickens, but can occur in parrots. Clients should be made aware of all possibilities in future medical care.
INTRODUCTION
Exotic pet veterinary technicians must be prepared to discuss all aspects of reproductive health with clients, as well as to then be able to competently execute the anesthetic and nursing care required for the procedure. While most clients understand the importance of spaying and neutering dogs and cats, most do not consider this procedure for their pet reptiles. Some are not even aware of how reptiles reproduce, and technicians can be instrumental in client education. Most reptile species are oviparous, which means they lay eggs. But some lizards (skinks and Jackson chameleons) and snakes (boa constrictors, vipers, and garters) are viviparous, meaning they give birth to live young. This is not a presentation on anesthesia, but it will cover some key points specific to reptiles.

Sexual Dimorphism:
- **Chelonians**: Males have a longer tail with more distal cloacal opening and plastral concavity and are generally more brightly colored.
  - Sliders, painted, and map turtles: Males are smaller with longer fingernails on the forelimbs.
  - Eastern box turtle: Males have a more distinct red or orange iris.
  - Leopard tortoise: Females have longer hind limb toenails.
  - Desert tortoise: Males have more prominent mental gland and gular scutes.
- **Lizards**: Some species are sexually dimorphic, but most are monomorphic. Male lizards have a pair of hemipenes, and some male species will have bulges caudal to the vent. Using a blunt tipped sexing probe may be useful.
  - Iguanids, Geckos, Agamids: Males have pronounced dewlaps and large femoral pores when adults.
- **Snakes**: Males have paired hemipenes, and therefore a thicker tail. The most effective way to determine sex is to use a blunt, lubricated sexing probe laterally and caudally to the vent. Males will probe deeper than females.
  - Boids: Vestigial hind limbs called spurs are larger in males.

**Female**
Egg development in reptiles starts in the liver. Vitellogenesis is the formation of yolk in the liver, with subsequent deposition around the ova within each follicle. This is a vital phase in reptile follicle development. Estrogens stimulate the liver to convert the adipose tissue and dietary lipids into vitellogenin which is selectively absorbed from the circulation by the follicles. During this stage, follicles can reach up to 100 times their original size. The liver also dramatically enlarges and becomes bright yellow.

Paired ovaries are located in a variety of locations depending on species, but primarily they lie dorsally in the body cavity, caudal to the lungs. The ovary and oviduct change in size based on age and breeding season. The mesotubarium connects the ovary to the oviduct, which lies lateral to the ovary, and the ostium (infundibulum) receives the ovulated follicle, passing it to the oviduct. For oviparous reptiles, the egg starts to form in the portion of the oviduct called the magnum where layers of albumin are added. Next they pass to the shell gland where protein and shell membrane and shell matrix are secreted. They next move to the vagina, where they will collect until they are deposited by passing through the cloaca out the vent. During this phase, total serum calcium concentrations are as much as two to four times higher than normal.

For female reptiles, many reproductive diseases, such as egg or follicular retention, is not considered an emergency situation as it is with birds. Reptiles may go weeks to months in this condition without appearing to have a problem. But, these reptiles may present with a decreased appetite, poor husbandry and sometimes have a distended coelom. Radiographs or ultrasound may reveal shelled eggs being retained, or large developed follicles that have not passed into the oviduct for development yet, but are causing discomfort. These conditions are often referred to as preovulatory follicular stasis or post ovulatory egg stasis/dystocia. Secondary anorexia tends to be the most complicating factor when
conditions go undiagnosed, however anemia and hepatic lipidosis are also possible complicating factors. Most reptiles do not eat when gravid, and when this turns into an extended period of time due to dystocia, the pet will become ill. The majority of reproductive diseases that affect reptiles involves females, and are caused by husbandry or nutritional related deficiencies. Without proper nutrition, calcium, UVB light, humidity, and appropriate nesting sites, reptiles often will not complete the egg cycle.

Chelonians primarily suffer from egg retention, or inability to lay eggs. Causes are generally environmental (females require a suitable nesting site where they can dig and excavate) or pathological (hypovitaminosis A, malnutrition, nutritional secondary hyperparathyroidism, dehydration, cystic calculi, or egg yolk coelomitis). Tumors are considered rare in chelonians, but it is possible that they are under-diagnosed due to the difficulty in opening the shells for necropsies.

Approximately 20% of lizards are viviparous (bear live young). Oviparous lizards often retain eggs (particularly iguanids), however more commonly they retain follicles that are largely developed, but fail to ovulate and undergo atresia as previously described as preovulatory follicular stasis. They can remain stagnant for months and may become necrotic or inspissated. Signs are anorexia, lethargy, coelomic distension, and sometimes dyspnea. Causes are similarly malnutrition or other husbandry related deficiencies such as dehydration, hypovitaminosis A or reduced calcium absorption, but can also include coelomic masses, cystic calculi, or malposition of the oviduct or eggs.

Dystocia occurs in snakes, but is more common in oviparous than viviparous. Generally, inappropriate nesting sites, stress, malnutrition, dehydration, or salpingitis are the causes. Anorexia is common, and eggs are generally palpable on physical exam as they are relatively large.

**Males**

The male gonads are positioned dorsally in the body cavity, caudal to the lungs, anterior to the kidney. The reproductive anatomy includes paired testes, epididymi, and vas deferens which all increase in size with age and in the breeding season, often twice as long as their diameter. The vas deferens leads to the cloaca at the base of the penis or hemipenes. Snakes and lizards have hemipenes located posterior to the vent within a hemipenal chamber that evaginates during mating, usually unilaterally. Turtles and crocodilians have a single penis located on the cranioventral floor of the cloaca which everts during mating. Both sex organs function similarly in that when they are evaginated or erect, the walls of the urethral groove meet dorsally to form a tube which functions to funnel sperm from the exit of the vas deferens into the female. The penis and hemipenes do not serve any urinary function.

Male reptiles do not tend to have gonadal reproductive disease that would require removal of the testes, however they can be neutered to try to control aggressive behavior with variable success at rectifying the aggression. Males also can have prolapses of the penis or hemipenes, often caused by husbandry problems or mating problems. Exposed prolapsed penile tissue that will not reduce may become swollen, ulcerative, or infected. Amputation of the phallus is a common procedure when required, and also to prevent reproduction as males cannot copulate without the phallus.

**Surgery**

Anesthesia during spaying or neutering of reptiles is the primary responsibility of the technician. Knowledge of each species POTZ (preferred optimal temperature zone) is important as reptiles should be kept at their POTZ for at least an hour prior to and after surgery. Usually a technician will be responsible for setting up the pre-surgical/recovery enclosure. Technicians should also prepare the surgery suite with the appropriate POTZ using radiant heat, forced hot air blankets, bubble wrap to secure extremities of large reptiles, or other gentle warming devices.

While pain is poorly understood in many reptile species, they do feel pain and a proper analgesic/sedative plan should be anticipated. Reptiles do well with a variety of combinations of drugs such as: opioids (morphine/hydromorphone), NSAIDs (meloxicam), dexmedetomidine, alfaxalone, anxiolytics (midazolam) and local blocks (lidocaine/bupivacaine). If reptiles have not been kept at the proper POTZ, or if these injections are given SC (subcutaneous) rather than IM (intramuscular) or IV (intravenous) these drugs may have a long onset of effect. Their slow metabolism and absorption should be considered when
planning the schedule of surgery, with SC/IM sedation often taking up to an hour. When possible, such as
with lizards, giving sedation IV is preferred, as onset of effect is immediate and predictable. IV access in
lizards is easily accessed via the caudal tail vein. While intracardiac phlebotomy is often performed on
snakes, it is not advisable to give injections directly into the heart if another option is available, such as
the tail vein. Turtles can be given IV injections in the jugular vein if the head is able to be withdrawn from
the shell.

Deep sedation will be required prior to placing IV or IO (intraosseous) catheters because these devices
will require aseptic technique and painful placement such as a cut-down for the IV or the IO placement
into the bone. Each species have different locations for IV or IO catheterization, and these should be
placed prior to surgery both to administer fluids and to be prepared in the even emergency drugs are
needed.

- Snake: surgical cut-down for jugular, palatine-pterygoid in large snakes
- Chelonian (turtles and tortoises): Jugular vein does not normally require a cut-down procedure
- Lizard: Surgical cut-down of the jugular of cephalic vein

Some IO options for reptiles include:

- Snake: none
- Chelonian: D
- Distal humerus or the carapace/plastron bridge
- Lizard: Distal femur, proximal tibia, or proximal humerus.

Intubation of reptiles is not difficult once they are appropriately sedated. The glottis is situated rostral in
the oral cavity in carnivores, central in omnivores, and distal in herbivores. High viscosity mucous can
make sliding the tube in challenging and light lubrication may assist. While some reptiles have complete
cartilaginous tracheal rings, and others do not, it is recommended to use uncuffed ET tubes as a general
rule for reptiles.

Use of a mechanical ventilator or preparing for manual intermittent positive pressure ventilation (IPPV) is
mandatory for all reptiles due to some key physiologic and anatomic differences from mammals.
Primarily, reptiles become apneic during anesthesia because they lack a muscular diaphragm and rely on
skeletal muscle movement for ventilation. Voluntary expiration is achieved by movement of the pectoral,
intercostal, and abdominal musculature. While the lungs are the major organs for gas exchange in
reptiles, some aquatic species utilize cutaneous gas exchange for the elimination of CO₂. Some can also
convert to anaerobic metabolism during times of extended apnea. Another complication in reptiles is right
to left pulmonary shunting which directs oxygen-poor blood that is returning to the heart from the body
back to the body, bypassing the lung. This causes a ventilation-perfusion mismatch, an event that occurs
in the lungs whereby exchange of air between the lungs and the environment (ventilation) and the
passage of blood through the lungs (perfusion) are not evenly matched. This can occur in all reptiles, but
is more common in aquatic species, during anesthesia with dorsal or lateral recumbency, and when there
are large coelomic contents (such as eggs). Reptiles tend to have larger lung volumes than most
mammals, however, the surface area for gas exchange is approximately only 20% in comparison. They
increase their minute volume by increasing respiratory rate, and the respiration is controlled by
environmental temperature, hypoxia, and hypercapnea. Unlike mammals, low oxygen concentrations
(hypoxia) is responsible for increasing respiratory rate, while increased carbon dioxide (hypercapnia)
causes increased tidal volume. Therefore, for reptiles, the stimulus to spontaneously breathe is from
hypoxia, which is important to remember during recovery, as they should be taken off 100% oxygen and
ventilated with room air until breathing on their own. These physiologic parameters must all be taken into
account when ventilating the patient. Respiratory rates should be less than that of mammals, with an
average of 4-8 breaths per minute. Peak airway pressure should not exceed 10-15 cm H₂O and
inspiration should not take longer than 1-2 seconds. Excessive IPPV can cause hypotension and
decreased cardiac output, therefore the lowest pressure and inspiration time necessary to provide
adequate ventilation should be used.

Aseptic technique when prepping reptiles for spay/neuter is important. Female chelonian spay incisions
are made in the skin cranial to the hind leg with the patient in lateral recumbency, while lizards will have a
ventral off-midline incision in dorsal recumbency, and the approach in snakes will vary greatly based on
the size of the animal. Use of sterile scrub brushes, toothbrushes, or other devices to clean between
scales or scutes are important to utilize during the surgical scrub. Warming the scrub and using warmed
saline instead of alcohol is important to not cool the animal.

Monitoring equipment is often overlooked by technicians who may not understand how to use equipment
with reptiles. One important instrument is an ultrasonic Doppler probe placed directly over the heart or
carotid artery to auscult the heart rate and rhythm. Electrocardiography can also be used to detect
changes in heart rate or arrhythmias; however, it does not determine mechanical performance of the
heart due to reptiles having 3-chambered hearts. Monitoring direct arterial blood pressure, while accurate
in reptiles, is impractical due to limited access of peripheral arteries. Arterial blood gas analysis is also
impractical for the same reasons. Using cardiac samples are inaccurate because of the mixture of arterial
and venous blood within the ventricle. Pulse oximetry is useful for monitoring trends in arterial oxygen
saturation but should not be interpreted literally. Probes can be placed on extremities of lizards and
chelonians or using rectal/cloacal probes. End-tidal PCO₂ for reptiles requires an analyzer with low
sampling rates of 50ml/min or less, and is an effective way to monitor respiratory performance. Changes
give valuable information such as airway leaks, obstructions, or malfunction of the ventilator or ET tubes.

Being prepared for emergency situations such as hemorrhage or cardiac changes will allow technicians to
focus on the patient rather than panicking while trying to calculate and draw up drugs. Emergency drugs
should all be calculated ahead and perhaps drawn up prior to anesthesia, and surgical devices such as
hemoclips or electrocautery should be prepared for use. Recovering reptiles can be fast, or very
prolonged based on the drugs used, absorption rates, and temperatures. Once surgery is complete,
technicians should focus on keeping the patient at its POTZ, and not above, as overheating creates a
higher demand for oxygen and can cause metabolic derangement. As described earlier, reptiles differ
from mammals in that the stimulus to breathe is from low oxygen concentration rather than high carbon
dioxide concentration, so it is important that ventilation continues using room air rather than 100%
oxygen. As reflexes return, jaw tone, blinking, righting, and spontaneous breathing, patients should be
extubated and monitored for continued respirations. Hospital enclosures should be ready with the correct
POTZ and comfortable settings. Clients should be instructed that reptile skin takes longer to heal than
mammals, so suture removal will be in 6 weeks if the pet doesn’t shed sutures out before that. While
soaking and bathing should be discouraged until they are healed, many reptiles require water to eat and
stay hydrated, and technicians should help clients develop a plan to keep the animal comfortable and
nourished during the weeks following surgery. Misting, humidifiers, and even holding the animal (such as
an aquatic turtle) with their head positioned over water to eat/drink multiple times a day may be helpful.
INTRODUCTION
Exotic pet veterinary technicians must be prepared to discuss all aspects of reproductive health with clients, as well as to then be able to competently execute the surgical and nursing care required for the procedure. While most clients understand the importance of spaying and neutering dogs and cats, technicians need to understand and educate clients about the importance of this procedure in exotic mammals as well. This is not a presentation on anesthesia, but it will cover some key points specific to the species discussed.

RABBITS
The most common mammal seen in exotic pet practice are rabbits. While controlling the feral rabbit population may not be as large a concern as it is for cats, spaying and neutering for controlling the pet population is important. Gender determination mistakes made at the pet stores when they are bunnies are common because gender can be difficult to determine if the testicles have not descended, which occurs around 12 weeks of age. Male rabbits (bucks) have two hairless scrotal sacs cranial to the penis. They do not have an os penis, making gender determination tricky prior to the descending of the testicles. Gently everting the genitals can be done in young bunnies under 12 weeks to observe either an everted tube-like opening in the male or a slit-like opening in the female. It is common for clients to bring in a male/female pair without knowing, or a female they thought was a male that is pregnant from the store.

Female rabbits (does) can develop many disorders of the reproductive tract. They reach sexual maturity by 4-6 months and are induced ovulators. Preventatively performing an ovariohysterectomy (OHE) at a young age, preferably between 6 and 12 months, will prevent all of the following reproductive disorders.

- **Uterine Adenocarcinoma**: Most common pet breeds of intact does have a 50%-80% likelihood of developing adenocarcinoma. It is generally a slow-growing cancer that if removed prior to metastasis (1-2 years) is curative. Early signs include hematuria or other vaginal discharge and will progress to lethargy and anorexia. Often clients will complain of "blood in the urine" which should not be confused with a urinary tract infection.

- **Endometrial Hyperplasia and Polyps**: Generally associated with the aging rabbit, this process can often mimic or be a precursor to adenocarcinoma. Bloody discharge, lethargy, appetite change, and sometimes abdominal distension can be signs.

- **Dystocia**: Rabbits usually have routine deliveries. Gestation averages 31 days and litter sizes can vary from 4-12 kits. Dystocia may be more common in captivity due to the general increase in obesity, poor diet, and lack of exercise. Signs may be straining, bloody vaginal discharge, or persistent contractions.

- **Others**: Less common reproductive problems include pyometra and uterine torsion (often caused by pseudo pregnancy), pregnancy toxemia, and endometrial venous aneurism.

Neutering bucks is also important, not only for medical reasons, but often more importantly for behavioral reasons. Intact bucks often display undesirable behaviors that can be reversed if neutered between 6 and 12 months. They will mount other rabbits, people, or inanimate objects constantly. They often emit a jet of urine ("spraying") and can be very territorially aggressive. Behaviors such as licking, rubbing, nipping, and even biting are all normal for intact bucks. Medical disorders of bucks include:

- **Orchitis or Epididymitis**: Depending on which part of the reproductive organ is infected, there may or may not be swelling present, but this disorder is often associated with abscesses. Causes could be Treponema paraluiscuniculi, P. multocida, or bite wounds from aggressive cage mates.

- **Testicular Neoplasms**: Most common would be seminomas, interstitial cell tumors, Sertoli cell tumors, or teratomas. Usually this is obvious by an enlarged testicle.

Surgical Considerations
Some key points to remember when anesthetizing rabbits (and all exotic pets) are to remain calm, quiet, and have things prepared ahead to minimize anesthetic time. The following are some unique issues to rabbits:
- Rabbits should not be fasted more than an hour or two prior to surgery, if at all. They lack the ability to vomit, and having ingesta in their stomach will aid in preventing ileus in the likely event they don’t eat right away after surgery.
- Rabbits core body temperature ranges from 100-103F. This should be monitored throughout and heat support provided to maintain.
- Rabbits have circulating levels of atropine esterase that make the efficacy of atropine unpredictable. Glycopyrolate may have a better affect, but technicians should be prepared and always monitor heart rates.
- Injected drugs have a shorter duration of effect than in larger animals at equal dosages. Higher doses may be necessary for rabbits and other herbivores.
- Rabbits have extremely delicate skin that is easily torn or cut using standard clipper blades. Using clean, sharp blades minimally and carefully helps prevent iatrogenic wounds.
- It is relatively easy to place a cephalic catheter in a rabbit. This should be used not only for maintaining homeostasis, but inducing anesthesia and delivering constant rate infusions where indicated.
- Multi-modal analgesics, sedation, and anesthetics should be used based on each patient’s American Society of Anesthesiologist’s (ASA) status. These should include benzodiazepines, alpha 2 adrenergic agonists, dissociative anesthetics, opioids, NSAID’s, inhalant anesthetics, and local anesthetics. It is imperative that rabbits are kept pain free, as pain is a key cause in causing anorexia, which can cause ileus.
- Use caution when tying limbs to the surgical table. Rabbits have very thin cortices and strong muscles that can lead to a fractured back if struggling occurs.
- Intubating rabbits can be achieved by blind techniques, using an endoscope, or using a supraglottal airway device (V-gel ® Dovsinnovent, UK).
- Anesthetic monitoring should include pulse oximetry (does not work well on the paws due to fur, but ears, tongue, and even forearm work well), ECG, capnography, Doppler probe on the heart (very cranial in rabbits) and most importantly- observation of the patient.

GUINEA PIGS

Gender determination is relatively easy in guinea pigs. Males (boars) have large scrotal sacs and tests with a penis that easily everts. Females (sows) have a clear “Y” shape surrounding the urethra and pointing to the anus. Sexual maturity occurs at 2-3 months of age and they have an induced polyestrous cycle. A unique problem for guinea pigs is that the sow’s pubic symphysis closes permanently if she is not bred before 7-8 months of age. Owners should be educated about this in the event their older guinea pig becomes pregnant without a prior pregnancy history.

The most common reproductive medical disorders of guinea pigs are ovarian cysts. 76% of intact sows over 18 months will develop them. While the cysts themselves are benign, they can grow large enough to cause discomfort, burst and cause inflammation, and if left can lead to leiomyomas, endometrial hyperplasia, and endometritis. Increased circulating hormones can also lead to ovarian and uterine neoplasms. Symptoms include bilateral hair loss, depression, anorexia, and sometimes vaginal bleeding or discharge (often confused with hematuria).

Pyometra, vaginitis, and metritis can also occur in sows, breeding or not. Signs include vaginal discharge, impacted vaginal area, abdominal swelling, depression, anorexia, hypo or hyperthermia, and polydipsia. In addition to an OHE, broad spectrum antibiotics should be given.

While boars have less reproductive disease, testicular tumors have been reported.

Surgical Considerations
- Guinea pigs should be fasted only an hour or two prior to surgery. Like rabbits, they lack the ability to vomit and having ingesta in their stomach will aid in preventing ileus in the event they don’t eat right away after surgery. However, unlike rabbits, they tend to retain a lot of food in their oral cavities and emptying them is important to prevent airway blockage.
Like rabbits, guinea pigs are herbivores and have adapted to detoxify plant chemicals, which can mean parenteral drugs may have a shorter duration of effect when given at “standard” mammal doses.

High metabolic rates also can influence dosage, making drugs less effective or having a shorter duration of effect than would be expected in larger mammals.

High metabolic rate can also predispose small animals to hypoglycemia in the peri-anesthetic period.

Because of the small thoracic size of guinea pigs, it is recommended to elevate their head/neck/thorax on a wedge. This will prevent abdominal organs from putting pressure on the diaphragm and prevent passive regurgitation from the esophagus that can result in aspiration.

Guinea pigs tend to hypersalivate and passively regurgitate fluid which can fill the oral cavity and block airways. Swab the mouth regularly if not intubating to prevent aspiration.

IV access is attainable in the cephalic veins and should be utilized. The cephalic vein tends to be lateral on the forearm.

Intubation is complicated and almost impossible due to the palatal ostium. Use of a micro-endoscope is helpful in obtaining safe intubation when available.

Multi-modal analgesia, sedation, and anesthetics should be used based on each patient’s American Society of Anesthesiologist’s (ASA) status. These should include benzodiazepines, alpha 2 adrenergic agonists, dissociative anesthetics, opioids, NSAID’s, inhalant anesthetics, and local anesthetics. It is imperative that guinea pigs are kept pain free, as pain is a key cause in causing anorexia, which can cause ileus.

RATS
Rat gender is identifiable by the large scrotal sacs in the males that descend at approximately 3 weeks. Prior to that, the anogenital distance is significantly wider in males. More and more pet rat owners are interested in pursuing preventative spaying/neutering of rats due to the high likelihood of preventing both negative behaviors (aggression, mounting) and hormone induced mammary tumors in females. Mammary gland neoplasia is the most common neoplasia of pet rats, and while most are benign, they can grow large rapidly, erupting in ulcers and causing physical incapacitation. While rat mammary tumors are known to be sensitive to hormonal stimuli (spaying them at 3 months significantly reduces the occurrence of mammary tumor development), estrogen does not appear to be the main cause. Current theory hypothesizes that excessive prolactin from pituitary gland tumors is what causes the tumors, while estrogen is considered to contribute to pituitary tumor growth.

Surgical Considerations

- High metabolic rates predispose rats and other small rodents to hypoglycemia when anesthetized.
- Small rodent size is associated with higher body temperatures and with rapid convective heat loss, hypothermia is a common problem without heat support. Rectal temperature should be monitored and use of warming devices utilized.
- Avoid using alcohol when prepping the surgical site due to the cooling effects. Use warmed saline with chlorhexidine scrub.
- Small rodents have higher tissue oxygen consumption rates and require more oxygen, which is associated with more rapid inhalant uptake and excretion.
- Pre-anesthetic sedation using anxiolytics (midazolam), dexmedetomidine, afaxalone, will all contribute to a lower stress induction than “boxing down” the patient. It will also help minimize employee exposure to anesthetic gasses.
- Blood volume can become an issue with small patients. Small amounts of blood loss can lead to hypovolemic shock and death. Technicians should be ready to place IV or IO catheters for fluid replacement as needed.
- Small animals imply tiny airways; care should be made to position the patient with a clear mouth and extended neck if not intubated.
- Pulse oximeters and Doppler flow devices work well for anesthetic monitoring. Small, atraumatic clamps on an ECG are also helpful.
• Analgesics are critical to prevent chewing of incisions post surgically. Opioids, NASAIĐs, local anesthetics, and dissociatives can all be safely used.

FERRETS
In the United States, most ferrets are spayed or neutered prior to purchase, often as young as 5 or 6 weeks of age. However, due to the current theory that early spay/neuter practices contribute to adrenal disease, it is becoming more common to see intact young ferrets in clinical practice. It is very important to understand the importance of spaying and neutering these pets due to some unique problems they can have in captivity.

Males (hobs) have an obvious os penis located on the ventrum, caudal to the umbilicus, and become sexually mature at 8-9 months. The main reasons for neutering hobs are behavioral rather than medical. While generally not being aggressive with humans, they can be aggressive towards other ferrets or pets. They also will release a pungent combination of oils and urine which can be overwhelming, and can become depressed from lack of mating.

Females (jills) are seasonally polyestrous. They are induced ovulators, and uniquely, if they are not bred or artificially stimulated to ovulate, they will remain in estrus. In over half of jills, this can lead to an estrogen induced toxicosis causing bone marrow hypoplasia. A non-regenerative anemia, neutropenia, thrombocytopenia, combined with vulvar swelling/discharge, anorexia, and lethargy are all signs. Because of this severe and common process, it should be strongly recommended to spay females before the first Spring after birth. Ovarian and uterine tumors are also common in intact jills.

Surgical Considerations
• Ferrets are easy to intubate, similar to cats. They have high jaw tone, even at moderate levels of anesthesia.
• The thoracic cavity of ferrets is significantly larger than that of rabbits and rodents. Their heart lies much farther caudally, which is important for Doppler probe placement.
• Cephalic catheters are easily placed in ferrets.
• Unlike rabbits and rodents, the paw of ferrets tends to be a good site for SPO2 probes.
• Multi-modal sedation, induction, maintenance, and post-surgical analgesia/anesthesia is generally tolerated well by ferrets.

SUGAR GLIDERS
Sugar gliders are marsupials, making it easy to distinguish the females with their pouch, and the males with a large scrotal sack on their ventrum. While there are reports of reproductive diseases in females, including tumors and abscesses being the most common, in general neutering the males is more frequently recommended for controlling reproduction and behavioral issues. Sexually frustrated males have an increased rate of genital self-mutilation. They will mutilate the tip of their bifid penis, and while amputation is possible, preventative neutering would be preferable.

Surgical Considerations
• It is possible to use a 1mm endotracheal tube to intubate gliders, however they tend to do well with a face mask of iso or sevoflurane.
• Thermoregulation is vital during anesthesia. Use warmed saline instead of alcohol for scrubbing and provide appropriate thermal support.
• Some parenteral sedatives have shown to be dangerous in gliders, such as Telazol. Be sure the anesthetic protocol is chosen minimally and carefully. Benzodiazepines, opioids, NSAID’s and inhalant gas have been shown to be safe combinations.
• Gliders tend to wake up and immediately begin chewing on incisions, particularly post castration. Using local blocks can help, but also distracting gliders with food items can be a way of keeping sutures in place and prevent mutilation.
• Anesthetic monitoring should include pulse oximetry (patagial skin or feet work well) ECG, and Doppler probe over the heart.
You are the only one living your life, no one can know your journey but YOU!

- Please in a few sentences, write down how you would like your coworkers, friends, family to describe you.
- What values do they think you express?
- Remember this is only for you, no one is going to read this.
- Think of you on your best day, you are not hungry or tired. Tell me how the people closest to you would describe you.
- We spend our lives as children learning to avoid bad feelings. We don’t like to feel diminished, hurt, or disappointed.
- The problem is trying to shield yourself all the time is exhausting, we typically think it is the answer when in fact it creates disconnection, takes away purpose, and it’s the easy way out.

Perfectionism

- Striving for perfection vs being a perfectionist not the same thing
- Perfectionism is a way to deflect shame, judgment, blame.
- It is more about perception, we want to be perceived as perfect. This presents a major problem as we cannot control someone else’s perception.
- This is at the core of trying to earn approval or acceptance from someone
- Research shows that perfectionism is correlated with depression, anxiety, addiction, and missed opportunities. It is a form of shame.

Self-Kindness

- Just like when the flight attendant tells you to put your oxygen mask on first, then assist others. You cannot be compassionate with others if you are not compassionate with yourself first

Humanity

- This bring connection to those around you. What makes us human is our imperfections. This can create connection!

Mindfulness

- Don’t go down the rabbit hole. When we are feeling our feelings is the time we tend to make thinking errors which when left unattended can run rampant.

What are boundaries?

- Remember that exercise we did and the person you are at your best?. How did you take care of yourself during those times?
- Why don’t we set boundaries:
  - FEAR of rejection and, ultimately, abandonment
  - FEAR of confrontation
  - GUILT
  - We were not taught healthy boundaries
  - Safety Concerns

- Why are they important?
- People who have and uphold their boundaries are shown to be happier in themselves. This does not mean they are the most popular person
- “It is impossible to set boundaries without setting consequences” (IPFW/Parkview Student Assistance Program). This means that when setting boundaries, it is important to explicitly state why they are important. It is also crucial to only declare consequences that one is willing to follow through on, or else the boundaries will not be effective.
How do you start upholding your boundaries? The best way is to start talking about them! Get an accountability partner. Encourage your teammates to uphold their boundaries! Make it a priority to make sure your coworkers take breaks and leave on time.

Shame is the real thing that holds us back. We don’t want to be seen as weak or needy. How many of you get frustrated when people uphold their boundaries? It’s not what is in the best interest of the “team” right? Well the problem comes back to, if you don’t take care of yourself first you can’t help others. Teamwork is more about helping people take care of themselves but not at your own expense.

Resilience is the ability to overcome adversity.

It is a wonderful and terrifying idea that you and only you have the power to change your situation. It all has to do with how your thoughts and behaviors. It is just as important to know that there will be ups and downs and know that you can face the hard times. The idea that you can get through anything is resilience.

Numbing is in my opinion one of the hardest things to let go of. The issue with numbing is that we can numb just one emotion. When we numb we numb every feeling, even the positive ones.

A: Have I been Abstinent today? (You define this for yourself)
E: Have I exercised today?
I: What have I done for myself today?
O: What have I done for others today?
U: Am I holding onto unexpressed emotions today?
Y: Yeah! What is something good that’s happened today?

A gratitude journal is a huge asset not only in your work environment but in your home environment as well. At work start a gratitude board, make sure to find a gratitude each day. What you will find is that even on your hardest days, in the mist of feeling tired and overwhelmed, what am I grateful for? This positivity is infectious. It helps you speak up when things are hard but in a productive manner.
Communication is the key to success in and out of the workplace
  ○ A survey in HR Magazine reports that of 4,000 employees, 46 percent said they routinely received confusing or unclear directions.

Why is communication so hard?
  ○ Three components of communication
    ■ What you mean
    ■ What you say
    ■ What the other person sees/hears
  ○ All of these have to align to get your message across the way you intend it to

What are some skills you need to be able to have clear communication?
  ○ Emotional Intelligence
    ■ Self-awareness
      ● Know what your pet peeves are and why we have them.
      ● Many of our interactions are influenced by how we view ourselves. When you interact with others, you may find that you have certain tendencies that reflect your own values.
    ■ Self-awareness skills
      ● Remove the stigma that you either feel good or bad. Use emotions as a doorway to learning about yourself and your effect on others.
      ● Learn what are triggers for you and why they trigger you. Make a plan for what you will do if you run into a trigger. Even if that means you will wait or ask for time to respond to a triggering event until you can have a logical conversation about it.
      ● Keep a mood journal.
      ● Know what your tendencies are when you are under stress.
    ■ Self-management
      ● Have clear and logical thoughts when others are highly emotional
      ● Keep calm in difficult moments, allowing you to be optimistic and positive.
      ● Stay in control with you are feeling anxious or distressed.
    ■ Self-management skills
      ● Practice naming your emotions
      ● Distancing yourself from the situation
      ● Have a clear idea of what your goal is
      ● Using logic to figure out how to best reach your goals
      ● Maintaining strong values, like honesty, openness, adaptability, and conscientiousness
    ■ Social-management
      ● If you lack Social Awareness you may come across as hard and unapproachable.
    ■ Social-management skills
      ● Sympathy Vs Empathy
      ● Use nonverbal communication
        ○ Other options include:
          ■ Body movement and postures
          ■ Tone of voice
          ■ Touch
          ■ Space
      ● Relationship management
        ● Relationship management is at the core of success. Whether this is with clients, employees, or even in your personal life.
      ● Relationship management skills
If you had to solve a problem without assigning blame to anyone and you could only state facts, no opinions, how would you go about that communication?

You might start to ask questions. The pros to understanding why mistakes and conflict happen

- What happened?
- What did that mean to you?

It can lead to systems to prevent future mistakes

Contributes to a culture of mutual respect and trust

- Why do difficult conversations go bad?
  - Blame
  - Judgment
  - Opinions
  - Lack of understanding

- What can you do to turn conflict into collaboration
  - Prepare
    - Process your emotions
    - Sort out your opinions
  - The communication formula
    - Use facts
    - Use “I” statements
    - Use the “yes, and…” technique
  - Other techniques
    - Reflective and active listening
    - Notice the difference between what is said and body language
    - Ask for breaks

- Practice!
Powerful Leadership
Kristina Guldbrand, CVT, BS, CSP

1) Leadership all comes down to who is following you and why; i.e. Influence

2) Not everyone who is in a leadership position is a leader
   i) Take a moment to assess your level of leadership. If you left your current position how many people would follow you? If you asked for your team or coworkers to evaluate your leadership skills what score would you get?
   ii) No company ever has a lack of leaders.
   iii) If you are in a position of leadership your success will be limited by your effectiveness.

3) If you lead by example you will inspire others to take responsibility and gain awareness. If you say to yourself “I want to change my team” you must change yourself first.

4) Tools to develop your leadership effectiveness
   i) Emotional Intelligence
   ii) The art of making mistakes
   iii) Asking powerful questions

5) Dr. David Walton describes EI as “being aware of feelings in yourself and in others, understanding them and managing their impact. It’s about being in control, interpreting body language, coping with negativity, working with others and building psychological well-being.”
   i) Emotional Intelligence allows you to:
      (a) Know yourself
      (b) Control your emotions
      (c) Use empathy with others
      (d) Use social skills to interact effectively with others

6) In your brain, your primary senses enter through your spinal cord, they then travel through the Limbic System and then all the way to the front of your brain where rational thinking can happen. The limbic system is the place where emotions are experienced. EI requires effective communication between the rational and emotional centers of the brain.
   i) People have emotions all day every day, they range in intensity, and come in different forms of Happiness, Sadness, Angry, Afraid, and Shame
   ii) Most of the time we are not even aware of our emotions, they just seem to happen.
   iii) EI is critical to success, research shows that it accounts for 58% of performance in all types of jobs.
   iv) The link between EQ and earnings is so direct to earnings that studies have shown that for every point increase in EQ is equivalent to $1,300 increase to an annual salary.

7) Many of our relationships are influenced by how we view ourselves. When you interact with others, you may find that you have certain tendencies that reflect your own values. These values can be a compilation of lessons you’ve learned, your need for closeness, views you have about yourself, your cognitive strategies, how you want to be seen by others, or your sense of purpose. Taking a deep look into yourself can give you a lot of insight into your needs and motivation when dealing with others.

   i) There are many tactics you can use to grow in this area.
      (a) Remove the stigma that you either feel good or bad. Use emotions as a doorway to learning about yourself and your effect on others.
      (b) “Lean into the discomfort” Make a plan for what you will do if you run into a trigger. Even if that means you will wait or ask for time to respond to a triggering event until you can have a logical conversation about it.
      (c) Keep a mood journal. If this is something you think would work for you I would encourage you to also do some CBT exercises along with the journal to help you work on logical thoughts through highly emotional situations.
      (d) Know what your tendencies are when you are under stress. Just start to think about your emotions when you are highly stressed. Are you short with your staff? Are you open to other ideas? If you don’t know where to start asking a team member what their impression of you is under stress.
      (e) Ask for feedback.
1. Some good questions to ask:
   i. What are three things you think I do well
   ii. What are three things you think I could improve on
   iii. Is there anything missing from our relationship
   iv. Is there anything you have wanted to talk to me about but never felt you could?

8) Self-management
   i) If you can manage your emotions well you are able to:
      (a) Have clear and logical thoughts when others are highly emotional
      (b) Keep calm in difficult moments, allowing you to be optimistic and positive.
      (c) Stay in control with you are feeling anxious or distressed.

9) Self-management tactics
   i) Distancing yourself from the situation
   ii) Using logic to figure out how to best reach your goals
   iii) Having a clear idea of what your goal is
   iv) We tend to lose sight of our overall goal when situations become emotional
   v) Maintaining strong values, like honesty, openness, adaptability, and conscientiousness
   vi) Admit when you are wrong
   vii) When dealing with change involve others as much as possible. When things are not clear it is hard for anyone to understand why decisions are being made and it can increase tensions.
   viii) Hold yourself accountable to the commitments you make. If you don’t do what you say you are going to do then no one has a reason to believe it will happen

10) Social Awareness
   i) If you want to understand others you will have to look outside of yourself to find understanding.
   ii) If you lack Social Awareness you may come across as hard and unapproachable. As a leader having low social awareness can cost you because it will be harder to gather information about your team.

11) Social Management Tactics
   i) Sympathy Vs Empathy
      (a) Sympathy stems from the judgment of someone’s situations and is about how YOU feel.
      (b) Empathy is about understanding how someone else is feeling and is about their perspective.
      (c) It is much harder to have empathy for someone than Sympathy.
   ii) Using empathy to understand another’s perspective and feelings
      (a) If empathy is not your strong suite try this:
      (b) The next difficult situation you come across, get curious. If you had to communicate with someone about failure and you could only use facts (no opinions), I statements, and you could not assign blame to anyone how would you go about it? The next logical step would be to understand why someone did what they did and what their motivation was behind it. Get into someone else’s shoes and use that information to manage your emotional input at that moment.
   iii) Use nonverbal communication
      (a) Other options include:
         1. Body movement and postures
         2. Tone of voice
         3. Touch
         4. Space
   iv) Practice listening
      (a) Next time your employee comes to talk to you, focus and listen. This not only will build trust but you will also gain a deeper understanding of that person in the process.

12) Relationship management
   i) Relationship management is at the core of success. Whether this is with clients, employees, or even in your personal life. There are many building blocks for this step but I will focus on the ones that will aid you in your pursuit to powerful leadership

13) Relationship Management strategies
   i) Show people you value them
ii) Seek mutual understanding and information sharing
iii) Communicate clearly, without blame, and address the other person concerns
iv) Be upfront

14) Once you have started to improve your level of emotional intelligence you can move onto how to utilize
mistakes in your hospital.
i) Not all mistakes are made equal.
   (a) 2%-5% of mistakes that are made are truly blameworthy
   (b) 70-90% are treated as such

ii) If you do not have a culture that allows for mistakes to be voiced you are missing a huge
   opportunity
   (a) Patient safety
   (b) Staff Wellbeing
   (c) Responsibility of staff

15) Perfectionism
i) Striving for perfection vs being a perfectionist not the same thing
ii) Perfectionism is a way to deflect shame, judgment, blame.
iii) It is more about perception, we want to be perceived as perfect. This presents a major problem
    as we cannot control someone else’s perception.
iv) This is at the core of trying to earn approval or acceptance from someone
v) Research shows that perfectionism is correlated with depression, anxiety, addiction, and missed
   opportunities. It is a form of shame.

16) Courage
i) If you want to be a leader you must have the courage to be the first.

17) Servant Leadership
i) If you want your staff to have awareness and show responsibility then you have to set the tone
ii) If you want to inspire your staff to grow and challenge themselves then you have to grow yourself
    and challenge yourself
iii) If you want to be a leader inspire others to be leaders

18) How to use a coaching approach to Leadership
i) Coaching is about utilizing powerful questions to help others realize their full potential

19) Understanding your staff’s needs
i) What motivates people?
   (a) You have to meet the basic needs first.

ii) Once money concerns are off the table money is no longer a motivator for most people. Pay
    people enough that they don’t have to worry about money and they will focus on the work they
    are doing.
   (a) Autonomy -To be self-directed
   (b) Mastery -to become a master in our field or interests
   (c) Purpose -to do things that have meaning. (this is very high in the veterinary industry)

20) Collaboration and Partnership as a leadership style
i) When you let go of a commanding and controlling relationship you will promote a culture of
   interdependence.
ii) When you adopt a collaborative and partnering leadership style then you can focus on strengths,
    solutions, and future success instead of Weakness, problems, or past performance.
iii) Most hospitals severely underutilize their staff.
    (a) In order to utilize your staff, you have to trust them and believe that they are capable
21) How to build trust in your team
   i) Make time for team building
      (a) Exercises you can do right in your clinic.
         1. Utilize resources like Myers Briggs personality tests to learn about you and your staff
         2. Have your staff interview each other and present on their personality type, communication style, and how they handle stress.
         3. Ask questions that will allow others to open up about a moment that shaped their life

22) How to inspire responsibility and awareness in the face of conflict
   i) If you had to solve a problem without assigning blame to anyone and you could only state facts, no opinions, how would you go about that communication?
   ii) You might start to ask questions. The pros of understanding why mistakes and conflict happen
      (a) It can lead to systems to prevent future mistakes
      (b) Contributes to a culture of mutual respect and trust
      (c) Raising responsibility in the workplace
      (d) Eliminates the FEAR that prevents your staff from showing you their full potential

23) What are some questions I could ask?
   i) What happened?
   ii) What’s not going well and why?
   iii) What did that mean to you?
   iv) What gaps in skills, Knowledge, or experience would you like to develop?
   v) What could I do to support you?
   vi) What are you committed to?
   vii) What specific actions will you take?

24) Don’t forget to give positive feedback
   i) If you only talk to your staff when you are giving negative feedback you can expect that they will associate talking with you as a negative experience.
   ii) Make sure your show gratitude to your staff and give regular positive feedback.
**Setting Boundaries in Veterinary Medicine**

Kristina Guldbrand, CVT, BS, CSP

- Why are boundaries important?
  - They define what is OK and not OK in your life
  - They help you to maintain and preserve your personal wellbeing
  - They help others know how to treat you

- What is a boundary?
  - By definition, a boundary is anything that marks a limit
  - You get to decide where your boundaries are
  - They are always changing and evolving

- Different types of boundaries
  - Physical
    - Personal space
    - Privacy
  - Mental
    - Thoughts
    - Values
    - Opinions
  - Emotional
    - When you don’t feel the need to take on other’s emotions
      - You can not get upset if someone has a difference in opinion
      - Don’t feel the need to “fix” people
      - Don’t take things personally
  - Moral
    - When your actions align with your personal values.

- Boundaries help you to recognize and not tolerate unhealthily behaviors
  - For some setting, boundaries is easy and can be challenging for others
  - Past trauma can influence boundary setting

- Some reasons boundaries are hard in Veterinary Medicine
  - Care based profession and peer pressure
  - Customer service is important and we aim to please
  - We don’t have many good examples of healthy boundaries

- Social and Self-awareness is key to being able to set good boundaries
  - If you are not aware when a situation or person is causing you distress it will be hard to identify when a boundary needs to be set
○ Self Awareness
  ■ Know your triggers and why they trigger you
  ■ Be able to name your emotions when they are happening
○ Social Awareness
  ■ Observe situations
  ■ Active listening
○ What happens when you don’t have good boundaries?
  ■ Feeling taken advantage of
  ■ Resentment
  ■ Rebellion
  ■ Guilt
  ■ Self-doubt
  ■ Anger
○ How can you build healthy boundaries?
  ■ Make a list of what are OK behaviors and not OK behaviors from others or situations
  ■ Be Ok with saying “no”
  ■ Clearly communicate
  ■ Live out of courage instead of fear
  ■ Check-in with yourself
  ■ Stop labeling things as good or bad
  ■ Get specific
  ■ Start small
  ■ Lean into the discomfort
  ■ Take your time
○ Sometimes fences make the best neighbors
Nutrition is an often-overlooked area of veterinary critical care but it is of vital importance in the healing process of our sickest patients. While it is true that calories and nutrients can be supplied parenterally with nutritionally balanced solutions, most general veterinary practitioners are not equipped to provide total parenteral nutrition. The old adage “if the gut works, use it” applies to all of our patients. Enteral nutrition is more economical, more cost-effective, and – in most cases – better for our patients. Providing nutrition to the gut directly improves the health of the intestinal mucosa, helping to maintain its viability and decrease the chance of gastrointestinal dysfunction.

Most sources state that anorexia of more than three days’ duration warrants immediate and aggressive nutritional intervention and support. Many of our patients have already been anorexic, or have been eating less than their nutritional needs for a period of time prior to presentation to the veterinary clinic and inappetance can be expected to continue during the course of hospitalization, either because of the inciting disease process or the stress of hospitalization. For many years practitioners relied on syringe feeding to either supplement or satisfy patients’ caloric needs. Recent studies have shown, however, that syringe feeding – especially of feline patients – is very stressful and may lead to more occurrences of food aversion so the current recommendation is to avoid syringe feeding most veterinary patients. Also, many patients cannot tolerate oral feeding due to reasons including trauma, uremic ulcers, and oral surgery, among others. Other indications for instituting nutritional support include actual or anticipated loss of more than 10% body weight, burns, large losses due to vomiting or diarrhea, and trauma – particularly involving draining wounds as large albumin losses can be expected.

Once the decision has been made to place a feeding tube, the practitioner has several options available. One of the first considerations is whether the tube will be maintained outside of the hospital setting. Next, the practitioner must decide where in the GI tract the nutrition will be best utilized. Finally, the patient’s ability to tolerate an anesthetic procedure must be considered and weighed against the benefit of providing enteral nutrition. In general, tube feeding should be started slowly and increased over time to provide the nutrition the patient requires.

**Nasogastric / Nasoesophageal Tubes**

**Indications and Advantages:** Nasogastric (NG) and nasoesophageal (NE) tubes are ideally suited for short-term, in-hospital feeding. The biggest advantage to using these tubes is that, in general, little to no sedation or anesthesia is required for placement and veterinary technicians can place them easily. Additionally, the tubes themselves are relatively inexpensive. Placement can be confirmed via lateral thoracic radiograph: in the case of an NE tube, the tip of the tube should be visualized past the heart in the distal third of the esophagus; and, in the case of an NG tube, the tip of the tube should be visualized in the fundus of the stomach. If properly placed, there are few complications and, if the patient removes or chews the tube, it will generally pass through the digestive tract without further complications. The biggest risk involved in the use of NG/NE tubes is improper placement into the trachea rather than the esophagus, which can lead to pneumonia if the tube is used for feeding.

**Disadvantages:** Because the tubes are generally smaller in diameter (3 – 8 Fr), only liquid diets can be used. These tubes are only used for short-term feeding (between three and ten days) because of the risk of them becoming dislodged by sneezing or vomiting or by the patient pawing the tube out of the nose.

**Contraindications:** Because the tube is placed through the nasal cavity, patients with thrombocytopenia are not candidates for NE / NG tubes due to the risk of potentially uncontrollable epistaxis. These tubes should not be placed in patients with respiratory compromise, as the tube may occlude the nasal passage, increasing respiratory distress.

**Required Supplies:**
- Proparacaine drops
- Water-soluble lubricant or lidocaine gel
- Red rubber tube, or commercial feeding tube
- Tape
- Skin stapler or suture
- E-collar

**Placement:**
- Place 1-2 drops of proparacaine in each eye and each naris. This will numb the rostral area of the muzzle, allowing for a more comfortable placement procedure.
- Measure the tube to the last rib if placing an NG tube. For NE tubes, measure to the 8th or 9th rib space. Make note of the distance the tube is to be inserted.
- Lubricate the tube and feed it rapidly into the naris in a ventromedial direction, inserting it to the measured distance. Some technicians find it easier to feed the tube if the nose is pushed dorsocaudally (“pug-nosed”) during insertion.
- Take a lateral thoracic radiograph to confirm proper placement. Once placement is confirmed, secure the tube and record in the record the distance of insertion.
- A test dose of sterile water or saline (10-15ml) may be instilled if the technician is unsure about placement location. If coughing is elicited, remove the tube and replace.
- Secure the tube with either suture, staples, or skin glue.
- The use of an e-collar is recommended to prevent the patient from pawing the tube out. Be aware that the e-collar may discourage some patients from eating on their own.

**Using the tube:** The tube can be used for feeding immediately upon placement, if no sedation was used for the procedure. One advantage to NG placement is that it allows aspiration of any residual gastric contents prior to feeding, leading to greater patient comfort. NG / NE tubes can be used for either bolus or CRI feeding, depending on clinician’s preference. Flush the tube with 3-10ml of water prior to using it for feeding to ensure patency. If feeding via CRI, no flushing should be necessary, unless it becomes occluded. If flushing is unsuccessful, the tube should be replaced. Removal of the tube is very simple: the tube is simply pulled from the naris. It is recommended that the tube be clamped during the removal process to prevent aspiration of any contents remaining within it.

**Esophagostomy Tubes**

**Indications, Advantages, and Disadvantages:** Esophagostomy tubes are ideal for long-term feeding, including maintaining nutritional intake after hospital discharge. These tubes can remain in place from weeks to months, they are tolerated well by patients, and most owners can easily manage feeding via the tube. Esophagostomy tubes are larger in diameter than NE / NG tubes so blenderized diets can be used. While placement requires anesthesia, the procedure is generally very short (10-15 minutes) and is appropriate for all but the most critical patients.

**Contraindications:** Patients with protracted and/or profuse vomiting are likely to vomit up – and possibly chew – the tube. Esophagostomy tubes should not be placed in patients with esophageal dysfunction such as megaesophagus, esophageal stricture, or pre-existing esophagitis.

**Using the tube:** The tube can be used as soon as the patient has regained the swallowing reflex. The tube should be flushed with several millimeters of water prior to using for feeding to ensure patency. Most patients tolerate warmed fluids and food better than cold or room temperature materials. Bolus feedings several times a day are also well tolerated, rather than large volumes less frequently. When the patient is eating well on their own (defined as consuming at least 85% of their nutritional requirements), the tube may be removed. Removal can be performed without anesthesia and the stoma allowed to close via granulation.

**Gastrostomy Tubes**

**Indications and Advantages:** Gastrostomy tubes (G-tubes or PEG tubes) are ideal for patients with pre-existing esophageal dysfunction or disease, or when the esophagus must be bypassed for any reason.
The tubes can remain in place for weeks to months, or even years, are very well tolerated by patients, and easy for owners to use. These tubes have the largest bores of all feeding tubes available, so a wider diversity of diets may be used.

**Disadvantages:** Placement of G-tubes requires general anesthesia, though the procedure is usually of short duration. The tube cannot be used for at least 12 hours after placement to allow for stoma formation between the stomach and body wall. If the tube is used before complete stoma formation, peritonitis is a potentially serious complication. Additionally, these tubes must remain in place for a minimum of seven to fourteen days (depending on source) before they can be safely removed – premature removal may result in gastric perforation. Potential complications during placement include splenic laceration, gastric hemorrhage, and pneumoperitoneum. Complications that may arise after placement include protracted vomiting, peritonitis, tube migration, or displacement.

**Contraindications:** G-tube placement is contraindicated in those patients with conditions in which the stomach cannot be apposed to the abdominal wall such as ascites or space-occupying masses. Patients with pre-existing gastric disease or neoplasia are also not candidates for G-tube placement.

**Using the tube:** Once stoma formation and adhesion has been confirmed, food and medications can be provided via the tube using procedures similar to those used for esophagostomy tubes. Patients can eat on their own with the tube in place and indications for removal are the same as those for esophagostomy tubes. Removal can be accomplished without anesthesia (though some patients will benefit from light sedation). It is generally recommended that patients be fasted for twelve hours prior to tube removal to minimize leakage of stomach contents during tube removal. The patient is placed in lateral recumbency and the tube is pulled firmly and steadily out of the stomach through the stoma on the body wall and the stoma is allowed to heal by second intention. Alternatively, G-tubes can be removed endoscopically via the oral cavity.

**Jejunostomy Tubes**

**Indications and Advantages:** Jejunostomy tubes (J-tubes) are used when the esophagus, stomach, proximal duodenum, and pancreas must be bypassed. Patients with protracted and uncontrollable vomiting, severe pancreatitis, or gastric disease or dysfunction can benefit from J-tube placement.

**Disadvantages:** J-tube placement requires general anesthesia and laparotomy, though they may also be placed via a G-tube. J-tubes are small in diameter and can only accommodate liquid diets, and feeding must be provided via CRI, meaning these tubes must be maintained in a hospital setting. Due to the small size of the tubes, and the propensity of the tubes to reflux into the duodenum, clogging and kinking are common problems.

**Using the tube:** J-tubes require CRI feeding, due to the limited reservoir capacity of the small intestine. Frequent flushing may reduce the incidences of clogging and kinking. J-tubes are removed by pulling them through the skin and the stoma site is allowed to heal via second intention. It is advised to leave J-tubes in place at least seven to ten days before removal to allow for some adhesion around the entry site, which may minimize leakage of intestinal contents into the abdominal cavity at tube removal.

**References**

*Available on request*
Not So Cute: Acute Anaphylaxis
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OVERVIEW
Anaphylactic reactions are among the most acute, life-threatening emergencies we see in practice. Without proper intervention, these patients have high rates of mortality but when treated quickly and appropriately, many if not all may make a full recovery. This presentation will review the pathophysiology of acute anaphylaxis, as well as diagnostic tools available to diagnose it. We will also review appropriate interventions to save these emergent patients.

Anaphylaxis is defined in the Merriam-Webster dictionary as a “hypersensitivity resulting from sensitization following prior contact with the causative agent.” In other words, it is a hyper-response from the immune system to an allergen, food, or drug that the immune system has seen at least once before. In order to understand this reaction, it is key to learn the pathophysiologic mechanisms of the immune response, sensitization, and hypersensitivity.

IMMUNOLOGY BASICS
Immunoglobulins (also know as antibodies) are some of the primary components of the immune system. There are five classes of immunoglobulins (abbreviated “Ig”) that are common to all mammals: IgA, IgD, IgE, IgG, and IgM. These antibodies each have specific functions within the immune system and work together to create humoral immunity. IgA is found primarily in the mucosa of the gastrointestinal, urogenital, and respiratory tracts. IgD works against microbes in the bloodstream. IgG is the primary illness antibody, is found in extracellular fluid and the bloodstream and fights many pathogens by activating the complement cascade, stimulating further immune response. IgM precedes IgG in the bloodstream and acts as a stopgap until enough IgG can be produced. IgE is the primary immunoglobulin involved in the body’s response to allergens and parasitic infestations. It is also the antibody responsible for anaphylactic reactions.

An antigen is defined as any substance foreign to the body that evokes an immune response, or production of immunoglobulins. It can be a particle of pollen, a drug, a venom, a vaccine, a food, or a microbe. Antigens are detected by immune system components called T-cells. T-cells present antigens to B-cells, which are then stimulated to differentiate into plasma cells – antibody factories.

IgE is the antibody produced when antigens are first encountered by the immune system. In individuals without allergies or active parasite infestations, IgE is the least abundant antibody in circulation, comprising approximately 0.05% of all circulating antibodies. Individuals with allergies, conversely, have high levels of circulating IgE: their immune systems are primed for responding quickly to any antigen presented. IgE has a short half-life, as it is only required for immediate response to allergens. If the allergen or parasite persists, more IgE will be manufactured to respond to the continuing need.

HYPERSENSITIVITY REACTIONS
There are four traditional classifications of hypersensitivity reactions: Type IV are delayed-type reactions such as contact dermatitis and keratitis sicca. These reactions are triggered by antigen activation of helper T-cells which lead to the production of toxic macrophages and other cells that infiltrate tissues. Type III are termed “immune complex” reactions and include rheumatoid arthritis and serum sickness. Immune complexes are formed when an antibody binds to an antigen. These complexes can become embedded in tissues, leading to inflammation most commonly in the skin, joints, brain, lungs and kidneys. Type II are cytotoxic reactions mediated by IgG and IgM wherein the antibodies bind to antigens on a cell’s surface. These reactions include hemolytic reactions like IMHA as well as destruction of white blood cells and platelets. Type I reactions are also known as immediate or anaphylactic hypersensitivities. Type I reactions usually occur within 15-30 minutes of exposure to the antigen and are mediated by IgE. These reactions can be minor and localized – characterized by swelling, hives and
itching – more systemic – such as atopic dermatitis – or serious and systemic - as anaphylactic shock, or acute anaphylaxis.

**ANAPHYLAXIS**

As discussed above, IgE antibodies are produced when an antigen is first presented to the immune system. As the IgE antibodies circulate, they attach to basophils and mast cells throughout the body, arming these cells to respond quickly the next time the antigen is presented. When the antigen is reintroduced (as is the case with a second bee sting, or a repeat vaccination, or repeated ingestion of an antigenic food), it will bind to the IgE on the surface of the basophils and mast cells, leading to a cascade of degranulation of these cells and a release of their cellular contents into the bloodstream. The contents of these cells are potent inflammatory mediators, which lead to systemic reactions in the body.

One of the first mediators released is histamine, along with chemotactic factors. The chemotactic agents draw more immune cells to the area of degranulation, leading to further inflammation. There are four primary histamine receptors and each one contributes to the cascade of anaphylaxis. H₁ receptors are found in smooth muscle and endothelium and activation leads to cardiac depression, pruritus, vasodilation, bronchoconstriction, and vascular permeability. H₂ receptors are located in the gastrointestinal tract and endothelium and their activation causes tachycardia, inotropy, and systemic vasodilation. H₃ receptors are found on the myocardium and endothelium and their activation inhibits the effects of norepinephrine leading to decreased contractility and vasodilation. Finally, H₄ receptors augment further mediator release and increase chemotaxis, drawing inflammatory agents and other immune cells to the area of stimulation, increasing inflammation and stimulating the release of more inflammatory mediators. As you can see by comparing the effects of the different histamine receptors some have opposing effects (cardiac depression vs. inotropy) but the cumulative effect of stimulation of these receptors is systemic vasodilation, reduced cardiac output, and shock. To make matters worse, histamine also suppresses the effects of catecholamines like norepinephrine meaning that the compensatory mechanisms of the body are blocked, preventing vasoconstriction and a needed increase in cardiac output.

In addition to histamine, secondary inflammatory mediators are released such as heparin, platelet activating factor, interleukins, tumor necrosis factor, prostaglandins, and phospholipase A₂. These mediators induce platelet aggregation, activating the coagulation cascade, and cause continued vasodilation and increased vascular permeability. Permeable vessels leak vascular contents into interstitial spaces throughout the body leading to the phenomenon known as “third spacing” – a volume of fluid in the body that is not contributing to perfusion – worsening hypovolemia and profound hypotension. Increased vascular permeability in the larynx and the thorax leads to laryngeal and pulmonary edema. This edema, combined with bronchoconstriction from stimulation of histamine receptors, is a primary cause of death related to anaphylaxis. Those that do not succumb to asphyxiation are subject to the effects of distributive shock and – in human patients that do not seek treatment – death occurs in approximately one hour in up to 50% of those affected.

Clinical signs of anaphylaxis generally occur within one hour, but rapidity of onset is associated with more severe reactions. The site of antigen exposure determines the clinical signs exhibited. If the allergen is ingested, gastrointestinal distress and dermal reactions are most common. Inhaled allergens lead to a predominance of respiratory signs. The clinical signs most of us think of when we think of anaphylaxis are the most common: itching, hives, wheals, and redness. GI signs include hemorrhagic diarrhea and vomiting. Respiratory signs are those associated with bronchoconstriction and are manifested as sudden onset dyspnea. The inflammatory mediators also have direct effects on the myocardium, leading to arrhythmias. While there are similarities among species, cats and dogs are affected differently by anaphylaxis as a reflection of their organs that are most susceptible to shock. In dogs, the “shock organ” is the GI tract and the liver. On necropsy, dogs will often exhibit liver and visceral engorgement, with as much as 60% of their blood volume trapped in this “third space.” Cats’ and most other mammals’) primary “shock organ” is the lungs. Necropsies of anaphylactic cats often show signs of epiglottal edema, bronchoconstriction, and pulmonary hemorrhage.
Blood values for patients with acute anaphylactic reactions may be completely normal. Most commonly, patients present with an elevated hematocrit and high total protein levels. Pre-renal azotemia may be evident as a sequela of decreased renal perfusion. In dogs, liver enzyme elevation and prolonged clotting times may be noted though these changes usually aren’t evident until a few hours after presentation. Due to bronchoconstriction, most patients will have decreased oxygen levels in their blood, an academia, and hyperlactatemia.

Treatment of anaphylaxis must start with restoration of intravascular volume with boluses of crystalloid fluids. Fluids should be administered until normalization of blood pressure and other clinical signs. Epinephrine should be administered to counteract the vasodilatory effects of the inflammatory mediators and may need to be repeated until the desired response is seen. Administration of an antihistamine (usually diphenhydramine) can help resolve dermal signs. Adding an H₂ receptor blocker (such as famotidine) will help treat the gastrointestinal side effects of anaphylaxis by preventing the effects of histamine in the GI tract. Corticosteroids are often used in the treatment of acute anaphylaxis, though they take four – six hours to have an effect. Bronchodilators are a useful adjunctive treatment to help alleviate the dyspnea. Calcium blockers in particular (e.g. theophylline and aminophylline) are especially helpful as they increase endogenous epinephrine release and inhibit further histamine release. These agents can cause hypokalemia so their use should be monitored closely.

Goal-directed therapy is important to keep in mind when treating acute anaphylaxis. The resuscitation end-points include: a systolic blood pressure of 100-120mmHg; normalized urinary output; a PCV > 25%; a normal lactate (<2 mmol/L); normothermia; and improved levels of consciousness. As you can see, all of these goals can be achieved by restoring intravascular volume and perfusion.

A complete and thorough history from the client can help narrow down the cause of the anaphylactic reaction. If the reaction is related to a vaccination, the medical record should reflect the need for pre-medication prior to future vaccinations. If it appears that the reaction is related to a potential food allergy, a hydrolyzed protein diet trial should be conducted to find the antigenic protein so that it can be avoided in the future. In the case of reactions to bee stings, or envenomations, it is important for owners to know the severity of the reaction so that they can be prepared to act quickly in case of another exposure.

ANAPHYLACTOID REACTIONS
Anaphylactoid reactions present the same way as anaphylaxis, are not mediated by IgE. These reactions occur from exposure to things such as contrast media, NSAIDs, opioid administration, and exercise, among others. Because anaphylactoid reactions are not IgE mediated, a one-time exposure to an allergen can induce a reaction. These reactions are difficult to differentiate from anaphylaxis but, fortunately, treatment is the same.

CONCLUSION
While having an allergic reaction present to the clinic often seems like an “easy” case, these patients can quickly become complicated and deadly if not managed immediately and appropriately. Providing oxygen, IV fluid therapy, appropriate medications, and close monitoring can mean the difference between surviving an anaphylactic episode and not. Understanding the different causes and types of hypersensitivities is key to being able to educate clients on how to prevent allergic and anaphylactic responses in the future.

REFERENCES/SUGGESTED READING
Available upon request
INTRODUCTION
Dyspnea is one of the most stressful emergency presentations that face veterinary technicians. We are taught in emergency medicine to treat first what kills first, and respiratory distress can degenerate to respiratory arrest quickly without appropriate interventions. Unfortunately, we as technicians can hasten that degeneration if we are not careful with handling and judicious in the timing of those interventions. These patients are among the most fragile we treat, balancing a fine line between life and death. We must treat them with care to ensure we tip the balance in our patients’ favor at every opportunity.

The American Thoracic Society defines dyspnea as “the subjective experience of breathing discomfort that originates from interactions among various physiological, psychological, social, and environmental factors.” Dyspnea shares common neurologic pathways as those associated with pain in human patients and it is reasonable to believe that veterinary patients also experience distress and unpleasant sensations when suffering from respiratory difficulties. Therefore, gentle handling, and the use of chemical agents to relieve distress and pain is indicated in this patient population.

BASIC RESPIRATORY ANATOMY AND PHYSIOLOGY
The respiratory system consists of the upper and lower airways, the lung parenchyma, and the pleural space. The upper airways include the nose mouth, naso- and oro-pharynxes, and the trachea. The lower airway consists of the bronchi, which each provide oxygen to one lung and branch into bronchioles – the smallest part of the respiratory system that does not participate in gas exchange. The terminal bronchioles lead to bundles of alveoli where gas exchange occurs.

This system is designed to provide oxygen to the bloodstream and remove the cellular waste product carbon dioxide via ventilation. Inhalation brings oxygen into the alveoli where it diffuses into the bloodstream and is carried throughout the body attached to hemoglobin found in red blood cells. Carbon dioxide also uses hemoglobin to travel through the body: from the tissue beds where it is produced as a waste product of cellular respiration and metabolism to the lungs where it diffuses from the pulmonary capillary beds into the alveoli and is subsequently exhaled from the body. The diaphragm and the intercostal muscles of the thorax control inhalation and exhalation so pathology of either of these muscle groups can lead to problems with ventilation. In order to allow for the proper expansion of the alveoli within the lungs the thorax must be able to expand and increase the intra-thoracic space; fluid or air in the pleural space or within the thoracic cavity will severely limit the ability of a patient to increase the intra-thoracic space sufficiently to inflate the alveoli and perform ventilation. Because movement of air is also dependent on a pressure gradient between the atmosphere and the interior of the thorax, any defect in the thoracic wall can impact ventilation as well.

While ventilation refers to the exchange of oxygen and carbon dioxide via the lungs, oxygenation is the amount of oxygen attached to hemoglobin in the bloodstream, which we can measure via pulse oximetry. Oxygenation is key to avoiding hypoxemia, or low levels of oxygen in the blood, which, in turn, can lead to hypoxia – low levels of oxygen in the tissues – and shock, the end stage of which is cellular death.

ASSESSMENT
As with any emergent patient, the first step in treatment is a thorough assessment of the patient’s status, focusing on the ABC’s of triage: Airway, Breathing, and Circulation. Patients in respiratory distress may show several signs, one of the most common being an orthopneic posture. This is characterized by a sternal body position, with an extended neck, and elbows abducted in an effort to open and expand the airways as much as possible to increase ventilation. Often patients who exhibit this posture may be open-mouthed breathing as well. Flaring nostrils, or a sucking in of the lips may also be observed. Asynchronous, or paradoxical, movement of the thorax and the abdomen may be seen; the abdomen and the thorax should move in concert, both expanding and contracting with inhalation and exhalation, respectively. If the thorax and abdomen are moving in opposite directions during the ventilatory cycle, it
is an indication that the patient is using extra musculature to aid in expanding the lungs and can be a sign that exhaustion of the respiratory muscles is imminent.

The vast majority of patients who present in respiratory distress will benefit from oxygen administration on presentation. This can be accomplished in many fashions and must be aimed at reducing a patient’s stress and anxiety. Many patients object to masks being placed over their nose, but may tolerate flow-by oxygen. It is difficult to reach high levels of oxygen supplementation via this method, however, and much oxygen can be wasted. An oxygen hood can be very helpful in your emergency triage area. There are many commercial options available, or an ad hoc hood can be created with an e-collar covered with plastic wrap through which the end of an oxygen tube is fed. If this option is used, be sure that a corner of the wrap is left open to allow for venting of exhaled carbon dioxide. Some practitioners may use an anesthesia induction box to provide short-term, emergency oxygen supplementation for smaller patients. This can be a good option in an emergency when handling of a patient may lead to additional stress, potentially causing the patient to decompensate. The ideal environment is an oxygen cage where the oxygen levels can be set to the desired concentration, and venting and temperature control is provided. These cages can provide high levels of oxygen supplementation while allowing observation of the patient without additional handling.

Recognizing breathing patterns is a key component of assessing patients in respiratory distress. Table 1 shows breathing patterns with correlating affected regions of the respiratory anatomy affected, and the potential cardiac and respiratory causes of the breathing pattern. The veterinary technician plays a key role in these observations and reporting findings to the veterinarian so that appropriate interventions can be instituted in a rapid fashion. For example, if a restrictive breathing pattern is observed – characterized by inspiratory effort and asynchronicity – a thoracocentesis must be performed rapidly to restore the patient’s ability to expand their alveoli appropriately and allow for ventilation. There is little harm in performing a negative thoracocentesis if pneumothorax is suspected, or if there is a suspicion of fluid build-up in the pleural space and experienced clinicians can perform this life-saving procedure rapidly. Therefore, a high index of suspicion must be maintained for pleural space or intrathoracic conditions that may respond to centesis in order to intervene quickly and early in those cases.

Table 1
Breathing pattern | Affected region | Causes
---|---|---
Rapid, labored, synchronous, inspiratory effort | Pulmonary interstitium | Cardiac
• Pulmonary edema due to CHF
Pulmonary
• Interstitial pneumonia
• Neoplasia
• NCPE
• Parenchymal bleeding

Rapid, labored, asynchronous, inspiratory effort | Pleural space | Cardiac
• Pleural effusion
Pulmonary
• Diaphragmatic hernia
• Diaphragmatic paralysis
• Hemothorax
• Pleural effusion
• Pneumothorax

Expiratory effort | Lower airway | Cardiac
• Alveolar flooding due to CHF
Pulmonary
• Acute bronchoconstriction
• Alveolar fluid accumulation
• Tracheobronchial inflammation

Loud, slow, and noisy | Upper airway | • Elongated soft palate
• Laryngeal disease
• Obstruction
• Tracheal disease

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• Tracheal disease |

Table 1. Breathing patterns and affected regions. (Adapted from Rudloff)

Additionally, veterinary technicians must be cognizant of non-respiratory “look-alikes” that may have dyspneic-like symptoms, without a respiratory basis such as metabolic acidosis, anemia, hypovolemia, pain, neurologic lesions, and hyperthermia. Panting must also be differentiated from dyspnea. Both a panting patient and a dyspneic patient may exhibit tachypnea, but a panting patient will not show signs of distress related to air hunger as their ventilation (the amount of gas exchanged with each inhalation and exhalation) remains normal. Dyspnea or “air hunger” ranks among the most difficult experiences in human patients. We can extrapolate that our patients also suffer from a form of pain when in respiratory distress. Therefore, it is advisable to administer sedative and/or anxiolytic medications such as low-dose opioids or benzodiazepines to help patients relax. This relaxation and reduction of anxiety will also allow further diagnostics and therapies to commence, without exacerbating a patient’s distress. While many veterinary technicians may rush to set up the radiography suite for diagnostics, it is advisable to wait until a patient has been stabilized and is breathing with less difficulty before proceeding to obtaining radiographs. A good diagnostic imaging modality that can be used while the patient is receiving supplemental oxygen, and that also allows a patient to maintain a posture that is most comfortable for them is ultrasound and practitioners experienced in TFAST (Thoracic Focused Assessment with Sonography for Trauma) can use it to diagnose many pleural space and intrathoracic pathologies.

In addition to observation, patients should be carefully auscultated for lung sounds on presentation. Crackles or “wet” sounding lungs may indicate pneumonia or pulmonary edema. Wheezes are due to narrowed airways and may indicate airway obstruction of some kind (mucus, inflammation, foreign object). Absence of lung sounds is a particularly alarming finding and should be immediately brought to the veterinarian’s attention. There are many diseases and conditions that may lead to an absence of lung sounds including pneumothorax, hemothorax, pyothorax, or even a lung lobe torsion or diaphragmatic hernia. Observation of breathing patterns, auscultation, and even TFAST can be performed while the
patient is in an oxygen-rich atmosphere (oxygen hood, oxygen cage, flow-by) and with minimal handling or additional stress to the patient.

**MONITORING THE DYSPNEIC PATIENT**

It can be challenging to monitor very fragile patients in as hands-off a manner as possible to avoid further stress and distress. While we are providing oxygen, many patients will tolerate a pulse oximeter probe on their ear, which can provide us with a measurement of oxygen saturation. If we are able to provide enough oxygen to saturate 92% of the hemoglobin in a patient’s bloodstream, we can avoid hypoxemia (assuming the patient is not anemic). While far from perfect, pulse oximetry is an excellent tool for monitoring trends and changes in a patient’s oxygen saturation levels. To monitor ventilation (and many other parameters), capnography is an excellent tool. Capnography does not require intubation: you can place a capnometer on a mask and get a reading that will be close to the end tidal carbon dioxide reading you would receive from an intubated patient. Accuracy of this measurement is improved with a tight-sealing mask, which many dyspneic patients will not tolerate. Another option for measuring end tidal carbon dioxide – and monitoring ventilation – is to modify a nasal prong device to accept side-stream capnography tubing. An example can be found here: http://ehced.org/wp-content/site/tutorials/etco2.pdf

The methods described above are indirect methods of measuring oxygenation and ventilation. The gold standard for measuring both oxygenation and ventilation is collection of an arterial blood sample for a measurement of arterial blood gases. Unfortunately, most patients in respiratory distress cannot be properly restrained for arterial puncture – or venipuncture – without causing additional stress and discomfort. Since many of these patients will require intravenous medication administration, IV catheter placement is an ideal time to collect blood samples. Although these will be venous samples, they can still be used to check electrolyte and serum chemistry values. Also, venous blood gases are an acceptable alternative for assessment of carbon dioxide levels and acid-base status, but cannot be used to accurately assess oxygenation. No attempt at venipuncture should be made until the patient is breathing with less difficulty and showing decreased levels of distress.

Patients in respiratory distress may have higher than normal body temperatures because of the inability to inhale enough air quickly enough to cool themselves. Keeping these patients in a temperature-controlled environment, preferably with active cooling like fans, ice packs, or air conditioners is mandatory. Patients with laryngeal paralysis especially seem to benefit from a fan blowing directly on the face. Serial temperature monitoring can help guide treatment and alert the veterinary technician to changes in the patient.

Other non-invasive monitoring parameters depend on close, careful, and constant observation of these patients. These patients often require intensive nursing care, as their condition can deteriorate rapidly with disastrous outcomes. Changes in posture – from sternal to lateral, or lateral to orthopneic – can be an early indicator of imminent respiratory muscle exhaustion and the need to intubate to support ventilation and prevent respiratory arrest. Changes in breathing patterns, particularly if a breathing pattern shifts from synchronous (both the thorax and abdomen moving in the same direction during inhalation/exhalation) to asynchronous is another sign of exhaustion and the patient may require intubation soon. Serial auscultation can help guide therapy and indicate the need for additional thoracocenteses.

**COMMON CONDITIONS LEADING TO RESPIRATORY DISTRESS**

When considering underlying conditions that may lead to a patient presenting with respiratory distress, it is helpful to localize potential problems using the anatomy of the respiratory system to help localize the area of the problem. This can be done through observation of breathing patterns (see Table 1), as well as history and auscultation.

*Airway Issues:* Starting in the upper airway, conditions such as Brachycephalic Syndrome, collapsing trachea, and laryngeal paralysis (LarPar) are common presentations in the veterinary emergency clinic. These are conditions that obstruct the upper airway and lead to decreased ventilation and oxygenation. Often, they can also lead to hyperthermia, as obstruction restricts airflow and patients are not able to dissipate heat. These patients may exhibit stertor if the obstruction is more rostral: a snoring sound on
either inspiration or expiration. Patients with LarPar or laryngeal obstruction will exhibit stridor: a high-pitched sound on inspiration. Both stertor and stridor may cause loud referred sounds during thoracic auscultation. Emergency treatment of airway obstruction includes oxygen supplementation, and administration of sedative or anxiolytic medications. If the obstruction is a foreign body, or a complete blockage of the upper airway, emergency tracheostomy may be needed until the obstruction can be removed. In extreme cases, obstruction of the airway can lead to non-cardiogenic pulmonary edema (NCPE) and these patients must be monitored carefully for changes in breathing patterns, posture, mucus membrane color, oxygen saturation, or other signs that may indicate an exacerbation of their respiratory distress.

Chest Wall Issues: If the thorax cannot adequately expand and contract, ventilation will be compromised. Fatigue or exhaustion of the respiratory muscles (intercostals and diaphragm) may be involved and will be seen in paradoxical or asynchronous breathing patterns. Trauma may lead to penetration of the thorax, causing a pneumothorax as air rushes into the hole in the chest wall; this is a life-threatening emergency and must be treated immediately with thoracocentesis and possibly chest tube placement. Blunt trauma may cause a flail chest, where a segment of broken ribs cannot move in conjunction with the rest of the rib cage causing extreme pain and an inability to properly ventilate. Neuromuscular diseases (myasthenia gravis, brain or spinal cord lesions) and neurotoxins (Coral snake venom, botulism) may lead to respiratory muscle dysfunction and failure. These patients may require intubation and manual ventilation (or mechanical ventilation) to give the respiratory muscles time to recover, or to allow repair of any chest wall defect.

Pleural Space Issues: If fluid, air, or space-occupying masses infiltrate the pleural space, the lungs cannot adequately expand to allow for proper ventilation. Thoracocentesis should be performed emergently in cases where pleural space conditions are suspected and serves both a therapeutic and diagnostic purpose, as fluid obtained can be evaluated cytologically to determine its nature, or the presence of air can alert the veterinary care team to search for chest wall defects. Patients will display an asynchronous breathing pattern (especially feline patients) with tachypnea and are often orthopneic as well. After thoracocentesis is performed close monitoring is required to ensure that fluid or air does not continue to accumulate in the pleural space.

Lung Parenchyma Issues: Pneumonia is the condition most commonly thought of in terms of diseases of the lung tissue itself. Pneumonia is inflammation of the lung parenchyma caused by an infectious agent (bacteria, virus), a fungus, or by aspiration of fluid into the lungs (drowning, near drowning, aspiration of GI contents). In the veterinary hospital setting, aspiration pneumonia of stomach contents is common following surgical procedures, or in patients with laryngeal paralysis who are unable to protect their airway. When auscultating these patients, loud lung sounds may be heard and may be accompanied by crackles or rhonchi (“wet” sounds). Often patients with pneumonia have a history of worsening cough and may present with a fever (though many pneumonias are not pyretic). Emergency treatment focuses on oxygen supplementation, antibiotic therapy (if bacterial pneumonia is suspected), and supportive care. Pulmonary edema is another parenchymal issue that may present as an emergency. Pulmonary edema is an accumulation of fluid in the lung parenchyma, around the alveoli, which leads to a reduction in ventilation (exchange of oxygen and carbon dioxide). Pulmonary edema can be divided into cardiogenic and non-cardiogenic causes and both will lead to respiratory distress (see DIFFERENTIATING above). Emergency treatment of either cardiogenic or NCPE includes oxygen supplementation in an effort to increase oxygenation. Patients in severe distress, or those who are unable to maintain adequate oxygenation, intubation and manual or mechanical ventilation may be indicated. Other treatments are dependent on the source of the edema and may include diuretics, vasodilatory medications, and bronchodilators.

DIFFERENTIATING BETWEEN CARDIAC AND RESPIRATORY CAUSES OF DYSPNEA
History collection is a key part of determining the underlying cause of a patient’s respiratory distress and can help the veterinary team differentiate between cardiac and pulmonary causes of dyspnea. Radiography will also be a key method of differentiating causes of respiratory distress, which can be frustrating to the emergency practitioner as we must wait to obtain radiographs until the patient is more
stable and breathing with less effort. Some general principles can help guide the veterinary team in the absence of radiographs, however. Clients may report a cough that has acutely worsened, which points to a primary pulmonary tree problem such as tracheal collapse, bronchitis, or pneumonia. On the other hand, a cough due to heart failure is generally of acute onset, soft, and worsening over days to weeks. Patients with heart disease are often cachetic (losing muscle mass and body condition), while patients suffering from a primary respiratory disease are generally able to maintain their body conditions. Patients with primary cardiac dysfunction may have a history or presenting signs that include a heart murmur or gallop rhythm, ascites, exercise intolerance, or syncope. Primary cardiac disease patients generally have a normal or below normal core temperature, while respiratory patients will have a normal or increased temperature.

Once the patient is stable enough for radiographs there are several signs that can be seen that will help differentiate between primary cardiac and respiratory disease. An excellent tool is calculating the vertebral heart score (VHS). Box 1 shows the procedure to follow to obtain this measurement from a lateral radiograph. While not specific in some breeds due to thoracic conformation, a VHS less than 11.4 in a dog can help rule out cardiac causes of respiratory distress. In cats, a VHS of less than 7.9 can help rule out heart disease (in conjunction with other exam findings).

If your team has access to echocardiography, it is the gold standard diagnostic tool for assessment of cardiac function and can be utilized at the cage-side, making it a very valuable tool in the emergency department for differentiating between primary cardiac and primary respiratory dysfunction.

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**Box 1.** Vertebral Heart Score calculation. *Boxers, bulldogs, Boston terriers, Cavalier King Charles spaniels, Labrador retrievers, pugs, Pomeranians, and whippets have been found to have much higher scores (on average) than other breeds.

**CONCLUSION**
Low stress handling and oxygen supplementation are the key points to remember when caring for respiratory distress patients.

**REFERENCES**
*Available on request*
As medical cannabis becomes legal in more states, and public opinion continues to shift toward acceptance of the use of cannabis for both medical and recreational use, more and more patients are using cannabis not only for their own medical conditions, but also for their pets’ conditions. In order to support our clients and our patients, it is necessary for the veterinary staff to be aware of available products and their uses in small animal veterinary medicine. These proceedings will also discuss the issue of inadvertent cannabis ingestion by dogs leading to toxicities and treatment methodologies.

**TERMINOLOGY**

*Cannabis* is the plant from which both “marijuana” and hemp derive; the difference is in the way the plant is bred. Some strains are cultivated to maximize the content of psychoactive substances and other cannabinoids (“marijuana” or “cannabis”), while others are cultivated primarily for fiber, oil, or other substances (“hemp”). *Industrial hemp* is defined by the Federal government as a *Cannabis sativa* plant with <0.3% of the psychoactive component or *cannabinoid* (specifically THC). These proceedings will refer to medical cannabis, rather than medical marijuana, as the word “marijuana” has many negative connotations and was given to the plant by people only interested in banning its use (see History/Timeline, below). A *cannabinoid* is any chemical compound that interacts with receptors found throughout the body and can be divided into three main categories: *endocannabinoids*, *phytocannabinoids*, and *synthetic cannabinoids*. *Endocannabinoids* are produced in cellular membranes throughout the body on an as-needed basis. *Phytocannabinoids* are found in plants like *Cannabis sativa*, *Echinacea purpurea*, and others that interact with receptors in the endocannabinoid system. *Synthetic cannabinoids* are created outside of the body and designed to interact with cannabinoid receptors. *Terpenes* are compounds that provide plants with their aroma. Similarly, *flavonoids* are compounds that provide plants with their flavor.

**HISTORY / TIMELINE** *(adapted from medicalmarijuana.procon.org)*

2900 BC - AD: The first written mention of the use of cannabis as a medicine is found in China in 2900 BC. Chinese herbalists and medicine practitioners continue to explore the use of cannabis for many conditions and the earliest written reference to medical cannabis is published in the Chinese Pharmacopeia in 1500 BC. Cannabis is used extensively throughout Asia, India, and the Middle East for conditions ranging from glaucoma, to leprosy, earaches, edema, and inflammation. It was also used as an anesthetic agent.

30 AD: Chrism, a cannabis-based anointing oil, is mentioned in the New Testament as the oil Jesus used to anoint his disciples. The recipe for this oil – found in Exodus – calls for between six and nine pounds of cannabis to be steeped in oil and then used for both anointing and fumigations.

70 – 1700s: References to cannabis for medical use/treatments continue to be found throughout China and the Middle East with some calling it medicine and others warning of the psychotropic effects. In the Middle Ages in England, hemp was crucial for every herbalist to carry and in China it is used to treat vomiting, parasite infections, hemorrhage, diarrhea and dysentery, and as an appetite stimulant. As the Jamestown settlers arrived in North America, they brought hemp with them and hemp fiber was an important export for the colonies; so much so that in 1762 Virginia imposed penalties on any who did not produce hemp on their land. Both George Washington and Thomas Jefferson grew hemp on their plantations. In 1799, Napoleon invaded Egypt and returned to France with cannabis, which was studied for its pain relieving and sedative effects, making cannabis more accepted in Western medicine.

1800s – 1905: Dr. William O'Shaughnessy was a British army surgeon serving in India who re-introduced cannabis into British medicine upon his return to England. It was used widely for many conditions including muscle spasms, menstrual cramps, and rheumatism. It is rumored that Queen Victoria may have used cannabis tincture (extract in alcohol) to treat her own menstrual cramps as her personal physician wrote extensively on the use of cannabis for many ailments. Additionally, cannabis was used as an anti-convulsant for those suffering from tetanus, rabies, and epilepsy. Several studies at this time were...
published including those by French psychiatrist Jacques-Joseph Moreau showing that cannabis was an appetite stimulant, a sleep aid, and a treatment for headaches. In 1850 cannabis was added to the United States Pharmacopeia as a treatment for neuralgia, tetanus, typhus, cholera, rabies, dysentery, alcoholism, opiate addiction, anthrax, leprosy, incontinence, gout, insanity, menstrual and uterine bleeding, appetite stimulation, and many others. In 1889, a study was published in *Lancet* on the use of cannabis to relieve symptoms of opium withdrawal.

1906: President Roosevelt signed the Food and Drugs Act (aka the Wiley Act), which established regulations for product labeling and specifically stated that packaging must have a statement of the quantity of alcohol, morphine, opium, cocaine, heroin, chloroform, cannabis, or any derivative thereof.

1911 – 1929: Fifteen US states pass anti-marijuana laws while US pharmaceutical farms grow 60,000 pounds of cannabis annually. This made the US self-sufficient in hemp. In 1925, the League of Nations signed a multilateral treaty that restricted the use of cannabis to scientific and medical purposes only and in 1928 cannabis is added to the UK’s Dangerous Drugs Act.

1930s: Parke-Davis and Eli Lily were selling standardized extracts of cannabis for analgesia, sedation, and as an anti-convulsant. In 1930, Harry Anslinger was appointed as the first Commissioner of the Federal Bureau of Narcotics. Anslinger was the architect of national prohibition and believed that cannabis caused insanity and increased criminality. In 1933 publisher William Randolph Hearst joined the anti-cannabis fray, publishing sensationalized stories linking violence to cannabis consumption. In his newspapers, he dropped the terms “cannabis” and “hemp” and replaced them with “marijuana” which linked cannabis to minorities (most notably Mexicans who were immigrating into the west in large numbers). Both Anslinger and Hearst used racially charged language and blamed marijuana use for everything from rape to dissolution to murder. The film Reefer Madness is released in 1936 which purported to prove that marijuana ruins lives and leads to violence and promiscuity.

In 1937 the Marihuana Tax Act is passed, with strong support from Anslinger and over the objections of the American Medical Association who argued for the medicinal use and effects of cannabis. This law imposed registration and reporting requirements on growers, sellers, and buyers of cannabis, leading to a decline in prescriptions because doctors found it too difficult to deal with the extra work imposed by the law. By 1938, both cannabis and hemp are illegal in all states.

1964: Dr. Raphael Mechoulam is the first person to identify delta-9-tetrahydrocannabinol (THC) as the primary psychoactive component of cannabis. He is also the first to synthesize THC.

1970s: In 1970, Congress passes the Controlled Substances Act, which established the scheduling structure used for controlled drugs to this day. Cannabis was placed in Schedule I: drugs with a high potential for abuse, no currently accepted medical use, and a lack of accepted safety data. A presidential commission (Schafer Commission) recommended removal of cannabis from the scheduling system in 1972 but President Nixon rejected this recommendation, having declared war on drugs in 1971.

1980s: Marinol, the first synthetic cannabinoid, is approved by the FDA for the treatment of nausea. Patients in the clinical trial reported less nausea and fewer unwanted side effects when using the whole plant, however.

1990s: In 1990 researchers at the National Institute of Mental Health discover the cannabinoid receptor system in the human brain and in 1992 Dr. Mechoulam identified the first endogenous cannabinoid (aka endocannabinoid): anandamide. In 1996, California becomes the first state to legalize the use of cannabis to aid in the treatment of AIDS, cancer, muscular spasticity, migraines, and other disorders. California was followed by Alaska, Oregon and Washington which all legalized medical cannabis in 1998.

2000s: Research into the endocannabinoid system continues and more and more states are legalizing cannabis for medical use. The DEA is currently considering rescheduling cannabis to a less-restrictive schedule, which should boost research and the development of new pharmaceuticals to take advantage of the largest receptor system in the human body.
THE ENDOCANNABINOID SYSTEM

The endocannabinoid system (ECS) is believed to be the largest receptor system in the mammalian body. Receptors are located in the brain and throughout both the central and peripheral nervous system; they are also expressed on the membranes of immune cells throughout the body. The ECS serves as a neuromodulatory and immunomodulatory system and is vital to maintaining homeostasis and aiding the body’s own stress-recovery systems. The ECS has many functions including neuroprotection, pain modulation, motor activity regulation, cardiovascular controls, and antiproliferative actions in neoplastic cells. In the central and peripheral nervous system, the ECS reduces the influx of calcium at the presynaptic neuron, which inhibits the release of excitatory neurotransmitters. This inhibitory effect lessens pain responses, as well as other excitatory processes such as muscle tremors and spasticity. In the case of inflammation specifically, the ECS increases receptor expression and local endocannabinoid levels at the site of inflammation, leading to a down-regulation of the production of inflammatory proteins and chemotaxis of inflammatory cytokines.

The ECS has two primary receptor types known as cannabinoid receptor type 1 (CB1) and cannabinoid receptor type 2 (CB2). CB1 receptors are highly expressed in the brain but are also found in organs and tissues throughout the body. In the mammalian brain, CB1 receptors are one of the most abundant G protein-coupled receptors found, allowing both endocannabinoids and exogenous cannabinoids to cross the blood brain barrier (BBB) and exert effects on the brain. CB1 receptors in peripheral organs including adipocytes and Kupffer cells in the liver may exert some control over metabolism, and CB1 receptors in the gastrointestinal (GI) tract modulate motility, inflammation, and secretion. These receptors are also found in vagal nerve terminals in the GI tract involved in gut-brain signaling and perhaps playing a role in modulating feeding behavior. CB2 receptors are expressed almost exclusively in immune and blood cells, where they perform immunomodulatory functions and mediate cytokine release. They have also been found to inhibit the activation of the cell-mediated immune process (e.g. IMHA) and to inhibit chemotaxis. CB2 receptors are also found throughout the GI tract and are a potential therapeutic target for a number of inflammatory bowel diseases, with CB2 agonists proving to reduce gut motility in human irritable bowel syndrome patients. CB2 receptor agonists are currently being researched as treatments for both inflammatory and neuropathic pain. Interestingly, the ECS (and not the endo-opioid receptor system, triggered by endorphins) is now thought to be the system responsible for the “runner’s high” that people experience after exercise.

At this time, research has identified five endocannabinoids – the endogenous ligands for the cannabinoid receptors in the ECS. The first identified was anandamide (N-arachidonoyl ethanolamide, AEA, the “bliss molecule”) followed by 2-arachidonoyl glycerol (2-AG). These molecules have been the most extensively studied and are thus far the best understood. Lesser-understood compounds are Noladin ether, Virodhamine, and NADA.

Phytocannabinoids are found in Cannabis spp. as well as other plants (such as Echinacea purpurea). Phytocannabinoids bind to both CB1 and CB2 receptors in the ECS and exert similar effects to endocannabinoids. More than 80 phytocannabinoids have been discovered in Cannabis with 20 of those currently under active study. The most famous of these is tetrahydrocannabinol, also known as THC. THC is the psychoactive component of Cannabis, with modern strains of the plant being cultivated and bred to increase the psychoactive effects through higher and higher percentages in the plant. THC also has medicinal properties and is used against glaucoma, insomnia, PTSD, and anxiety disorders. Cannabidiol (CBD) is the second half of the “power couple” of cannabis. CBD is non-psychoactive and has been found to be efficacious in seizure and pain control, while also being used for its anti-inflammatory, analgesic, anxiolytic, antipsychotic, and anti-carcinogenic effects. CBD modulates the psychoactive effects of THC and helps to temper the “high” experienced. Cannabinol (CBN) is closely linked to CBD and is sometimes referred to as “CBD Lite”. It functions primarily as a sleep aid, with recent research showing some promise in its use as a topical treatment for methicillin-resistant Staph aureus (MRSA) infections. In the UK and Canada Sativex® (nabiximol) mouth spray has been approved to treat neuropathic pain, cancer pain, spasticity, overactive bladder, and symptoms of multiple sclerosis. It is a 1:1 ratio of CBD to THC extracted from the Cannabis plant.
Synthetic cannabinoids have been developed in an effort to avoid the legal issues surrounding the use of 
cannabis for medical purposes and to try to isolate compounds for specific therapeutic effects. With the 
advent of legalization of cannabis for medical use in several states, the need for synthetic cannabinoids has 
lessened but they still have an important role to play in treating patients who may be reticent to buy and use 
cannabis, due to its stigmatization over the last 80 years. Many scientists also wish to isolate the 
compounds that provide specific therapeutic effects, minimizing the need for using the entire plant. The first 
FDA-approved synthetic cannabinoid is Marinol® (dronabinol), which is approved for the treatment of 
anorexia associated with weight loss in AIDS patients. Another FDA-approved synthetic cannabinoid is 
Cesamet (nabilone), which is approved for the treatment and prevention of nausea and vomiting associated 
with chemotherapy. Both dronabinol and nabolone are synthetic analogs of THC and, thus, bind to both CB1 
and CB2 receptors. Nabilone is used off-label to treat chronic pain (especially associated with fibromyalgia), 
Parkinsonian muscle tremors, multiple sclerosis, and inflammatory bowel disease.

Synthetic cannabinoids are reported by patients to be less effective treatments compared to the use of the 
whole plant. This is attributed to the "entourage effect" which is not yet fully understood. For example, 
*Cannabis* contains over 100 different terpenes, which interact synergistically with phytocannabinoids and 
provide mediation for the ECS by modulating the psychoactive effects of THC, increasing its therapeutic 
index by modifying how much THC passes through the blood brain barrier. Additionally, terpenes have 
effects as serotonin uptake inhibitors, norepinephrine enhancers, GABA augmenters, and dopamine activity 
enhancers. Some of the terpenes that have been researched include caryophyllene (gastroprotective, auto-
immune modulator), myrcene (sleep aid, analgesia, alleviates depression), and limonene (mood elevator, 
anti-fungal, anti-bacterial, anti-neoplastic). Beta-caryophyllene is anti-inflammatory molecule, found in 
rosemary, basil, cloves, and black pepper and is an FDA-approved additive for food products. Another 
component in the entourage effect is the impact of flavonoids, whose role is still not well understood.

**VETERINARY MEDICAL CANNABIS USE**

Although there is limited research in the United States, particularly in regards to veterinary species, many 
owners are using cannabinoids to treat a number of different conditions in their own pets. There are several 
companies providing products for the small animal market in various formulations and combinations of 
cannabinoids. Anecdotal reports collected by these companies show that owners are using cannabis 
products to treat pain, seizures, arthritis, anxiety, irritable bowel syndrome, nausea, and inappetance; these 
are conditions for which human patients are also using medical cannabis. Because there is little research in 
therapeutic uses in small animal medicine, dosing and product selection has been done on an ad hoc basis 
as requested by owners. Many of the companies providing medical cannabis for veterinary use have 
developed dosing information and are happy to consult with veterinarians and owners interested in 
recommending cannabis for their patients and pets. Additionally, the American Holistic Veterinary Medical 
Association is encouraging research into the safety, dosing, and uses of cannabis in veterinary species. To 
that end, they conducted an online consumer survey of owners who have used medical cannabis for their 
pets. According to the survey conducted by researchers at the Colorado State University Veterinary School 
(published Spring 2016 in the *Journal of the American Holistic Veterinary Medical Association*), 93% of all 
respondents favored the use of medical cannabis over some medications. Dog owners reported that 
medical cannabis helped either a moderate amount or a lot for:

- Pain relief (95%)
- Age-related changes in behavior including sleep/wake cycle disturbances, excessive vocalization 
  and neediness, and some repetitive behaviors (93%)
- Seizures (92%)
- Inflammation (92%)
- Sleep quality (89%)
- Anxiety relief (83%)
- Nausea reduction (82%)
- Muscle spasms (79%)
- Anti-cancer activity (73%)
- GI issues (unspecified) 71%
Cat owners reported beneficial effects for:

- Pain relief (100%)
- Sleep quality (96%)
- Inflammation (90%)
- Nausea reduction (86%)
- Anti-cancer activity (82%)
- Skin conditions (unspecified) 75%

The most common side effects reported in both dogs and cats were sedation and increased appetite. Most respondents reported they had not spoken with their veterinarian regarding their use of medical cannabis (43%) and that they opted to begin using medical cannabis because they thought it would work as an adjunct to therapies they were already using (50%). 89% of respondents rated medical cannabis as very safe.

In 2018, a double-blind, placebo controlled, double-crossover study (https://www.frontiersin.org/articles/10.3389/fvets.2018.00165/full) was published demonstrating safety and efficacy of a whole-plant, hemp-based CBD product (ElleVet®) for the treatment of canine osteoarthritis. This study demonstrated that a dose of 2 mg/kg of CBD provided pain relief and increased mobility, as shown by force-plate gait analysis tests performed on the study dogs.

Cannabis has been found to be useful in treating pediatric epilepsy in human patients and is beginning to be more widely used in veterinary neurology as well, taking advantage of the ECS’s down-regulatory effect on neurons. Stimulation of CB1 receptors has been found to limit cell death after damage from excitotoxic lesions and CB2 receptors provide immunomodulatory and anti-inflammatory effects by reducing the expression of inflammatory proteins. Cannabidiol (CBD) in particular has been found to provide neuroprotection during acute brain injury and ischemic events.

A double-blind, placebo controlled pilot study published in 2019 in JAVMA (https://avmajournals.avma.org/doi/abs/10.2460/javma.254.11.1301) demonstrated safety and efficacy of CBD in reduction of seizures in canine patients, though the difference between the treatment group and the placebo group was not found to be statistically significant. A full study is still enrolling subjects, and it may show that a higher dose than 2 mg/kg of CBD may be required for better seizure control in dogs.

Determination of the appropriate dose of cannabis for companion animals with different conditions has been challenging, due to the individual nature of the ECS and its response to cannabinoids, from both endogenous and exogenous sources. While we can use the published data as a guideline, most dosing is done on a case-by-case, trial-and-error basis. Because the therapeutic index of cannabis is very large, it is considered safe to experiment with different doses to determine what works best for each animal and each disease process. The recently established certification program for Veterinary Cannabis Counselors provides credentialed veterinary technicians who learn about clinical applications of cannabis in companion animals and can provide cannabis harm reduction education to clients. More information can be found at www.veterinarycannabis.org.

While cannabis therapeutics can be expensive, at least one pet insurance company (Trupanion) promises to cover medical cannabis treatment when it is “recommended” by a veterinarian. However, “recommendation” is challenging in several states, including those where cannabis and its derivatives have been legalized or decriminalized, as some Veterinary Medical Boards have barred veterinarians from even discussing the use of cannabis in their patients, while others are completely silent on the issue (see CURRENT LEGAL STATUS for more information). Those who filed claims for reimbursement with Trupanion (according to a 2015 report) reported using medical cannabis: alongside traditional treatments for cancer; to reduce seizure severity and/or frequency; to treat chronic and neuropathic pain; as an anti-
inflammatory agent; as an anti-emetic agent and appetite stimulant; and as an anxiolytic. The vast majority of Trupanion claims involving cannabis were for reimbursement for treatment of toxic ingestions, to the tune of $78,000 in suspected cannabis intoxication claims, with an average cost of $525 per claim.

**CANNABIS INTOXICATION / TOXICITY**

The increase in the use and acceptance of both medical and recreational cannabis has resulted in an increase of reported cases of cannabis intoxication seen in companion animals. In the first quarter of 2019, the ASPCA Animal Poison Control Center recorded a 765% increase in cannabis-related calls compared to the same time period the previous year. Pet Poison Helpline (PPH) has reported a 448% increase in calls due to cannabis intoxication between 2012 and 2018, and the actual number of cannabis toxicity cases seen in practice is undoubtedly much higher than that, as many practitioners do not feel the need to consult with a toxicologist to treat cannabis toxicity. 98% of the calls to PPH were in regards to dogs. Trupanion has also seen an increase in claims for treatment of cannabis intoxication since 2013. The top five states in which Trupanion has paid claims are California, Washington, New York, Colorado, and Florida. While cannabis intoxication is an increasingly common presentation to veterinary facilities, mortality associated with the intoxication is very low; the lethal dose in dogs is > 9 g/kg of THC ingested, with the exact dose still unknown. A 2012 study in the *Journal of the Veterinary Emergency and Critical Care* (JVECC) reported two deaths in a group of 125 dogs treated for cannabis intoxication in two facilities in Colorado, but the deaths could not be attributed to cannabis due to confounding from coingestions. With newer, more concentrated forms of cannabis reaching the market (i.e. vape cartridges, shatter, wax), it is reasonable to expect more severe intoxications to present at veterinary facilities.

Dribbling urine is perhaps the cardinal clinical sign that veterinary team members think of when they think of cannabis intoxication. However, the 2012 JVECC article referenced above found ataxia to be the predominant clinical sign in dogs, with 88% of cannabis intoxicated dogs presenting with ataxia. Only 47% of dogs in the study exhibited urinary incontinence. Other clinical signs included disorientation (53%), mydriasis (48%), hyperesthesia (47%), tremors or twitching (30%), and vomiting (27%). More than half the dogs (58%) were treated on an outpatient basis, showing that few patients require hospitalization for this toxicity.

Treating cannabis intoxication is made difficult by coingestions of substances that are toxic to dogs including chocolate, xylitol, macadamia nuts, or other drugs. If treating only cannabis ingestion or exposure, providing supportive care is the mainstay of treatment including fluid therapy for blood pressure support (if needed), monitoring of respiratory rate and other vital signs, and protecting patients from further harm (i.e., aspiration pneumonia if vomiting while somnolent). Activated charcoal may be used within four to six hours of ingestion and benzodiazepines are indicated if a patient is tachycardic or shows signs of excessive CNS stimulation. Intralipid emulsion (ILE) therapy has been used to treat cannabis intoxication, due to the very high lipophilicity of cannabis, but it is rarely indicated. Because the FDA does not have the power to regulate cannabis products, there may be issues of cross-contamination with pesticides, insecticides, fungicides, or fertilizers used in the cultivation of the plant, or mycotoxins present due to poor product handling and storage after harvest. When the plant is concentrated during distillation or extraction (i.e., into tinctures, butters, hashish, shatter, or other concentrated forms), these substances will also be concentrated and may exert a toxic effect. Unfortunately, it is virtually impossible to know what may be present in the ingestion.

**CURRENT LEGAL STATUS (JUNE 2019)**

In most jurisdictions where cannabis is legal for medicinal use, veterinarians have been excluded from the right to recommend it for their patients. Because cannabis is still classified by the DEA as a Schedule I substance, it cannot be prescribed by any practitioner – human or veterinary. In California, the Veterinary Medical Board has provided guidance advising veterinarians against recommending cannabis for their patients, and against selling hemp-based products directly from their clinic. A bill (AB 2215) passed the California legislature in 2018 that gives veterinarians the ability to discuss cannabis as a treatment for their patients; a similar bill is currently pending in New York (S8772/AB10104). While AB2215 gave California
veterinarians the ability to discuss cannabis use in their patients, it does not allow them to recommend cannabis as a therapeutic. A new bill (SB67) is pending to remedy this situation.

Most recently, the 2018 Farm Bill, which includes the 2018 Hemp Farming Act, was passed and signed into law. This Act, sponsored by Senator Mitch McConnell (R-Kentucky), fully legalized hemp (defined as Cannabis plants containing less than 0.3% THC) and all its derivatives. In addition, the FDA has just approved a CBD medication derived from hemp for the treatment of severe childhood seizure disorders (Epidiolex®). This approval further confuses the current legal status of CBD. The FDA has just held public hearings regarding the regulation of hemp-derived CBD products, but there is no news on the DEA’s classification of Cannabis. Legal experts expect to see a re-scheduling of CBD derived from hemp and/or marijuana soon.

CONCLUSION

While many veterinary practitioners want to see more evidence as to the efficacy of cannabis products as therapeutics, our clients are already using these substances for many different conditions in their animal companions. It behooves us as veterinary professionals to help our clients make safe choices for their pets and to educate ourselves on the potential uses of this powerful plant.

REFERENCES

Available on request.
Sugar High!  Nursing the DKA Patient

Liz Hughston, MEd., RVT, CVT, LVT, VTS (SAIM, ECC)
VetTechXpert, San Jose, CA

Pathophysiology of Diabetic Ketoacidosis:

- **Deficiency of insulin**
  - Anabolic steroid (hormone) produced by the pancreas
  - Glucose cannot get inside cells without it = hyperglycemia
- **Excess of stress hormones**
  - Glucagon, cortisol, epinephrine
  - Usually the result of some underlying condition (pancreatitis, infection)
- **Hyperglycemia**
  - Causes an osmotic diuresis = dehydration
  - Increases osmolarity of blood = tissue and cellular dehydration
  - Cells resort to using proteins and fats for energy = formation of ketone bodies
- **Ketonemia**
  - 3 ketone bodies formed: acetone, acetoacetate, beta-hydroxybutyrate
  - Ketones lower the blood pH, resulting in a metabolic acidosis
- Often the first indication that a pet is diabetic, though can result from unregulated diabetes

Typical history reported by owner includes:

- Polyuria
- Polydipsia
- Polyphagia
- Weight loss
- Vomiting
- Lethargy

Signs often seen at presentation:

- Plantigrade stance
- Dehydration
- Tachypnea or Kussmaul respirations (long, deep respirations characterized by expiratory effort)
- “Fruity” or acetone odor to breath
- Low blood pressure
- Decreased level of consciousness
- SHOCK

Recommended diagnostics:

- Blood glucose measurement
- Urinalysis
  - Include culture and MIC
- Serum or urine ketone check
- Electrolytes
- PLi
- Blood gas
- Imaging
  - Abdominal ultrasound
  - Thoracic radiography

Laboratory abnormalities:

- Blood glucose
  - Generally high to very high (>250mg/dL)
- Urinalysis
  - Glucosuria
- Ketones
  - Isothereuria
  - +/- proteinuria
  - Signs of infection (bacteria, WBC’s)
  - Culture + MIC should be recommended
- Ketones
  - If negative on urine, check serum
  - If negative on both, but suspicion is high, mix small drop of hydrogen peroxide to break down beta-hydroxybutyrate ketone body into a form that will react with the pad
- Electrolytes
  - DO NOT OVERCORRECT!
  - Many values will normalize with rehydration, correction of hyperglycemia, and resolution of ketonemia
  - Sodium
    - Hyperglycemia will falsely lower sodium concentrations
    - Osmotic diuresis = hyponatremia
    - Will usually normalize with correction of hyperglycemia
  - Potassium
    - May be high, normal, or low at presentation (will usually require supplementation)
    - Total body depletion + insulin therapy + osmotic diuresis = hypokalemia
    - Safe supplementation = 0.5mEq/kg/hr
- PLi
  - Pancreatitis is often a concurrent finding
  - Low fat diet is warranted if PLi is abnormal
- Blood gas
  - Metabolic acidosis due to ketonemia
    - Low Ph with low HCO3
  - May see respiratory compensation (decreased PCO2) or CO2 may be normal
- Imaging
  - Abdominal ultrasound
    - Pancreas: generally hyperechoic pancreas
    - Adrenals: hyperadrenocorticism is a common concurrent finding
  - Thoracic radiography
    - Rule out pneumonia or other inflammatory conditions in the thorax that can contribute to DKA

**IV Access:**
- Consider patient needs
  - Long term (3-5 days) fluid therapy
  - Need for sampling
  - Need for dextrose supplementation (at possibly high levels)
- Peripheral needed at emergency presentation for correction of shock
- Consider central lines
  - Multiple lumens allow for fluids, sampling, TPN/PPN if needed
  - PICC
  - IntraCath

**Therapies:**
- IV Fluids
  - Which fluid to use?
    - Buffered crystalloid (Plasmalyte, Normosol-R, LRS)
      - Pros: contain potassium, buffered, isotonic
      - Cons: may be more expensive than saline, not as much sodium as saline
    - 0.9% NaCl – traditional fluid of choice
• Pros: contains sodium to help correct hyponatremia, inexpensive, isotonic
• Cons: may lead to overcorrection of hyponatremia once hyperglycemia is corrected, no potassium, no buffer so potential to worsen metabolic acidosis
  o Replace deficit in the first 12-24 hours of therapy
    • Replacement requirement:
      • Body weight (kg) x % dehydration = deficit (L)
      • Maintenance requirement = 40-60ml/kg/day
  o Delay insulin therapy for 1-2 hours in cases of severe dehydration
    • IV fluid therapy alone will begin to decrease blood glucose and serum ketone concentrations
    • Starting insulin before rehydration may worsen hypokalemia
    • Rehydration will begin to correct electrolyte abnormalities
  o Potassium supplementation
    • Do NOT exceed 0.5mEq/kg/hr (aka “K-MAX”)

<table>
<thead>
<tr>
<th>Measured potassium (mEq/L)</th>
<th>mEq KCl to add to 1L IVF</th>
<th>Max rate of infusion (ml/kg/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2.0</td>
<td>80</td>
<td>6</td>
</tr>
<tr>
<td>2.1 – 2.5</td>
<td>60</td>
<td>8</td>
</tr>
<tr>
<td>2.6 – 3.0</td>
<td>40</td>
<td>12</td>
</tr>
<tr>
<td>3.1 – 3.5</td>
<td>28</td>
<td>18</td>
</tr>
<tr>
<td>3.6 – 5.0</td>
<td>20</td>
<td>25</td>
</tr>
</tbody>
</table>

(from DiBartola, *Fluid, Electrolyte, and Acid-Base Disorders*)

• Insulin
  o Corrects hyperglycemia
  o Corrects ketonemia
  o Goal is to decrease BG by 50-75mg/dL/hr
  o Delivery:
    • Intermittent IM dosing
      • Initial dose 0.2 U/kg, then 0.1 U/kg until BG <250mg/dL
      • Either q1h or q2h
    • CRI
      • 2.2 U/kg for dogs; 1.1 U/kg for cats in 250ml bag of 0.9% NaCl
      • Run at least 50ml of solution through IV tubing as insulin will adsorb to the plastic
      • Needs its own IVC
      • Check BG q2h and adjust CRI as needed

<table>
<thead>
<tr>
<th>BG (mg/dL)</th>
<th>Dextrose supplementation in IVF</th>
<th>Insulin CRI (ml/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 250</td>
<td>0%</td>
<td>10</td>
</tr>
<tr>
<td>200 – 250</td>
<td>2.5%</td>
<td>7</td>
</tr>
<tr>
<td>150 – 200</td>
<td>2.5%</td>
<td>5</td>
</tr>
<tr>
<td>100 – 150</td>
<td>5%</td>
<td>5</td>
</tr>
<tr>
<td>&lt; 100</td>
<td>5%</td>
<td>discontinue</td>
</tr>
</tbody>
</table>

(from DiBartola, *Fluid, Electrolyte, and Acid-Base Disorders*)

Monitoring:
• Perfusion and hydration status
  o Frequent (q2-4h) monitoring of forward perfusion parameters
    • Mentation is very important: tells you about BG, perfusion
    • Check pulse quality with every set of vitals
- Ensure correction of shock
- **IV Access sites**
  - Phlebitis
  - Signs of infection
  - Strict aseptic technique when handling central lines
- **Blood Glucose**
  - Check every 1-2 hours
  - Monitoring response to insulin therapy
  - Provide dextrose supplementation PRN
- **Urine production**
  - Normal = 1-2ml/kg/hr
  - These patients may exceed this amount!
  - Glucosuria = osmotic diuresis
  - Ensure matching ins and outs
  - Consider urinary catheter for quantification of UOP
- **Ketones and electrolytes**
  - Clinician preference but usually between 2 and 4x per day
- **Kidney values**
  - To monitor hydration status – usually SID
- **Nutrition**
  - Ensure that patient is eating
  - Anti-emetics (metoclopramide, maropitant) may be indicated
  - If not eating >3 days (including at home), consider NG/NE tube feeding
  - Cats: High protein, low carbs
  - Dogs: High fiber, low fat
- **Recumbent patient care**
  - Cleanliness is key!
  - Avoid urine scald with frequent bedding checks and changes PRN
  - Ensure regular walks for dogs
  - Rotate recumbent patients
    - Maintain integrity of integument
    - Prevent atelectasis
  - PROM, massage
The Solution to Pollution: Top Toxicity Tips
Liz Hughston, MEd., RVT, CVT, LVT, VTS (SAIM, ECC)
VetTechXpert, San Jose, California

Poison Control Experts
Consultation with an animal poison control center is recommended when dealing with any toxicity in your clinic or hospital. These centers are open 24/7 and staffed with specialists in Toxicology with an encyclopedic knowledge of toxicants and their treatment in many veterinary species. Consultations with these services are very reasonable, and one fee provides you with unlimited follow-up as needed while treating that patient.

ASPCA Animal Poison Control Center: 1-888-4ANI-HELP (1-888-426-4435)
- www.aspca.org/pet-care/animal-poison-control
- $65 consultation fee

Pet Poison Helpline: 1-855-764-7661
- www.petpoisonhelpline.com
- $59 consultation fee

Many toxic product manufacturers may also cover the cost of consultation with poison control centers, or may have treatment information for exposure to veterinary species. Having the owner call the manufacturer may be helpful in many cases.

Basics of Treating Toxicities
As in any emergency presentation, the first priority must be ensuring that the patient is stable by performing a thorough primary survey, starting with the ABCs: Airway, Breathing, and Circulation. If a patient presents seizing or in shock, these life-threatening conditions must be ameliorated prior to instituting treatment for the specific toxicant. While the patient is being stabilized, a history can be obtained from the owner, including the specifics of the toxicant involved. When gathering this information, it is critical to obtain as much information about the toxicant as possible. Ideally the owner will bring any packaging or bottles along with the pet at presentation. If not, web searches can be helpful for identification of active ingredients and contents of the product if the owner knows the brand name. Other important history information to gather includes the length of time the animal has been exposed to the toxicant (i.e. how long since ingestion or application), the route of exposure (i.e. topical, ingestion, inhalation), and how much of the toxicant the pet was exposed to.

Decontamination
Once the patient is stable, the toxicant has been identified, and the details of the exposure are known, measures should be taken to prevent further absorption of the toxicant. Different decontamination methods are employed depending on the method of exposure and the patient’s status at presentation.

**External decontamination:** In cases of topical exposure (i.e. alkali cleaning agents, chemicals, pyretherins in cats), thorough bathing in tepid or room temperature water with degreasing dish soap (such as Dawn®) is indicated with copious rinsing to be sure that the agent is completely washed away. Rinsing of the affected area should continue for a minimum of 10 minutes, and longer if signs persist. Extra care must be taken when bathing patients who are recumbent or with reduced responsiveness or reflexes to avoid aspiration while bathing. Ensure that the patient is thoroughly dried after rinsing and check core body temperature frequently to avoid hypothermia, especially in pediatric, geriatric, or debilitated patients. Patients exposed to chemical agents, particularly alkali agents, will likely need extensive wound care in the post-decontamination period. Those patients exposed to alkali agents require longer rinsing periods to help remove and dilute as much of the agent as possible and prevent it from penetrating more deeply in the patient’s tissues.

**Internal decontamination:** In cases where a toxicant was ingested, there are several methods we can employ to prevent absorption from the GI tract:

**Emesis induction:** In many cases, inducing emesis is the first step in GI decontamination, which – if successful – will remove between 40 and 80% of stomach contents. If a patient has altered mentation, is recumbent, or lacks a gag reflex, emesis is contraindicated. Emesis is also contraindicated in the ingestion of agents that are corrosive, alkali, acidic, or hydrocarbons. Inducing emesis should ideally be done in the controlled environment of the
animal hospital, under supervision of trained veterinary staff. Emesis induction is far from a benign procedure and can lead to dangerous sequelae, including aspiration pneumonia, which can be a life-threatening complication.

In dogs, the pharmaceutical agent of choice for inducing emesis is apomorphine (aka “apo”). Apo binds to dopamine receptors in the chemoreceptor trigger zone (CRTZ), the area of the brain responsible for inducing nausea. Apo comes in pill form, which can be crushed and placed in the conjunctival sac; vomiting usually begins within five to ten minutes. While relatively effective at inducing emesis, administering apo in this manner can lead to conjunctival irritation, as well as corneal damage. Apo is compounded into an injectable agent by several compounding pharmacies and can be administered IV; vomiting usually begins two to three minutes after administration. Most patients vomit four or five times, but nausea may persist after the stomach is “emptied” so administration of an anti-emetic such as maropitant is indicated, particularly if the clinician wishes to administer agents orally to further decontaminate the gastrointestinal tract (see below). Because apo is a derivative of morphone, it can cause sedation, particularly if multiple doses are administered. Dogs should be monitored closely to ensure they maintain their gag and swallow reflexes. While apo is very effective in dogs it is not an effective emetic agent in cats because cats don’t have as many dopamine receptors. Alpha-2 agonists such as xylazine or dexmedetomidine are better choices, though cats are notoriously difficult patients in which to induce vomiting. Dosing for apo, xylazine, and dexmedetomidine can be found in veterinary formularies.

Hydrogen peroxide at a concentration of 3% is often recommended to induce emesis at home. While I prefer to have patients in the controlled environment of the animal hospital for emesis induction, if clients live far away, or the agent is particularly lethal, reducing absorption as much as possible before presentation to the animal hospital may be indicated. Hydrogen peroxide must be used with caution as its method of inducing vomiting is from direct irritation of the stomach lining. It is a caustic substance, with a pH of around 4 or 5 and aspiration leads to a vicious pneumonia and chemical pneumonitis. A study published in the Journal of Veterinary Emergency and Critical Care this year found “[s]ignificant visual and histopathologic gastric lesions” after administration of two doses of 3% hydrogen peroxide. No lesions were found in dogs who were administered apo in the conjunctival sac. It may be prudent for these patients to receive gastric protectants upon presentation to your hospital.

Agents such as salt or mustard powder or other substances are not recommended for emesis induction.

**Dilution:** Induction of emesis is contraindicated in the case of ingestion of caustic or corrosive agents that may cause damage to the physical structure of the GI tract or hydrocarbons that can be quite dangerous if aspirated. In case of ingestion of these types of toxicants, dilution with milk or GI-coating agents (e.g. milk of magnesia) is indicated, potentially followed by a cathartic agent. If a lethal amount of toxicant is ingested – or the toxicant is especially dangerous – gastric lavage may be employed to empty the stomach (see below).

**Adsorptive agents:** Activated charcoal can be administered in many toxicity cases to prevent absorption from the GI tract. The charcoal will adsorb certain toxic agents and facilitate excretion with the feces. Often a cathartic agent such as sorbitol is added to the charcoal to speed emptying of the GI tract (see below). Not all toxicants bind to activated charcoal so consultation with a trusted toxicology reference or an animal poison control center is warranted prior to administration.

**Cathartic agents:** Sorbitol, lactulose, magnesium salts, or bulk fiber can be used to speed transit of a toxicant through the intestinal tract. The increased speed decreases chances of absorption from both the large and small intestine. These agents are especially effective when combined with an adsorptive agent, such as activated charcoal.

**Gastric lavage:** Ingestions of hydrocarbons, caustic substances, bulky (e.g. bread dough), or very lethal toxicants may require gastric lavage. Gastric lavage requires general anesthesia and must always proceed with a cuffed endotracheal tube in place to protect the patient’s airway from both lavage fluid and stomach contents. Thorough lavage can achieve 80-90% emptying of the stomach contents.

**Surgical removal:** If the toxicant is solid (i.e. intact batteries, pennies), endoscopic or surgical removal is the most effective method to prevent toxicant absorption.

In cases where absorption has already occurred such as ingestion of xylitol, alcohol, or if presentation to the animal hospital is delayed, it may be possible and necessary to decontaminate the patient’s bloodstream. A
mainstay of treatment is dilution using IV fluids. IV fluid administration will help to dilute any toxicant absorbed, as well as increasing renal blood flow, which may increase excretion of toxin while also providing supportive care for the patient. There are agents available to practitioners to help clear toxins from the blood stream.

Intralipid Emulsion Therapy: Intralipid emulsion (ILE) is the fat portion of partial or total parenteral nutrition solutions. The use of ILE for intoxications began with the study of the treatment of local anesthetic overdoses in the 1990’s. Since then, ILE has been viewed as the “holy grail” of blood stream decontamination in cases of lipophilic toxicants. The more lipophilic the toxin, the more likely it is that ILE could be used as a treatment. Lipophilicity is described using a log P value. If the toxin’s log P value is > 1, it is considered lipophilic. The higher the log P value, the more lipophilic it is. Toxicants with high log P values are listed in Table 1. There are two theories as to how ILE exerts its anti-toxin effects. The first theory posits that providing the body with free fatty acids (FFAs) in the form of ILE floods the mitochondria and blocks the action of many of the toxicants that interfere with proper mitochondrial function and normal use of FFAs. The second theory is called the “lipid sink” theory and posits that providing a large amount of free fat in the bloodstream creates a biochemical compartment that can pull lipopholic substances out of free circulation and into the fat “sink”, inactivating the substance and encouraging its metabolism and excretion along with the ILE.

Dosing for ILE can be found via consultation with poison control centers, or in veterinary formularies. When using ILE, it is important to monitor the patient for side effects such at fat emboli, allergic reactions, and Fat Overload Syndrome (FOS). Many of the substances listed in Box 1 may have other, more conventional therapies, antidotes, or reversal agents available. In those cases, ILE should be used only if the patient is not responsive to conventional therapy.

Chelation therapy: In the case of metal ingestions (e.g. lead, iron supplements, zinc), it may be necessary to perform chelation therapy. Chelating agents cause precipitation of metals out of tissues for excretion by the kidneys. This therapy can be nephrotoxic so close monitoring is required.

Antidotes / Reversal Agents
In cases where an antidote or reversal agent is available it should be administered as early as possible, while also pursuing stabilization, decontamination, and supportive care efforts.

Nursing and Supportive Care
Regardless of the toxicant the patient is exposed to, supportive care is key to good patient outcomes. Most patients will require IV fluid therapy and, in some cases, intensive nursing care and monitoring.

<table>
<thead>
<tr>
<th>Amlodipine (hypotensive agent)</th>
<th>Baclofen (muscle relaxant)</th>
<th>Bupropion (local anesthetic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bupropion (antidepressant; Wellbutrin®)</td>
<td>Carbamazepine (anticonvulsant)</td>
<td>Carprofen (NSAID; Rimadyl®)</td>
</tr>
<tr>
<td>Chlorpheniramine (antihistamine; Chlor-Trimeton®)</td>
<td>Chlorpromazine (antipsychotic)</td>
<td>Clomipramine (TCA; Clomicalm®)</td>
</tr>
<tr>
<td>Cyclosporine (immunosuppressant; Atopica®)</td>
<td>Dexamethasone (glucocorticoid)</td>
<td>Diazepam* (sedative)</td>
</tr>
<tr>
<td>Digoxin* (cardiac glycoside)</td>
<td>Diltiazem (Ca+ channel blocker)</td>
<td>Indomethacin (NSAID)</td>
</tr>
<tr>
<td>Itraconazole (antifungal)</td>
<td>Ivermectin (antiparasitic; Heartgard®, Ivomec®)</td>
<td>Ketoprofen (NSAID)</td>
</tr>
<tr>
<td>Lidocaine (local anesthetic, antiarrhythmic)</td>
<td>Loratadine (antihistamine; Claritin®)</td>
<td>Metoprolol (Beta blocker)</td>
</tr>
<tr>
<td>Moxidectin (antiparasitic; Advantage Multi®)</td>
<td>Naproxen (NSAID; Aleve®)</td>
<td>Nicotine (stimulant)</td>
</tr>
<tr>
<td>Nifedipine (antianginal; Procardia®)</td>
<td>Promethazine (antihistamine)</td>
<td>Pyrethrin insecticides</td>
</tr>
<tr>
<td>Trazadone (SSRI)</td>
<td>Verapamil (Ca+ channel blocker)</td>
<td>Vinblastine (chemotherapeutic)</td>
</tr>
</tbody>
</table>

Table 1. A short list of substances with high log P values

*antidote / reversal agent available

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technicians who are familiar with toxicology concepts and treatment modalities are central to ensuring that patients recover with minimal long-lasting effects.

References available on request
Ace the Interview

Fetch 2019

Tosha Zimmerman CVT & Tasha McNerney CVT

Applying for a new position can be an intimidating process especially if it has been a while. From creating a resume to interviewing face to face, making a great impression is crucial to landing the job of your dreams. Selling your personality and skillset through a resume and phone interview to guaranteeing an invitation to interview in person can be terrifying but only if you are not prepared. Our goal in this presentation is to guide you through how to ace an interview with some do’s and don’ts as well as provide with you some great advice on how to find the right practice that is ideal for you.

Creating a resume that is professional is your first impression as an applicant. Think of it as a quick snapshot of your background to include your objective, experience, skillset and education so that a potential employer can easily see if you are qualified. It should be formatted in a way that is easy to read and not be too busy while also catching the eye of the hiring manager.

Resume Do’s

- Simple Format/Font
- Objective/Summary
- Core Skills that are relevant to the position
- Employment history relevant to the position in chronological order (present to past)
- Education
- Spell Check, proofread x 100
- One Page

Resume Don’ts

- Indeed Format
- Current contact information
- Too Creative (hard to read fonts, pictures, colors)
- Unprofessional email
- Exaggerating job descriptions – Don’t lie
- References Upon Request
- No abbreviations
- “I” Statements

Phone interviews are more common than ever these days and anyone applying for a new job should be prepared for one as if it was an in-person interview. This includes researching the practice, assembling answers ahead of time to common asked questions and being engaged. Phone interviews can be scary for some so practicing with a friend or family member would be a great way to prepare as well. It is very easy for the person conducting the interview to tell when someone or has no personality over the phone or is not interested in the conversation at all. Personality goes a very long way!
Phone Interview Do’s

- Be prepared – research employer, services, hours etc
- Take it seriously
- Sell your personality
- Smile when you speak
- Be professional
- Professional language
- Prepare for common questions such as strengths and weaknesses and what would you do if?
- Prepare your own questions
- Job listing in front of you to reference
- Quiet location

Phone Interview Don’ts

- “I love animals” is not a reason to apply
- Speak negatively of current or past employers
- Noisy Location – outside, subway platform, child crying, dogs barking
- Use of profanity
- Use casual slang
- Dull personality
- Put interviewer on hold or ask to call back
- Multi task
- Use speakerphone
- Mumble
- Dominate the conversation

The in person and working interview is your time to shine and show that your resume and personality were worth the hiring manager inviting you in. You should already know what is expected of you based off your phone interview so be prepared. It is also where an employer can spot if you really know how to calculate CRI’s or take the most perfect thoracic radiograph.

In Person/Working Interview Do’s

- Be 10-15 minutes early
- Dress appropriately – Business casual
- Bring clean scrubs
- Bring hard copy of resume with separate sheet for references
- Ask questions - the more the better, culture fit for you
- Make eye contact
- Show interest in patients and cases
- Anticipate written test (s)
- Be confident
• Introduce yourself

**In person/Working Interview Don’ts**

• Dress casually or inappropriate  
• Be late  
• Speak negatively of current or past employers  
• Have your phone on you  
• Wear scrubs from your current/past employer  
• Use of profanity  
• Body language – like you have somewhere else to be, not interested  
• Lie or oversell your skillset  
• Wear too much perfume/cologne  
• Smoke right before

Following up with the hiring manager within 24 hours after the interview thanking them for the opportunity to interview goes a long way and shows them you are serious about the position. This is also a great time to reiterate why you believe you are a good fit. You can always use some examples of what you observed during your interview and how it was of interest to you….etc….. Good luck!
How Low Can You Go- Managing Hypotension Under Anesthesia

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Fetch 2019

Many injectable and inhalant anesthetic drugs cause some degree of hypotension. This can be caused by many factors including a direct effect on heart rate, afterload, preload, contractility, and/or systemic vascular resistance. Hypotension is one of the most common intra-operative anesthetic complications seen in small animal anesthesia. Hypotension can lead to decreased perfusion to vital organs such as the liver, kidneys, and the heart. This decrease in perfusion leads to a reduction in oxygen delivery. The anesthesia technician should have an understanding of factors that lead to hypotension, how to measure blood pressure, and how to properly address hypotension under anesthesia.

Palpating pulses is important in your anesthetized patient. However, simply palpating a pulse is not a string indicator of adequate blood pressure. When you are feeling pulse pressure you are thought to be “feeling” the difference between systole and diastole. However, when anesthetic agents are on board, especially agents that cause vasoconstriction, you may feel a decrease in pulse pressure despite normal blood pressure readings. Therefore, simply feeling a pulse should not be used as a barometer for your patients' blood pressure.

There are 3 ways you can measure blood pressure while under anesthesia:

**Direct**- The placement of an arterial catheter and continuous readings via a pressure transducer.

**Doppler**- The use of a Doppler ultrasound crystal and a sphygmomanometer.

**Oscillometric**- The use of a machine that automatically inflates and deflates a cuff to determine blood pressure continuously.

Remember placement of the cuff is important, and the most consistent cuff location for small patients is the mid-foreleg For the purpose of this article we will use the mean arterial pressures less than 60mmHg to determine hypotension. If using a Doppler method we will use any readings below 100mmHg systolic to describe hypotension. Once a hypotensive trend has been identified, the next step is to work with your clinician to determine a course of action for correction.

There are several different ways to treat hypotension once you have identified the underlying cause. If your patient is bradycardic and the hypotension may be caused by a decrease in heart rate you can consider an intra-operative dose of glycopyrrolate at 0.01mg/kg IV until the heart rate returns to an acceptable range. It is also important to note what the patients’ pre-operative heart rate was. This way you know what is considered “normal” for this patient. However, if your patient is bradycardic and hypotensive due to administration of an alpha-two agonist such as dexmedetomidine, consider administering a reversal drug such as atipamezole.

If your patient is hypotensive due to the vasodilating effects of anesthetic inhalant agents, consider decreasing the dose of these agents. Also, inhalant anesthetic agents can cause a decrease in cardiac output and when combined with vasodilation, lead to decreased organ
Increased circulating volume can be achieved by administering boluses (5-10mL/kg) of intravenous crystalloid fluids. This will help if the hypotension is secondary to hypovolemia and peripheral vasoconstriction. In some cases, reducing anesthetic depth and administering increased volume of crystalloid fluids is not enough to correct hypotension. In these cases, the addition of colloids and/or inotropic agents should be considered. Colloidal solutions can be either natural (e.g. albumin) or synthetic (e.g. dextran, hydroxyethyl starch). Colloids may be more effective than crystalloids for correcting intravascular volume deficits. Patients that do not respond to these treatments will need intervention with drugs that can improve systemic vascular resistance and contractility. These drugs act on alpha and beta receptors. Stimulation of these receptors can have varying effects at varying doses. Before starting any of these therapies talk with your clinician to verify drug and dosages. Dopamine- a short acting norepinephrine precursor that has alpha and beta agonist properties. Dosing is important with this drug as different dosing can stimulate different receptors and cause different effects. Medium doses such as 3-5 mcg/kg/min will cause stimulation of beta adrenergic receptors which acts as a positive inotrope and increases heart rate. Higher doses from 6-10mcg/kg/min stimulate alpha adrenergic receptors which cause an increase in systemic vascular resistance. Doses higher than 10mcg/kg/min can cause tachycardia, increased afterload which leads to a decrease in cardiac output. (Mazzaferro, 2001) Dobutamine- a synthetic catecholamine that stimulates cardiac contractility, cardiac output, and coronary blood flow. Low doses cause an increase in cardiac output while causing a moderate increase in heart rate. The therapeutic dose ranges are from 2-20mcg/kg/min. (Mazzaferro, 2001) Ephedrine- a noncatecholamine sympathomimetic drug that stimulates alpha and beta adrenergic receptors. Another benefit of ephedrine is improved oxygen delivery to tissues via increased hemoglobin. (Wagner, 1993) Ephedrine can also be administered as a single bolus injection of 0.10mg/kg IV. Norepinephrine- acts at alpha and beta receptors. It increases MAP and peripheral vascular resistance. Coronary blood flow is also increased. Norepinephrine may be beneficial in increasing MAP in septic patients or patients currently on beta blocking drugs such as atenolol. (Mazzaferro, 2001)

During anesthesia it is important for the anesthesia technician to monitor trends in blood pressure and work with the clinician to develop a protocol to appropriately treat hypotension.

References:  Mazzaferro, E. and Wagner, A. Hypotension during Anesthesia in Dogs and Cats: Recognition, Causes, and Treatment. Compendium Vol. 23, No. 8 August 2001
The measurement of end tidal CO2 (ETCO2) is currently the optimal method of non-invasively and continuously monitoring the adequacy of ventilation and circulation in veterinary patients. The ETCO2 monitor measures expired carbon dioxide. This measurement can be used to evaluate adequacy of ventilation, metabolic status and circulatory status. (Moens, 2010) This is different than the pulse oximeter which is used to measure hemoglobin and saturation with oxygen.

Capnography directly reflects the elimination of CO2 by the lungs to the anesthesia device. Indirectly, it reflects the production of CO2 by tissues and the circulatory transport of CO2 to the lungs. For example, an increased metabolism will increase the production of carbon dioxide increasing the ETCO2. A decrease in cardiac output will lower the delivery of carbon dioxide to the lungs decreasing the ETCO2 measurement (Canning, 2007)

Under normal circumstances, ETCO2 reflects the partial pressure of CO2 in the alveoli of the lungs. It is important to note that ETCO2 is not the same as PaCO2, which measures the partial pressure of CO2 in the blood. However ETCO2 can give you a close approximation of PaCO2 as the difference in these numbers in a normal, healthy patient is between 2-6mmHg. (Digicare, 2009)

Normal ETCO2 values for canines and felines ranges from 35-45mmHg. An increase in ETCO2 can indicate many things including hypoventilation, increased anesthetic depth, exhausted soda lyme, airway obstruction, and hyperthermia. Increased ETCO2 can also be seen during laparoscopy due to absorption of CO2 (that is used to inflate the abdomen) from the peritoneum. A decrease in ETCO2 can be indicative of hyperventilation, an inadequate plane of anesthesia, hypothermia, decreased cardiac output. Absent ETCO2 readings can indicate respiratory arrest, cardiac arrest, or technical problems such as an obstructed or dislodged endotracheal tube.

The capnograph can be extremely helpful to the veterinary technician that is not well experienced with intubation. The capnograph can be used to show proper endotracheal intubation. (Dorsch, 2008) If patients have an endotracheal tube in the esophagus, the ETCO2 reading will be at zero, sine little to no CO2 is produced in the esophagus and stomach. Patients that are properly intubated with the endotracheal tube in the correct position will display and nice plateau on the capnograph.

The capnograph can also be a valuable tool to assess the adequacy of chest compressions during CPCR. The quality of CPR can be monitored using end tidal CO2 levels during compressions. Studies have shown that the greatest chance for the return of spontaneous circulation happens when CO2 levels are maintained above 20mmHg during cardiac arrest.
CO2 levels below 20 mmHg may be an indicator of inadequate chest compression depth and rate.

End tidal CO2 monitoring can provide an early warning sign of shock. A patient with a sudden drop in cardiac output will show a drop in ETCO2 numbers that may be regardless of any change in breathing. This has implications for trauma patients, cardiac patients – any patient at risk for shock.

In 2000 a study was done examining the relation of ETCO2 cardiac output. 5 pigs had hemorrhagic shock induced by bleeding, 5 pigs had septic shock induced by infusion of e-coli, and 6 pigs had cardiogenic shock induced by repeated episodes of v-fib. The pigs’ cardiac output was continuously measured as well as their ETCO2. The study showed that CO and ETCO2 were highly related in diverse experimental models of circulatory shock in which cardiac output was reduced by >40% of baseline values… measurement of ETCO2 is a noninvasive alternative for continuous assessment of cardiac output during low flow circulatory shock states of diverse causes. (Xiahua, 2000)

A capnometer only gives a numerical display of ETCO2. It will not give you a waveform, therefore it is hard to assess ET tube placement, cuff seal, and associated problems you can otherwise derive from the waveform analysis.

A capnograph should have four basic parts.

1- Carbon dioxide is cleared from the anatomic dead space, also known as return to baseline.
2- Expiration of dead space and alveolar carbon dioxide.
3- The plateau, this is the highest point and gives you’re your ETCO2 measurement.
4- Return to baseline, as the patient begins to inhale fresh gas.

If your baseline does not return to zero, this can signal the anesthesia technician to problems such as excessive mechanical dead space, exhausted soda lyme, inadequate fresh gas flows.
on a non-rebreather and possibly a faulty one way valve. In the case of dead space, as dead space volume increases, effective alveolar ventilation decreases. Mechanical or equipment dead space is made up of any portion of the endotracheal tube that extends beyond the patients incisors as well as patient monitor adapters (such as ETCO2 sampling line connectors, and mainstream sampling connectors).

A capnograph can be an invaluable tool for the patient under general anesthesia. A capnograph can alert the veterinary technician to possible problems with either the anesthetic equipment or the patient themselves before it is too late.

For more information on capnography, consider visiting www.capnography.com.

References:


Xiahua, (2000) End-tidal carbon dioxide as a noninvasive indicator of cardiac index during circulatory shock, Critical Care Medicine, Vol 28, No 7
The pressures facing anesthetists today are great; the anesthetist must fully understand physiology, pathophysiology, pharmacology, anesthesia equipment, and monitoring devices as well as recognize their limitations. Although the goal is to assure a successful surgical or procedural outcome while ensuring the patient receives the finest possible anesthetic care, mistakes can and do happen.

Patient Considerations

Careful pre-anesthetic assessments are essential to identify physiological, pathological or drug-related factors that may complicate a patient's anesthetic management. The value of obtaining an in depth and accurate history cannot be overemphasized. In addition to the presenting complaint, essential components of a thorough history include the patient's name, species and breed, age, sex (altered vs. intact) and breeding/estrus status, current diet and housing conditions (indoors versus outdoors), preventative health status (e.g., date of last vaccine, fecal exam, heartworm or feline leukemia test), as well as the patient's prior medical history, current medications, and prior adverse reactions to anesthetic agents or drugs. Additionally, preoperative bloodwork consisting of a complete blood count and age-appropriate chemistries should also be performed.

A good physical exam is also an imperative element of the pre-anesthetic assessment. In addition to documenting the patient's overall demeanor, hydration status, weight and body condition (e.g., obese, emaciated), the patient's temperature, pulse, respiratory rate, mentation, mucous membrane color and capillary refill time should be obtained. Assessment of the reproductive, cardiovascular and respiratory systems, evaluation of the skin, oral cavity and lymph nodes as well as abdominal palpation should also be performed. Again, all abnormalities should be further investigated.

Potential patient-related areas of concern:

- Undiagnosed underlying disease
  - (e.g., renal or cardiac disease; feline hyperthyroidism; diabetes)
- Mismanaged pre-existing conditions
  - (e.g., overhydrating patient with unstable cardiac disease; renal disease patient not provided with adequate fluid therapy perioperatively)
- Inadequate preoperative stabilization of patient's condition or disease
  - (e.g., shocky patient still dehydrated at time of surgery; persistent arrhythmias ignored resulting in negative impact on blood pressure; pneumothorax patient anesthetized before condition resolved)
- Significant clinical pathology abnormalities that have been ignored, untreated, or undiagnosed
  - (e.g., hyperkalemia; azotemia; hypercalcemia)
- Emergent situation- Not enough time to assess organs and systems
- Fractious/feral patient (and subsequent limitations)
- Other patient-related human errors
  - (e.g., incorrect weight recorded- lbs vs kg; using obese vs lean body weight; different patient medicated; inadequate history; missed abnormalities on physical exam)

Pharmacology Considerations

Many anesthetic and analgesic drug companies provide ranges rather than specific doses because each patient's anesthetic requirements can vary. Ideal drug choices and dosing should be selected based on each patient's history, temperament and physical health status as well as the procedure being performed. Additionally, beware that medications prescribed for certain medical conditions can interact with those used during the anesthetic episode.

Potential drug-related sources of errors:
• Drug overdose
• Drug underdose
• Incorrect drug calculations
• Used wrong/different drug than intended
• Adverse drug interaction with current medications
• Assuming drugs used for anesthesia and analgesia are interchangeable
• Blood transfusion reactions under anesthesia
• Human errors
  o Charting errors
  o Allergies, current medications not properly documented

Before administering ANY drug to EVERY patient, please consider the following:
The 6 ‘Rights’ of Patient Medication Administration. Do you have the Right___?:
1. Patient
2. Drug
3. Dose
4. Route
5. Time/frequency
6. Documentation

Anesthesia and Monitoring Equipment Considerations

Intra-operative monitoring documentation is imperative for optimizing all anesthetic procedures. In addition to allowing informed, flexible and well-timed responses to changes in the patient’s status, it can also serve as a database for comparison prior to subsequent anesthetic episodes. A variety of equipment is available to monitor the patient’s physiologic parameters, including but not limited to stethoscopes, blood pressure monitors (using either indirect or direct methods), electrocardiograph (ECG) tracings, pulse oximeters, end-tidal carbon dioxide monitors, and temperature probes. Each monitoring device certainly has its own distinct set of advantages and disadvantages, and every anesthetist should be familiar with them.

Several studies have indicated that between 60-93% of anesthetic complications can be detected by an electronic monitor before a trained anesthetist; therefore, alarms should never be ignored (or silenced as being ‘annoying’.) The data assimilated from anesthetic monitors can provide invaluable information on the anesthetized patient’s status, but only if used prudently.

It is also important to use a ‘hands-on’ approach during every anesthetic procedure. Technicians can gather a lot of information from the anesthetized patient simply by consistently palpating pulses, assessing mucous membrane color and capillary refill times, feeling jaw tone, and tracking palpebral and withdrawal reflexes. Regularly evaluating these parameters can also help to alert the anesthetist of an impending problem before some monitors will sound an alarm. Any concerns about a patient’s status during the anesthetic period should always be brought to the veterinarian’s attention.

Additionally all anesthetic equipment should be kept well maintained and serviced at regular intervals. Any malfunctions should be promptly addressed or repaired. A service record should be kept on each anesthetic machine.

Potential errors posed by monitoring and anesthetic equipment issues:
• Data provided by monitors not accurate
  o (e.g., incorrect blood pressure cuff size selected, ECG leads double-counting)
• Incorrect monitoring devices used for patient’s condition or procedure
• Patient’s abnormality not accurately reflected by monitor
  o (e.g., arrhythmias not detected by pulse oximeter)
• Relying too heavily on monitors
• Accurate data provided by monitor but ignored/alarms silenced
• Unidentified anesthesia machine malfunction
  o System leak ‘somewhere’…
  o Vaporizer out of calibration
• Improper flow rates (nitrous oxide/oxygen)
• Human errors
  o Incorrect use of anesthetic machine or equipment
    ▪ (e.g., closed pop-off valve resulting in barotraumas; training deficiencies; brain ‘fart’)
  o Breathing and ventilation concerns
    ▪ Improperly intubated
      ▪ (e.g., cuff over- or under-inflated; endotracheal tube length wrong [too long or too short])
    ▪ Premature tracheal extubation complications
      ▪ (e.g., hypoventilation; apnea; obstructive breathing)
    ▪ Immediate postoperative hypoventilation
      ▪ (e.g., due to opioid effects; incomplete reversal of neuromuscular blockade; presence of redundant oral tissues [brachycephalic airway syndrome])
  o Substandard patient monitoring
    ▪ Unrecognized patient deterioration
    ▪ ‘Reactive’ vs ‘proactive’ approach
    ▪ Patient’s deterioration left untreated/ignored
    ▪ Anesthetist over-worked or stretched too thin (e.g., dual role as circulating nurse) to properly monitor patient
    ▪ Inattentive / distracted anesthetist
      ▪ (e.g., chatting on phone, checking electronic devices, overly engaged in casual conversation)

It is also imperative to remember that diligent care of the anesthetized patient should not end once the procedure is finished: even though it is at this point where many anesthetists tend to let their guard down. The magnitude of this problem was identified in the 2008 article entitled, The Risk of Death: the Confidential Enquiry into Small Animal Perioperative Fatalities, published in Veterinary Anaesthetics and Analgesia (Brodbelt, et.al.)

“The postoperative period was the most common time for dogs, cats and rabbits to die usually within 3 hours of surgery…greater patient monitoring and management during this time period is recommended.”

As it turns out, patients in this stage of recovery can be subject to life threatening problems such as hypothermia, hemorrhage, hypoxia, hypotension, post-anesthetic vomiting, emergence delirium, cardiac arrhythmias, or cardiopulmonary arrest. Furthermore, these patients will need to be regularly assessed for pain. Only experienced personnel should continuously monitor these patients in a designated recovery area. The recovery area should be stocked with necessary drugs and equipment to treat common post-anesthetic emergencies.

The Anesthetist “Too (sic) Err is Human”

As you have probably noticed by now, the decisions of the anesthetist plays a pivotal role in assuring patient safety in every one of the aforementioned categories. Therefore, even with the availability of the best training and equipment, many exterior factors can impact the outcome of each anesthetic episode. Veterinary technicians are often faced with low wages, working long hours (and sometimes while short-staffed, and without a break!) and compassion fatigue. These factors, combined with things like poor communication and the stress of day-to-day life (such as an impending divorce, childbirth, etc!), can increase the potential for errors in all aspects of a patient’s anesthesia care. Fortunately, with proper
advanced patient preparation, expert training and experience, and the availability of adequately functioning monitoring devices and anesthetic equipment, most every anesthetic mishap can be averted.

“There are no safe anesthetic agents; there are no safe anesthetic procedures; there are only safe anesthetists.”

Robert Smith, MD

References
Sigrist N, Cats are not Dogs—Not Even in the ER: In proceedings 14th International Veterinary and Emergency Critical Care Symposium, 2008; Phoenix, pp175-177
http://www.vtarc.com April 2011
Tefend M: Hemodynamic Monitoring in the Postoperative Patient, In Proceedings for American College Veterinary Surgeons, 2003; Washington DC (no page numbers indicated.)

Recommended Reading:
National Association of Veterinary Technician in America (NAVTA) Journal; Anesthetic Monitors – Understanding Their Use and Limitations, Spring 2008
The care and maintenance of surgical instruments and equipment generally falls under one of the many job duties of a typical veterinary technician. It is hard to ignore all of the media reports about MRSA and other resistant infections that abound in human medicine, which also served as a way to raise awareness of healthcare-associated (nosocomial) infections (HAI) in veterinary medicine. Today’s veterinary technicians are uniquely poised to make a difference in the lives of veterinary patients, in part by ensuring that proper protocols and procedures are in place to help prevent perioperative infections. Iatrogenic surgical site infections (SSIs) can prolong recovery, increase patient morbidity and mortality and incur unnecessary costs for the client. However, it is important to note that any successful infection control program must consist of a multi-pronged approach, which may incorporate areas such as perioperative antibiotic use, choice of antiseptics and disinfectants, atraumatic clipping, best practices for prepping and draping, and proper housekeeping methods in addition to sterile processing.

Adequate sterile processing depends on the performance of people, processes and equipment to achieve the highest level of sterility assurance all the time. Therefore, the goals for individuals participating in a sterile processing program include: 1.) Assuring every item in each load is sterile 2.) Minimizing the risk of HAIs 3.) Meeting regulatory requirements using ‘best practices’ 4.) Maintaining integrity 5.) Assuring quality outcomes.

Inconsistent processes and/or equipment malfunction can jeopardize the goals of sterile processing. Susan Flynn, BESc, CSPDT (Central Sterile Processing and Distribution Technician), explains that although utility (e.g., due to poor steam quality) and equipment problems (e.g., due to low temperatures or inadequate air removal) may negatively impact the outcome of steam sterilization methods, human error accounts for ~85% of processing related issues. These errors may be attributed to selection of the incorrect instrument packaging materials, incorrect loading of the autoclave, selection of the incorrect biologic indicator (BI) or process challenge device, or due to selection of the incorrect cycle parameters for the load. Therefore it is imperative that each facility strives for continuous quality improvement and implements a system that continually seeks methods for improvement while eliminating or minimizing waste.

Instrument Decontamination and Cleaning

Blood, pus and other secretions may contain chloride ions which, when left to dry on surgical instruments, may cause staining (leading to rust), pitting and corrosion. Instrument decontamination and cleaning should be performed as soon as possible after use to prevent the formation of a thin film of proteinaceous material that may cause staining (leading to rust), pitting and corrosion. To prevent this, instruments should be rinsed under running water within 10 minutes after use. If it is not possible to clean the instruments immediately after use it is advised to keep them damp until appropriate cleaning can be performed.

Gross Debris Removal- Wash instruments within 20 minutes after surgery in a neutral pH soap (7-8) designed specifically for surgical instruments. High pH (> 8) cleaners, dish soap, iodine, bleach, cold-soak solutions, chlorhexidine-based solutions, laundry soap and hand scrubs may cause spotting and corrosion and therefore are not recommended. Since soil cannot be sterilized, instruments should be cleaned of all visible debris by hand washing prior to placement in the ultrasonic cleaner. It is important to use an instrument cleaning brush to clean all organic material from the jaw serrations, teeth and hinged areas.

Ultrasonic Cleaning- The use of ultrasonic cleaners is growing in popularity, predominantly due to their ease of use, efficiency and effectiveness. Placing instruments in an ultrasonic cleaner containing a neutral pH solution for a 3-minute cycle removed 99.9% of residual blood contamination.

Ultrasonic cleaning is created by high frequency sound waves produced by a generator located within the unit. Millions of tiny bubbles form cavities during the low pressure stage resulting in a process called cavitation. These bubbles collapse or implode during the high pressure stage and release enormous amounts of energy.

Always use a well-mixed, neutral pH ultrasonic cleaning solution to avoid increased surface tension and optimize the ultrasonic cavitation process. Freshly made ultrasonic cleaning solutions should be ‘degassed’ to ensure all air bubbles are removed by operating the ultrasonic cleaner for 10 minutes immediately after each new batch of cleaning solution is made and prior to introducing instruments for cleaning. Place instruments into the ultrasonic cleaner with ratchets and box locks fully opened and exposed to maximize cleaning. Do not overload the ultrasonic cleaner. Try to avoid mixing instruments.
of varying metal content in the same cycle to prevent cross-plating, which results in stubborn bluish-black surface stains. Instruments should be thoroughly rinsed upon exiting the ultrasonic cleaner. Moreover, it may be advisable to rinse the instruments in distilled water since tap water can contain high concentrations of minerals that contribute to staining. Nonetheless, prolonged immersion of surgical instruments in any solution can be damaging; never immerse surgical instruments for more than 20 minutes. Ultrasonic cleaning solutions should be changed daily, or sooner if the solution appears cloudy or dirty.

**Lubrication** - The lubrication process should be performed after surgical instruments have been thoroughly cleaned and dried. Proper lubrication of surgical instruments prevents rubbing and scraping and helps ensure that dulling and staining are minimized. All surgical instruments containing moving parts such as joints, box locks, ratchet and screws should be lubricated prior to autoclaving. Only lubricants approved for steam sterilization should be used. The use of lubricant sprays may be preferred over lubricant baths due to the fact that bacteria from previously dipped instruments may linger in the bathing solutions. Furthermore, lubricant sprays are associated with decreased costs and spray bottles consume less counter space than most typical instrument bath solution containers.

**Steam Sterilization**

Most veterinary practices employ steam sterilization—using gravity displacement autoclaves—as the predominant sterilization method. All instruments should be thoroughly cleaned, lubricated and properly packaged prior to steam sterilization. Autoclaving instruments with the ratchets open helps prevent box locks from cracking and assures appropriate steam penetration. Important tips to remember prior to steam sterilization include:

1. Always use distilled water to fill the sterilizer reservoir. Tap water contains minerals that can cause staining when left standing or to dry on the instrument.
2. Clean the steam line filter (if present) regularly, as per the manufacturer’s guidelines.
3. Clean the autoclave chamber weekly. Weekly cleaning prevents scale build-up, thereby allowing the sterilizer to function optimally.

**Pack Assembly and Sterile Processing**

All packaging materials should be equilibrated for at least 2 hours at room temperature (68° to 73.4° F or 20° to 23 °C) and at a relative humidity of 30- to 60% prior to use. It is imperative to ensure that all items to be wrapped for sterilization be dry and inspected for cleanliness and damage. Instruments with multiple parts should be disassembled prior to sterilization.

**Use of Peel Pouches** - Peel pouch wraps should allow adequate penetration and removal of both the sterilants and air, resist tearing and prevent contamination the contents. They should also be easy to seal, easy to open beyond the chevron seal without tearing and able to withstand the conditions of the sterilization process. When labeling peel pouches, use an indelible (permanent) marker, and write only on the film (transparent) part of the pouch. Never write on the paper or breathable side. Items packaged in pouches too small (or too large) may allow shifting during processing and rupturing of the pouch.

When sterilizing items in pouches, ensure that the item is inserted so that the handle or ‘grasping end’ is inserted first. Remove as much air as possible and ensure proper sealing conditions are met (e.g., prevent wrinkles and air bubbles.) When heat sealing tubing always leave about 1.5” to 2” beyond the seal to assure the item can be easily grasped after the sterilization process.

Sharp or delicate instruments should have a protector tip applied over the ends to prevent damage and provide cushioning during the sterilization process. Protector tips may be either vented (fenestrated) or non-vented.

Never fold a pouch either before or after sterilization as it can interfere with the sterilization process or compromise the material. To prevent ‘blow out’, or a ruptured pouch due to items being packaged too tightly, allow at least 1 inch of space between the item to be sterilized and the pouch seals on all 4 sides. Use cut-to-size sterilization tubing for pouching long items. Items that can rupture the pouch to its size, weight or shape should be packaged using another method.

Double pouching may be considered when multiple small items must be sterilized or to facilitate aseptic presentation to the sterile field. To assure adequate sterilant penetration, double pouched items should be wrapped so the paper side of the inner pouch is on the same side as the paper side of the outer pouch. Protector tips used to cushion or secure instruments contained in double peel pouch wraps must be placed in such a fashion to assure that contact with the sterilant is not impeded. Avoid the use of
rubber bands and latex tubing to secure items together. Internal chemical process indicators should be placed inside of the innermost pouch prior to sealing.  

Use of Drape Materials- Good central sterilization wraps should be engineered to allow sterilants to pass through readily while blocking bacteria. Although no stringent FDA standard exists for class II sterilization wraps, manufacturers have agreed that each type of packaging material developed must demonstrate appropriate scientific evidence that the 1.) Material is specifically designed and suitable for the recommended sterilization methods and cycles, 2.) Material provides an effective barrier to contamination when used according to the manufacturer’s written instructions, and 3.) Manufacturer provides adequate in-service education and instructions for use/reuse. 

Loading the Autoclave- Every item to be sterilized should be loosely packed in the autoclave. Moreover, ensure that all contained items do not come into contact with either the inside walls or top of the chamber. Peel pouches should be loosely placed paper-to-plastic (resulting in all pouches facing the same direction or ‘in line’), so that the sterilant can easily reach all surfaces. Never fold pouches since folding can create areas of trapped air and prevent proper penetration of the sterilant. Furthermore, do not stack pouches on top of one another during sterilization. Using a spiral metal letter holder as a standing aid permits proper steam flow. 

Surgical packs wrapped in drape materials should be placed in the autoclave so that steam can freely circulate around all items in the sterilizer. Pans or trays utilized for sterilizing surgical packs should ideally be fenestrated to enable better steam sterilization and more effective drying. For most efficient sterilizing, place heavier instruments on the bottom shelving and lighter, more delicate instruments on upper shelving.

It is also important to cool items at the end of the sterilization cycle and prior to handling to avoid compromising the barrier properties of the packaging materials and contaminating the contents. Furthermore, it is advisable to avoid placing warm or hot processed items on cool or cold surfaces since the formation of condensation may cause breaches in the packaging materials.

Sterilization Process Monitoring 

Proof that adequate sterilization has been achieved is not easy to verify with the naked eye. The rationale behind utilizing monitoring devices to assure adequate sterilization is as follows: 1.) Ensure the probability of the absence of all living organisms on medical devices being processed 2.) Detect failures as soon as possible 3.) Verify failures as soon as possible 4.) Remove medical devices involved in failures before used in the patient 5.) Improve patient outcomes and safety 6.) Control costs.

Physical monitors- Physical monitors (previously referred to as mechanical monitors) can verify that the parameters of a sterilization cycle were met to within the guidelines established by the manufacturer. These physical monitors may include unit-specific devices such as recording charts, printouts, gauges or digital displays. However, most physical monitors only record the conditions of one location in the sterilizer and are not capable of assessing other physical conditions that may impact the sterility of the load such as whether or not proper packaging and loading protocols were achieved.

Chemical indicators- Chemical indicators (CI) are used to monitor the presence or attainment of one or more parameters required for a successful sterilization process, or used during specific tests of sterilization equipment. Chemical indicators may be used either externally or internally. Chemical indicators designed for external use are predominantly used to differentiate between processed and unprocessed units. Internal CIs should be placed inside the pack in an area considered most difficult for the sterilant to penetrate. While internal indicators do not guarantee sterility, they do represent an indication of whether or not sterilization conditions were met within a package. 

There are 5 classes of chemical indicators that are used to assess parameters identified as being essential or ‘critical’ to the sterilization process. For example, the variables considered critical for effective steam sterilization may include parameters such as time, temperature and water (as delivered by saturated steam), but those considered critical for effective EO sterilization may consist of time, temperature, relative humidity and EO concentration. 

Class 1 indicators (process indicators): They are used externally as an exposure control (e.g. indicator tapes) and are used with individual units to distinguish between processed and unprocessed items. Class 1 indicators are relatively simplistic and designed to react to one or more of the critical process variables. 

Class 2 indicators (test sterilizer performance during a specific test procedure such as a Bowie-Dick test): Bowie-Dick testing can detect anomalies such as air leaks, inadequate air removal,
inadequate steam penetration or the presence of non-condensable gases (air or gases from boiler additives) in vacuum-assisted sterilizers.

**Class 3 indicators** (single-variable indicators): Designed to react to one of the critical variables and indicate exposure to a sterilization process at a stated value (SV) of the chosen variable. For example, the SV of 250°F/121°C must be > 16.5 minutes.

**Class 4 indicators** (multi-variable indicators, usually paper strips): Designed to react to 2 or more of the critical variables. This type of CI indicates exposure to a sterilization cycle at a SV of the chosen variables.

**Class 5 indicators** (integrating indicators): Designed to react to all critical variables. The SV for Class 5 indicators are equivalent to the performance requirements for biologic indicators (ISO 11138 series: 2006). Their response must correlate to a BI at three time/temperature relationships: 250°F/121°C, 275°F/135°C, and at one or more temperatures in between, such as 263°F/128°C. Stated values must be listed on the product or provided on the label/instructions for use.

**Biological indicators**: Biological indicators contain > 100,000 viable spores of a highly resistant organism (e.g., Geobacillus stearothermophilus, formerly named Bacillus stearothermophilus) on a strip, and therefore are considered the most reliable level of testing available. Using BIs during a sterilization cycle provides the only direct method of demonstrating lethality within that particular load. Nonetheless it is possible to have a negative BI and still have a CI failure elsewhere in the load. Reasons for failure can be due to a number of things, based on the sterilization method.

**Huck Towel, Gown and Cloth Drape Care**

If reusing towels and drapes is necessary, attempt to use as little laundry detergent to launder them as possible. Laundered towels and drape materials can retain soap particles and deposit them on the surface of the instruments during the sterilization process. It may be prudent to run all laundered surgical textiles through an extra rinse cycle to remove excess soap particles. For reasons indicated above, the use of bleach is often precluded while laundering surgical cloth drapes, gowns and huck towels. Bleach can also damage fabric threads and as such will decrease the usable life span of fabrics over time.

Some gown manufacturers provide a *usage grid* near the lower hem of the gown. The grid is designed so that one box is marked each time the gown is used. Once all of the grid boxes on the gown have been filled in, the gown should be removed from service.

The final step to assuring a successful sterilization process monitoring system is to maintain a record keeping system that can be used to document all items processed as well as evidence of their monitoring devices. Once the appropriate methods and techniques are employed to assure quality sterilization, proper storage and use protocols must also be followed to guarantee ‘the probability of sterilization’ at multiple levels.

HYPOTHERMIA—WHAT’S THE HYPE?
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Thermoregulation Physiology

Animals attempt to tightly regulate their core body temperature to within a very narrow, species-specific range called a ‘set-point’. Thermoregulation occurs when the body’s thermal receptors detect temperature changes in the peripheral zone (e.g., skin, extremities and subcutaneous tissues) and core zone regions (e.g., brain, heart and great vessels). These thermal variations are then transmitted from the thermal receptors to the posterior hypothalamus via the spinal cord. Since heat travels from high to low temperatures, thermal homeostasis (or maintaining the set-point) is achieved by modulating the distribution and flow of blood to organs, viscera and skin by way of either vasoconstriction or vasodilation. Initial heat loss induces cutaneous vasoconstriction in an attempt to limit heat loss and maintain core temperature. When vasoconstrictive efforts fail to resolve hypothermia, heat production is augmented via increasing muscle tone and shivering. Furthermore, non-shivering thermogenesis occurs with epinephrine secretions.

Hypothermia may be the result of heat loss, decreased heat production, or thermoregulation alterations due varying clinical problems, and can occur as a primary (accidental), secondary (e.g., due to sepsis, trauma, or disease) or therapeutic (induced) condition. Hypothermia is most commonly associated with anesthetized patients as well as emergent shocky or hemorrhaging patients. Other factors that can influence thermoregulation include age (e.g., pediatric or geriatric), body condition, hormones, or circadian rhythm.

Factors Influencing Thermoregulation

The body is protected from heat loss by 3 tissue layers: skin, subcutaneous fat, and hair. Additionally, the thickness of these 3 layers can vary from patient to patient. (e.g., they are the least thick in most pediatric patients.) Heat loss occurs from the insulating layers to the environment in 2 stages. Stage 1 occurs when heat is transferred from the core to the skin. Stage 2 occurs when heat is ultimately lost to the environment by conduction, convection, radiation, and evaporation, and by the excretion of urine and feces. Conduction is the process that occurs when heat is transferred away from an animal’s body as a result of direct contact with a cooler object or surface such as a stainless steel table or electrocautery plate. Convection may be referred to as the ‘wind chill’ effect, relying on circulating air or liquid to carry heat away from or towards an object. Convection may be influenced by the ambient room temperature, air conditioning or furnace settings. Radiation occurs in a similar fashion, but instead relying on passive dissipation of thermal energy through air or space and into the environment. Seventy-percent of the body’s dissipation of heat can be attributed to radiation and convection. Evaporation may be due to heat and moisture loss from the skin and respiratory tract, open body cavities, or after the use of surgical prep solutions (e.g., alcohol or scrub solutions.)

Hypothermia’s Adverse Effects

Almost every major organ and system is adversely affected by hypothermia. Hypothermia can cause decreased cardiac output, resulting in an increased risk of arrhythmias and hypoxia, and leading to reduced tissue perfusion. Hypothermia-induced bradycardia is typically non-responsive to anticholinergics. Hypothermia also diminishes the effect of positive inotropes on blood pressure, heart rate and cardiac output. Delayed drug metabolism, hypomotility, and decreased hepatic metabolism result in a prolonged recovery and potential drug toxicity. Clotting times can be prolonged due to impaired platelet function and hemoconcentration with sludging. A suppressed immune function can lead to increased infection rates and delayed wound healing.

Even moderate hypothermia (<95°F), (e.g., due to trauma, shock, or as observed in the postoperative period) is associated with a significant stress response. There is a 2- to 7-fold increase in the release of catecholamines associated with hypothermia, resulting in vasoconstriction and hypertension, and causing bradycardic patients to become tachycardic. Severe hypothermia (<86°F) results in an increased risk of atrial fibrillation. Profound hypothermia (75.2°F- to 82.4°F) can result in refractory ventricular fibrillation and death. Subsequently, the net effect of hypothermia results in an increased incidence of morbidity and mortality.

Hypothermia is not only one of the most common anesthetic complications, but also the easiest to document without special equipment. Tympanic membrane temperatures are the most accurate due to the shared blood supply by the middle ear and hypothalamus. However, ear thermometers are relatively expensive and can be technically challenging to use properly.
Almost all anesthetized or sedated patients will lose body heat under general anesthesia, with the exception of adult Nordic breeds (e.g., Samoyed, Siberian husky, Alaskan malamute), which can actually become hyperthermic. Initially, general anesthesia-induced vasodilation allows for core body heat to be redistributed to the skin and extremities. This results in hypothermia-induced vasoconstriction that limits the amount of blood and heat delivered from the core to the periphery. Ultimately, this cascade of events cannot restore the core body temperature. Small patients are at the greatest risk, due in large part due to their small body-surface-to-mass ratio.

The majority of heat loss occurs within the first 20 minutes of general anesthesia for a multitude of reasons—the presence of cooler ambient temperatures and stainless steel induction tables, drug-induced vasodilation, breathing of dry anesthetic gases, shaving and surgical preps. Hypothermia is exacerbated by prolonged surgical procedures, especially those in which open body cavities remain exposed or when cold lavage solutions are utilized. In fact, one study demonstrated that the risk of skin infection increased by 0.5% for each additional minute of anesthesia time! This equates to a 30% increase in skin infections for each additional hour of anesthesia time!

Hypothermia is also considered a form of general anesthesia, as it increases the solubility of inhalants in the body, thereby effectively increasing the dose delivered. When using volatile anesthetics, for every 1.8°F decrease in body temperature there is a 5% decrease in MAC requirements. Critically ill or otherwise compromised patients may face adverse challenges when core body temperature decreases by as little as 2°F.

Although shivering is very effective in restoring body temperature, it is also associated with a significant increase in metabolic oxygen demand (40% to 200%, or more)! As this phenomenon may result in an oxygen debt, it may be prudent to administer all recovering shivering patients with supplemental oxygen.

Treating Hypothermia

The body obtains heat from 2 sources: endogenously, as described above, or exogenously, from environmental sources. Although there is controversy regarding the optimal methods, duration and rates for rewarming, there is no question that preventing hypothermia is easier and more efficient than treating it. Important considerations for evaluating rewarming systems include the heating element itself as well as the body surface area in contact with said heating element, and tissues in contact with the heating element. Furthermore, all cutaneous warming methods designed to transfer heat into the thermal core will rely on skin temperature, tissue insulation, and the body’s ability to circulate the heat (convection.) Since the core body surfaces (e.g., thorax and abdominal cavities) are relatively isolated from distal skin surfaces (e.g., extremities, tail), the efficacy of various warming devices can be unpredictable. Nonetheless, skin temperature is an important variable in regards to delivering effective cutaneous heat transfer.

Rewarming should be considered when hypothermic patients are less than 97.6°F. There are 3 techniques for rewarming chilly patients: 1, Passive external rewarming (PER), 2, Active external rewarming (AER), and 3, Active core rewarming (ACR).

PER is typically used to treat mild hypothermia and involves placing the patient in a warm environment and providing towels, blankets, or articles of clothing (e.g., sweaters) to help the patient regain thermal homeostasis. Patients treated with PER must be able to generate heat (e.g., shiver) to be effective. PER is the slowest method for treating hypothermia.

AER involves the application of external heat sources such as heating blankets, radiant heat lamps, heated rice bags, or immersion in warm water. Overall these methods tend to be non-invasive, inexpensive, readily available, and are easily employable rewarming methods. There are a variety of ways to maintain an envelope of warm air around hypothermic patients. Convection-type warm air devices (e.g., BAIR Huggers®) and electrically conductive fabric warmers such as the HotDogWarmer® (Augustine Biomedical + Design) are the most effective, followed by other warming units such as carbon-based conductive polymers by PetTherm (Inditherm plc), and circulating warm water blankets. At least 60% of the body surface area must be in contact with the external heat source for rewarming efforts to be most effective.

Other methods of maintaining body temperature during anesthesia include decreasing the oxygen flow rate (e.g., low flow anesthesia), utilizing a Humid-Vent adapter on the endotracheal tube, or using a coaxial (F-circuit) anesthesia hose. The coaxial design of the latter allows the patient’s exhaled breath to warm the incoming cold, fresh gases (e.g., oxygen, +/- an anesthetic agent.)

If latex gloves or bottles of warm water are to be used for smaller patients, it is essential that they are initially warmed to a temperature of ≤107°F and removed once they cool to the temperature of the patient, as at that point they begin contribute to heat loss rather than a heat gain. As such, these items are considered rather ineffective for raising body temperature, and pose an increased risk of thermal burns along the contact site.
Extreme caution is essential when using a microwave to warm rice bags, water bottles, lavage or intravenous solutions since ‘hot spots’ may occur due to uneven heating. Commercially available wire electric heating-pads and heat lamps have been associated with thermal injury and/or electrocution and should be avoided.

‘After-drop’ is another potential disadvantage of AER and occurs when heat is transferred from the core to the periphery of a hypothermic patient, thereby creating a large temperature gradient when colder peripheral blood is subsequently transferred to the relatively warmer core. The overall effect of an ‘after-drop’ results in a reduced core temperature. As a result, it has been suggested that warming the torso region while the (cooler) extremities remain vasoconstricted can be advantageous.

Although ACR is the most rapid method of rewarming, it is also considered the most invasive and therefore reserved for patients with severe hypothermia or with an arrest cardiac rhythm. ACR involves heat application to the body’s core via methods such as heat humidified oxygen, heated intravenous fluids, or warmed peritoneal, thoracic, gastric, rectal or urinary bladder lavage. One study demonstrated that when abdominal lavage fluids were warmed to 110°F and left in the abdomen for as little as 2- to 6-minutes, the patient’s temperature increased from 94°F to 97°F. However, the efficacy of warmed gastric, rectal or bladder lavage has not been well documented. Furthermore, warm fluid enemas will preclude the use of a rectal thermometer, necessitating the use of an ear thermometer instead. Esophageal warmers (Gaymar 800.828.7311) are also available to help warm hypothermic patients ‘from the inside out.’

The simplest core rewarming method entails warmed, humidified, inhaled oxygen (104°F- to 107.6°F), but this technique results in only a mild to modest heat gain. It should also be noted that the efficacy of warmed intravenous fluids is greatly dependent on the fluid rate and volume administered. Therefore severely hypothermic patients should be treated with multiple rewarming techniques such as combined truncal AER and ACR (e.g., warm peritoneal lavage or warm fluid enemas) methods.

Other investigational techniques being explored for the treatment of hypothermia include high temperature intravenous fluids and diathermy. Studies performed utilizing intravenous fluids heated to 149°F demonstrated rewarming rates of 37.2°F to 38.6°F per hour with minimal intimal injury. Diathermy involves delivering heat to deep tissues via ultrasound, low-frequency microwave radiation, or energy waves.

An important consideration when employing any rewarming technique is to taper or step-down the rewarming efforts as the patient’s temperature approaches normal parameters. This approach can help avoid causing a rebound hyperthermia, especially in cats and small dogs. Therefore it is prudent to monitor the temperature of hypothermic patients every 30 minutes.

Combating hypothermia should be paramount for every hospitalized patient; it’s easy, non-invasive, inexpensive, and a vital component of providing good patient care.

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See also: *National Association of Veterinary Technician in America (NAVTA) Journal*

Hypothermia—What’s the Hype? (June/July 2013)
PAIN SCORING FOR DUMMIES

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Pain can be defined as an adverse sensory and emotional experience.¹ Negative sequela resulting from pain may include immobility, inappetance, insomnia, catecholamine release, decreased pulmonary function and increased myocardial oxygen consumption. Additionally, cats may become mildly pyrexic.² As such, it is essential to be able to identify signs of acute and chronic pain in non-verbal species.

The pain pathway occurs during tissue damage when stimulated nociceptors release inflammatory mediators, which are subsequently encoded (transduced) into electrical activity, and transmitted along afferent nerve fibers to the dorsal horn of the spinal cord (modulation), before being projected to the brain (perception).³ Acute pain may be the result of trauma, surgery, medical conditions, infection or inflammation.⁴ Chronic pain in veterinary patients may be best described as pain extending beyond the anticipated healing period, with lasting neurophysical or psychological manifestations.⁵

Evaluation for pain should be part of every physical exam. Ideal pain scoring scales would create minimal inter-observer variations, and incorporate factors such as the type and duration of surgery, severity of pain associated with the procedure, hospitalization, age and concurrent diseases as well as individual variability.⁶

Pain Scales

Validated scales for assessing acute pain in companion animals include the Glasgow Composite Pain Scale, Melbourne Pain Scale and the Colorado Acute Pain Scale (see Figures below). Many objective scales for assessing chronic pain have been validated in dogs, including the Canine Brief Pain Index (CBPI), the Helsinki Chronic Pain Index (HCPI), the Cincinnati Orthopedic Disability Index (CODI), the Liverpool Osteoarthritis in Dogs (LOAD), and the Health-Related Quality of Life Scale, for dogs with chronic pain due to cancer.⁵

Pain Assessments in Dogs

Pain recognition in dogs should entail factors such as breed, age, individual temperament and stressors such as anxiety or fear. Behavioral signs of pain include change in body posture, demeanor, vocalization, decreased appetite, altered reactions to touch or interactions with people, or altered mobility.⁴

Fear Free Patient Handling

The concept of Five Freedoms surrounding the welfare of livestock animals was developed by the United Kingdom (UK) Government in December 1965, and subsequently formalized by the UK Farm Animal Welfare Council in 1979.² Dubbed the Brambell Report, it outlined the importance of five key aspects of animal welfare under human control, including 1) access to fresh water and food, 2) adequate shelter and bedding, 3) pain, injury or disease prevention, including rapid diagnosis and treatments, 4) sufficient space, proper facilities and the company of other animals, and 5) conditions and treatment to avoid mental suffering. Comprehensive veterinary patient knowledge extending beyond mere anatomy and physiology has prompted some training programs to add behavioral medicine into the curriculum for future veterinarians and veterinary nurses.

The Fear Free Certification Program was launched by the American Animal Hospital Association (AAHA) in March 2016, based on Dr. Marty Becker’s belief that providing a Fear Free patient experience leads to more accurate physical exams and laboratory testing, while resulting in less stress (including immune
suppression, vomiting and diarrhea) for veterinary patients, and ultimately leading to more pleasurable interactions with the veterinary staff.

According to a 2014 Bayer Veterinary Healthcare Usage Study, 37% of dog owners and 58% of cat owners reported that their pet hated going to the veterinarian, necessitating that Fear Free initiatives begin with travel desensitization at home.3 Perhaps the biggest impact on the physical and emotional well-being of all veterinary patients entails the ability to identify the signs of anxiety by interpreting body language, employing gentle handling methods, and using sedation before fearful or stressed patients reach critical thresholds.4 Other Fear Free handling strategies may incorporate the use of pheromones (e.g., Feliway®, MultiCat, Adaptil® available through CEVA Animal Health) on clothing, carriers and toys, the availability of soft, warm bedding, a constant flow of tasty treats, soothing music (e.g., iCalmDog®, iCalmCat® by Through a Dog’s Ear) and the use of non-harsh, odorless chemicals for cleaning between patients. Fear Free concepts in hospital design may entail quiet, slip resistant flooring and separate dog and cat triage and housing areas. Dr. Becker’s mantra— “Take the pet out of petrified and put pets back into your practice”—succinctly describes how Fear Free veterinary practices of tomorrow will experience increases in patient visits while strengthening the client-veterinarian team-patient bond.3, 5

### Colorado Acute Pain Scale for Dogs

<table>
<thead>
<tr>
<th>Pain Score</th>
<th>Example</th>
<th>Psychological &amp; Behavioral</th>
<th>Response to Palpation</th>
<th>Body Tension</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Comfortable when resting</td>
<td>Happy, content</td>
<td></td>
<td>Minimal</td>
</tr>
<tr>
<td>1</td>
<td>Content to slightly unsettled or restless</td>
<td>Distracted easily by surroundings</td>
<td></td>
<td>Mild</td>
</tr>
<tr>
<td>2</td>
<td>Looks uncomfortable when resting</td>
<td>May whimper or cry and may lick or rub wound or surgery site when unattended</td>
<td></td>
<td>Moderate</td>
</tr>
<tr>
<td>3</td>
<td>Unsettled, crying, groaning, tilting or chewing wound when unattended</td>
<td>May be unwilling to move all or part of body</td>
<td></td>
<td>Serious</td>
</tr>
<tr>
<td>4</td>
<td>Constantly groaning or screaming when unattended</td>
<td>May bite or chew at wound, but unlikely to move</td>
<td></td>
<td>Severe</td>
</tr>
</tbody>
</table>

**Colorado Acute Pain Scale for Dogs**

**Colorado State University**

### Pain Assessments in Cats

Pain assessments in cats pose unique challenges, and are often based on subjective behavioral changes. Physiological parameters such as heart and respiratory rate and pupil size are not consistently
linked to acute pain symptoms in this species, while factors such as patient temperament and environmental context can further confound interpretation. The only pain assessment scale validated in cats (following ovariohysterectomy) is the UNESP-Botucatu Multidimensional Composite Pain Scale (MCPS) (www.animalpain.com.br/en-us/avaliacao-da-dorem-gatos.php), although it is time consuming and may not be applicable for other types of surgical procedures or pre-existing health condition variables. Alternatively, the Glasgow Feline Pain Scale (CMPS-F) is an easier to utilize option, but has not been fully validated at this time.⁶,⁷

Pain assessments for cats should include both undisturbed cage observations and reactions associated with gentle handling techniques and wound palpation. Behavioral markers associated with discomfort may include a hunched posture or splinting of the abdomen, anorexia, lowered head position, growling, closed eyes, hiding or reaction to palpation, but facial expression changes such as furrowed brow, squinted eyes, whisker positioning or ear tip distance are currently under investigation. Cats in severe pain are usually depressed, motionless, and silent.⁴,⁶

**Colorado Acute Pain Scale for Cats**

Colorado State University
References:


Key Points:
- Improve surgical patient comfort and emotional well being
- Introduce the concept of a fear-free veterinary experience
- Augment perioperative surgical patient care experience for pet owners
- Describe the benefits of utilizing a surgical checklist

Veterinary nurses play a vital role in the care and management of surgical veterinary patients. Veterinary nurse responsibilities may encompass client education, participation in peri-operative diagnostics and work up, as well as serving in a scrub or circulating nurse role during the surgical procedure(s). As such, it is imperative that veterinary nurses can demonstrate knowledge in several key areas as part of a team approach to assure successful surgical outcomes, comfortable, happy pets and satisfied pet owners.

History

The value of obtaining an in depth and accurate history cannot be overemphasized. In addition to the presenting complaint, essential components of a thorough history include the patient's name, species and breed, age, sex (unaltered or intact) and breeding/estrus status, current diet and housing conditions (indoors versus outdoors), prior adverse reactions to anesthetic agents or drugs as well as the patient's prior medical history, preventative health status (e.g., date of last vaccine and fecal exam, etc.), and current drug therapy. Additionally, a brief summary of the status of major organs and systems can be obtained from the client utilizing specific (and non-leading) lines of questioning. For example, questions that provide pertinent information regarding the cardiovascular system would establish if the patient has exhibited recent coughing, difficulty breathing or exercise intolerance. Similarly, the state of the gastrointestinal tract (e.g., presence of vomiting, diarrhea, or altered water consumption), the neurological status (e.g., existence of seizure activity or history of collapse), and a dermatological (e.g., presence of rapidly enlarging masses, erythema, or pruritis) as well as general history (e.g., identifying behavior changes or decreased activity) can be obtained. All abnormalities should be further investigated with particular emphasis provided to geriatric patients with cardiovascular anomalies.

Physical Exam

A good physical exam is an imperative element of the pre-anesthetic assessment. Physical exams should be performed using a routine and systematic method. In addition to documenting the patient's overall demeanor, hydration status, weight and body condition (e.g., obese, emaciated), the patient's temperature, pulse, respiratory rate, mentation, mucous membrane color and capillary refill time (CRT) should be obtained. Hence, astute veterinary nurses can support the veterinarian by assisting in the detection of the presence of anemia, dehydration or hypovolemia. Assessment of the reproductive, cardiovascular and respiratory systems, evaluation of the skin, oral cavity and lymph nodes as well as abdominal palpation should also be performed. Again, all abnormalities should be further investigated. Preoperative work-up may consist of blood work, radiographs, or other diagnostic testing (e.g., blood pressure measurement, ECG rhythm strip, echocardiogram and/or abdominal ultrasound), which will be dependent on the abnormalities uncovered.

Fear Free Patient Handling

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Surgical Checklists

Preflight safety checklists have been used by the aviation industry since World War II, where they were considered a critical tool for helping pilots fly complex airplanes such as the Boeing B-17. The value of checklists in the human healthcare industry was popularized by Atul Gawande’s The Checklist Manifesto – How to Get Things Right, with inroads slowly creeping into the veterinary milieu.

Checklists have been used for a variety of clinical applications including anesthesia, dentistry, emergency medicine, and routine health care. Surgical checklists can be used to track all aspects of perioperative care, such as diagnostics, procedures and anesthetic requests, sample collection, and outline post-operative requirements so that critical tests, treatments and participant team roles are clear. Since communication issues play a factor in 80% of surgical errors, surgical checklists can prove pivotal in preventing medical errors while improving outcomes by increasing patient safety, reducing patient morbidity, and improving perioperative teamwork. In fact, dramatic decreases in post-operative morbidity (36%) and mortality (48%) were noted after implementing checklists.

Peri-Operative Client Communications

Veterinary nurses must counsel clients as to perioperative expectations surrounding scheduled surgical procedures, such as answering questions about admission guidelines, hospitalization policies, and postoperative care and expectations.

- **Obtain authorized signatures**: Ensure that the treatment plan (estimate) is signed and dated, and that resuscitation orders are clearly documented. A consent form outlines the surgical procedure(s) to be performed and details anticipated or potential complications associated with the procedure. Although all team members should be updated on the daily surgical schedule, veterinary nurses can help clarify medical jargon for pet owners and ensure the consent form lists the correct procedure and side (left vs right) and is appropriately signed.

- **Fasting instructions**: Current pre-operative fasting instructions include withholding food for 6 hours prior to surgery, while offering water until the patient is pre-medicated. One study demonstrated that dogs fed a canned food meal equivalent to ½ daily ration three hours prior to anesthesia had gastric volumes similar to dogs fasted for 10 hours. In fact, gastroesophageal reflux (GER) was noted in 15% of dogs fasted for 12- to 18 hours, yet none of the dogs fed two- to four hours prior to anesthesia developed GER.
• **Medications:** Approved medications and timing of administration are also necessary pre-surgical discussions. Non-steroidal anti-inflammatory drugs (NSAIDs) may be discontinued at the discretion of the surgeon. Preoperative administration of maropitant (Cerenia® Zoetis) can help prevent peri-operative emesis and encourage a quicker return to spontaneous eating post-operatively.

**Pre-Operative Preparations**

Preoperative planning and preparation are imperative for assuring successful surgical outcomes. Customized procedure and doctor specific checklists can be utilized to ensure that all necessary instrumentation, equipment and supplies are sterile, set up and readied in advance, which can help to expedite the surgical procedure. One study demonstrated that the risk of skin infection increased by 0.5% for each additional minute of anesthesia time. This equates to a 30% increase in skin infections for each additional hour of anesthesia time! Therefore, once the patient is anesthetized all efforts should be concentrated on getting that patient off the table and into recovery as soon as possible.

Anesthetic plans should always include appropriate analgesics (such as opioid, local anesthetic and NSAID combinations), airway management and intravenous fluids and/or colloids and blood products, as well as appropriate monitoring. Monitoring should focus on pulmonary, cardiovascular, central nervous system, and body temperature, since these systems are most affected by anesthetic drugs and surgical procedures. Moreover, the American College of Veterinary Anesthesia and Analgesia (ACVAA) recommends blood pressure monitoring as a minimum standard for managing the anesthesia care of moderate to severely ill patients. Other considerations may be based on the patient's size, breed, temperament, current health status or medications, or may be dictated by the type or length of surgical procedure. The ability to anticipate potential untoward effects and respond appropriately is also critical, necessitating a well-stocked crash cart and team members trained to use it.

Maintaining written anesthetic records allow informed, flexible and timely responses to changes in the patient's status while also serving as a legal document. Prior anesthetic episodes should be carefully reviewed with the intent of improving subsequent anesthetic events. It is also important to assign an American Society of Anesthesiologists (ASA) status, since ASA rankings > III are associated with increased patient morbidity and mortality.

**Postoperative Considerations**

"The postoperative period was the most common time for dogs, cats and rabbits to die usually within 3 hours of surgery...greater patient monitoring and management during this time period is recommended."

The care of the surgical patient does not end once the surgical procedure is finished; yet many anesthetists often neglect this crucial time period. It is imperative to understand that surgical patients can be faced with life-threatening situations during the postoperative period. These patients may already have or develop hypoxia, hypothermia, hypotension, hemorrhage leading to anemia, emergence delirium, vomiting, or cardiac arrhythmias (possibly leading cardiopulmonary arrest), in the minutes and hours following surgery. Furthermore, post-operative patients should be frequently assessed for pain, and additional analgesic medications administered as needed.

It is best to designate one person specifically to care for patients in the recovery ward. If this is not possible, then surgical patients should be recovered in a busy area of the hospital. This area should also be stocked with drugs and supplies necessary to address the most common post-surgical complications.

Aesthetically pleasing post-operative text images, phone updates (immediate and 24- hour post-operative calls) and Get Well Soon cards are gestures sincerely appreciated by most pet owners. Scheduled discharge appointments allows pet owners to be counseled on expected at home care such as medication administration tips, dosages and frequency, wound or bandage care, rehabilitation and other pertinent aftercare. Verbally reinforcing all written information helps increase compliance, and should be organized in a discharge folder containing other applicable information (e.g., drug inserts, doctor's business card and hospital contact information, rehabilitation brochures, incision care or activity toy handouts).
References:

2. www.sheltervet.org, accessed on December 5, 2016
Role of the Veterinary Surgical Scrub Technician
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Often described as the surgeon’s primary assistant, veterinary surgical scrub technicians (VSST) play an important role as part of the surgical team. Notable features of a great scrub technician include familiarity with the surgical procedure, surgeon’s preferences and needs, as well as the ability to anticipate, even when things go wrong. VSSTs can augment the veterinarian’s recommendations to the client surrounding their pet’s surgical procedure as well as assist with client education during all phases of care (pre-, intra- and post-operative.)

History

The value of obtaining an in depth and accurate history cannot be overemphasized. In addition to the presenting complaint, essential components of a thorough history include the patient’s name, species and breed, age, sex (unaltered or intact) and breeding/estrous status, current diet and housing conditions (indoors versus outdoors), prior adverse reactions to anesthetic agents or drugs as well as the patient’s prior medical history, preventative health status (e.g., date of last vaccine and fecal exam, etc.), and current drug therapy. Additionally, a brief summary of the status of major organs and systems can be obtained from the client utilizing specific (and non-leading) lines of questioning. For example, questions that provide pertinent information regarding the cardiovascular system would establish if the patient has exhibited recent coughing, difficulty breathing or exercise intolerance. Similarly, the state of the gastrointestinal tract (e.g., presence of vomiting, diarrhea, or altered water consumption), the neurological status (e.g., existence of seizure activity or history of collapse), and a dermatological (e.g., presence of rapidly enlarging masses, erythema, dermatitis or pruritis) as well as general history (e.g., identifying behavior changes or decreased activity) can be obtained. All abnormalities should be further investigated with particular emphasis provided to geriatric patients with cardiovascular anomalies.

Physical Exam

A good physical exam is an imperative element of the pre-anesthetic assessment. Physical exams should be performed using a routine and systematic method. In addition to documenting the patients overall demeanor, hydration status, weight and body condition (e.g., obese, emaciated), the patient’s temperature, pulse, respiratory rate, mentation, mucous membrane color and capillary refill time (CRT) should be obtained. Hence, astute veterinary nurses can support the veterinarian by assisting in the detection of the presence of anemia, dehydration or hypovolemia. Assessment of the reproductive, cardiovascular and respiratory systems, evaluation of the skin, oral cavity and lymph nodes as well as abdominal palpation should also be performed. Again, all abnormalities should be further investigated. Preoperative work-up may consist of blood work, radiographs, or other diagnostic testing (e.g., blood pressure measurement, ECG rhythm strip, echocardiogram and/or abdominal ultrasound), which will be dependent on the abnormalities uncovered.

Surgical Checklists

Preflight safety checklists have been used by the aviation industry since World War II, where they were considered a critical tool for helping pilots fly complex airplanes such as the Boeing B-17. The value of checklists in the human healthcare industry was popularized by Atul Gawande’s *The Checklist Manifesto – How to Get Things Right*, with inroads slowly creeping into the veterinary milieu.

Checklists have been used for a variety of clinical applications including anesthesia, dentistry, emergency medicine, and routine health care. Surgical checklists can be used to track all aspects of perioperative care, such as diagnostics, procedures and anesthetic requests, sample collection, and outline post-operative requirements so that critical tests, treatments and participant team roles are clear. Since communication issues play a factor in 80% of surgical errors, surgical checklists can prove pivotal in preventing medical errors while improving outcomes by increasing patient safety, reducing patient
morbidity, and improving perioperative teamwork. In fact, dramatic decreases in post-operative morbidity (36%) and mortality (48%) were noted after implementing checklists.

**Peri-Operative Client Communications**

Veterinary nurses must counsel clients as to perioperative expectations surrounding scheduled surgical procedures, such as answering questions about admission guidelines, hospitalization policies, and postoperative care and expectations. Obtain authorized signatures: Ensure that the *treatment plan* (estimate) is signed and dated, and that resuscitation orders are clearly documented. A consent form outlines the surgical procedure(s) to be performed and details anticipated or potential complications associated with the procedure. Although all team members should be updated on the daily surgical schedule, veterinary technicians can help clarify medical jargon for pet owners and ensure the consent form lists the correct procedure and side (left vs right) and is appropriately signed.

Fasting instructions: Current pre-operative fasting instructions include withholding food for 3-6 hours prior to surgery, while offering water until the patient is pre-medicated. One study demonstrated that dogs fed a canned food meal equivalent to ½ daily ration three hours prior to anesthesia had gastric volumes similar to dogs fasted for 10 hours. In fact, gastroesophageal reflux (GER) was noted in 15% of dogs fasted for 12- to 18 hours, yet none of the dogs fed two- to four hours prior to anesthesia developed GER.

Medications: Approved medications and timing of administration are also necessary pre-surgical discussions. Non-steroidal anti-inflammatory drugs (NSAIDs) may be discontinued at the discretion of the surgeon. Preoperative administration of maropitant (Cerenia® Zoetis) can help prevent peri-operative emesis and encourage a quicker return to spontaneous eating post-operatively. Careful assessment of nutritional supplements is also advised.

**Pre-Operative Preparations**

Preoperative planning and preparation are imperative for assuring successful surgical outcomes. Customized procedure and doctor specific checklists can be utilized to ensure that all necessary instrumentation, equipment and supplies are sterile, set up and readied in advance, which can help to expedite the surgical procedure. One study demonstrated that the risk of skin infection increased by 0.5% for each additional minute of anesthesia time. This equates to a 30% increase in skin infections for each additional hour of anesthesia time! Therefore, once the patient is anesthetized all efforts should be concentrated on getting that patient off the table and into recovery as soon as possible.

The VSST may organize the surgical case load for the day, as well as ensuring the operating room (OR) is clean and disinfected prior to beginning surgery. One study indicated that up to 20% of hospital acquired infections were caused by contaminated surfaces. Routine OR cleaning (including walls, floors, counters, OR tables, mayo stands, and patient tables, OR lights, and other equipment present in the room) should be performed routinely, as well as between cases. All organic material (blood, pus, fecal material, etc) must be completely removed from surfaces prior to disinfection.

Accelerated hydrogen peroxide disinfectants have gained popularity over quaternary ammoniums due to their efficacy and low odor properties. Regardless of the disinfectant used, always follow manufacturer’s guidelines regarding proper dilution and surface contact time. Agent specific dip stick tests can be used to verify the strength of diluted disinfectants. It is important to remember that all surfaces should remain wet throughout the entire contact time frame. HaloFogger™, an EPA-approved hydrogen peroxide dry mist, is effective against 99.999% of *Clostridium difficile* (*C. diff*) spores. Change mop heads daily, and store empty mop buckets in a manner that allows them to completely dry between uses.

Prior to the procedure the VSST assembles all surgical supplies and equipment in a chronological use organization, while carefully assessing packaging integrity and inspecting external chemical indicators. The VSST oversees surgical patient preparations, ensuring trauma-free hair removal, and the application of appropriate topical antiseptics. A preliminary ‘dirty’ patient scrub may consist of a 2-minute (minimum) application of 2% or 4% chlorhexidine gluconate scrub (at least 3 consecutive scrubs) followed by a
similar application process utilizing 70% alcohol. It is important to note that use of friction while prepping skin is the key to preventing infection. Chloraprep® is a one-step final prep, consisting of 2% chlorhexidine gluconate and 70% isopropyl alcohol. Chloraprep® is effective against a broad spectrum of microorganisms, including methicillin-resistant staphylococcus aureus (MRSA), vancomycin-resistant Enterococcus (VRE), C diff, coagulase-negative staphylococci and most viruses and fungi. Chloraprep® becomes fully effective after 2-3 minutes, with 48+ hours of long term efficacy. Other important roles of the VSST include the timely administration of perioperative antibiotics, and assisting with proper patient positioning once inside the OR.

Central suction canisters should be clean and hooked up to prevent leaks. Nitrogen- or battery-powered orthopedic equipment should be checked to ensure there are adequate gas or charge levels available to complete the surgical procedure. Electrosurgery units must be adjusted to the correct setting, and the grounding plate adequately prepared. Radiosurgery units utilize 4 MHz radio waves (e.g., Ellman Surgitron) that pass from the active electrode (cautery pencil) to the passive electrode (grounding plate). This type of cautery unit has a plastic coated grounding plate that acts similar to an antenna, and should be located adjacent to the surgical site. Bovie Medical developed electrosurgery units in the 1920’s. The metal grounding plate should be located as close to the surgical site as possible, using appropriate amounts of conduction gels to contact patient skin, thereby decreasing cutting power and minimizing lateral thermal damage. Patient burns may be associated with high currents, long activation times, and use of conductive fluids. (3MBulletin 2007)

During electrosurgery, aerosolized blood droplets can be propelled a distance of up to 30 cm. Surgery smoke can contain viable viruses, bacteria, hazardous chemicals and carcinogens. Viable human papilloma virus (HPV) has been identified in vapor of genital warts treated with electrocoagulation. Use face masks, eyewear, surgery gloves, and smoke evacuation systems to minimize exposure to OR personnel. Disposable or sterilized electrodes should be used.

The VSST may facilitate gowning and gloving of the surgical team, while surveying for breaks in sterile technique. Intra-operatively, the VSST should ensure the instrument table is kept organized and clean, track sponge counts and sharps in addition to assisting with hemorrhage control, retracting or moistening tissues and cutting sutures. Memorization of the surgical procedure allows the VSST to anticipate the surgeon’s next move, and recognizing complications can improve response time. As such, it is beneficial if team members are cross-trained with anesthesia technicians for this role.

Postoperative Considerations

“The postoperative period was the most common time for dogs, cats and rabbits to die usually within 3 hours of surgery...greater patient monitoring and management during this time period is recommended.”

The care of the surgical patient does not end once the surgical procedure is finished; yet many anesthetists often neglect this crucial time period. It is imperative to understand that surgical patients can be faced with life-threatening situations during the postoperative period. These patients may already have or develop hypoxia, hypothermia, hypotension, hemorrhage leading to anemia, emergence delirium, vomiting, or cardiac arrhythmias (possibly leading cardiological arrest), in the minutes and hours following surgery. Furthermore, post-operative patients should be frequently assessed for pain, and additional analgesic medications administered as needed.

It is best to designate one person specifically to care for patients in the recovery ward. If this is not possible, then surgical patients should be recovered in a busy area of the hospital. This area should also be stocked with drugs and supplies necessary to address the most common post-surgical complications.

Aesthetically pleasing post-operative text images, phone updates (immediate and 24- hour post-operative calls) and Get Well cards are gestures sincerely appreciated by most pet owners. Scheduled discharge appointments allows pet owners to be counseled on expected at home care such as medication
administration tips, dosages and frequency, wound or bandage care, rehabilitation and other pertinent aftercare. Verbally reinforcing all written information helps increase compliance, and should be organized in a discharge folder containing other applicable information (e.g., drug inserts, doctor’s business card and hospital contact information, rehabilitation brochures, incision care or activity toy handouts).

References
Browning, D., and Tobias, K., Preoperative Roles and Responsibilities of the Veterinary Surgical Nurse, Today’s Veterinary Technician, Vol. 1, No. 4, 2016, pp7-15
Welsh, E. Anaesthesia for Veterinary Nurses Blackwell Science, Ltd. Malden, Massachusetts, 2003; 34-54.
Frankel, C., Veterinary Medicine Needs Checklists, Cliniciansbrief.com, pp. 60-62, February 2017
Haynes, AB, et.al., A surgical safety checklist to reduce morbidity and mortality in a global population, New England Journal of Medicine, 2009; 360; 491-499.
Liska, Wm D., Technician Utilization in a Surgery, Proceedings American College of Veterinary Surgeon’s Symposium, Nashville, TN, pp 745-748
Zeltzman P., Hypothermia is Surgical Patients, Proceedings American College of Veterinary Surgeons Symposium, 2009; 980-982.
American Association of Feline Practitioners 2018 Feline Anesthesia Guidelines
Veterinary Nursing in Action 2016 Jun/Jul pp47 (pdf)
Medscape Dec 14, 2017
Adams, D et al, J Hosp Infection 2005 61:287-90,
Crosby, CT Mares, AK, J Vasc Access Devises, 2001: Spring 26-31
Employee reviews: A guide to effective fearless performance management

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All too often employee reviews center around digging up past errors and are not focused enough on motivating the employee and setting goals. Reviews should be performed at least every 6 months, with check-ins with the team member in between. Employees are less likely to become anxious at review time, or taken by surprise with the feedback if you have already been regularly speaking with them, and discussing concerns in the moment. Another method of reviewing employees is implementing a 360 review. While this should not serve as the only evaluation for an employee, it can help provide feedback from their peers and subordinates on how they perform in their eyes day-to-day.

Reviews may include a rating system, but it’s of the utmost importance to include commentary for each item that clearly backs up the reasoning for the rating. If the manager enters the meeting with supplemental examples, this can help the employee better understand instances where they need to improve and will add weight to your development items. It is also advisable to add notes from your discussion with the employee onto the review, so you can notate if they agreed or disagreed with the feedback, had ideas to share regarding how they plan to improve (so that you can hold them accountable to these plans in the next review), and if they had sufficient reasoning for why certain goals were not met.

Try to also ask open-ended questions so that you can understand their strengths and weaknesses through their eyes. It helps to ask what kind of additional support they feel like they need day to day. Encourage team members to set goals of their own as well as those that you feel are necessary. It can make the conversation more fluid to ask the employee where they feel their strengths are before going into your own feedback, the same goes for criticisms of their work. It’s possible that the employee may already be very aware of their deficiencies, making it not as difficult for you to reveal these to them. Be sure to have your positive feedback outweigh the concerns. Create a follow up plan to meet again and come up with actionable goals to reach by certain deadlines.

For a different perspective, 360 reviews can help share feedback with an employee on how team members at all different levels feel they are performing. These reviews can actually make providing difficult feedback easier as you will have the backing of multiple employees. It’s important to not go over every single negative comment in a 360 review, as there is the potential for one team member to share feedback based off of a recent incident that isn’t a true representation of the individual’s overall performance. I typically look for 2-3 themes within 360 feedback, where there are repeated comments and examples supporting the need for further development in a certain area. Focusing on 2-3 development goals is also advisable so the employee does not feel overwhelmed or discouraged.

The 360 approach can also help reveal strengths the manager may not be aware of if they are not regularly on the floor with the team member. This can be a great motivator for team members, and help to positively reinforce good behaviors. From a big picture perspective, this process helps the entire team feel that their voices can be heard without negative repercussions. Often in environments without 360 reviews, there is the awkwardness of having to pass along negative feedback while worrying that somehow it will come back to you or could place your job in jeopardy if the feedback involves a supervisor.
It’s vital to ensure privacy when 360 feedback is submitted. There shouldn’t be one person who pulls all of the data-only supervisors should be able to see the feedback pulled. Without this ensured privacy, it’s less likely that staff members will provide honest feedback and the integrity of the process will be questioned.

The questions asked in a 360 review should be carefully thought through. It helps to tie in the core values of the company into the questions, to further perpetuate the importance that these hold in day to day operations. There shouldn’t be so many questions that team members feel they can’t give genuine thought to each item and have to rush through them. It’s also important to provide time off the floor for team members to complete 360 feedback, so they do not feel they either have to do it off the clock or not at all. Be sure to create a follow up plan to administer the feedback versus handing all of the raw data over to the employee without any discussion. It’s advisable to create a summary of the data and pull a few supporting comments from the review to include along with that summary.

Employee reviews take a great amount of effort and time, but if you plan to build your employees and help them succeed, they are vital. If done correctly, these meetings can help your team members feel supported and have greater direction. Reviews also help keep an open line of communication between management and the team.
High impact communication: Say what you mean quickly, effectively and harmlessly

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In veterinary medicine, it’s safe to say we are way better at communicating with animals than with our fellow human colleagues. Even outside of veterinary medicine, the workplace is generally considered not a safe place to be 100% honest with your feelings and opinions. However, it doesn’t have to be this way and in fact, it can be much healthier to have more open communication. While approaching these difficult conversations can be scary at first, with practice, they become second nature.

Prior to any meeting, you should consider what you want to gain out of the meeting. You never want to enter a difficult conversation with the purpose of being vengeful or critical to be mean. The person initiating the discussion should have genuine interest in helping the other team member improve and strengthen their relationship and work environment. It’s helpful to try to consider the other person’s point of view prior to the situation as well. Most of the time, employees aren’t lacking in their performance due to not caring or being lazy. There’s often more going on that is causing this change in behavior, and it’s important to give the benefit of the doubt before meeting. This will make the conversation more even-keeled and the person initiating the conversation more approachable. When discussing the issue, focus on the behavior versus assessments of the behavior. If you make an inference such as someone being “lazy”, that will automatically send the individual into defense mode and lead to a completely unproductive conversation.

It’s important to come to the table with specific examples to back up concerns you want to address. Trying to address issues without examples only leads to defensive behavior and damaged relationships. This also helps the individual improve in the future as they can more easily catch themselves in the moment and course correct if they know when they tend to exhibit the problematic behavior. Along with outlining the examples you plan on using, it’s important to plan out how you want to deliver the information and prepare for how the other person may react. Prepare ahead of time with questions they may ask and how you will respond. The last thing you want to do during a tough conversation is to be thrown off and appear flustered, which often turns to agitation.

It can be helpful to have a supervisor, who you can trust with the information, to role play with you to practice an upcoming tough conversation. Not only can they provide feedback and guidance, but practicing how you will approach the conversation will make you more relaxed and comfortable when you have the real one. Role playing can help produce potential responses you may not have considered, which allows you to be more prepared with a thoughtful response when the actual meeting occurs.

Focus on yourself and the team and the effect the behaviors have on these individual(s) versus pointing fingers during the meeting. Avoid “you” statements, and try to predominantly utilize “I” statements, such as “I feel like when X happens...”, or “When I see X, it makes me frustrated”. This makes the conversation less confrontational and aggressive towards the individual, but more so focused on the behavior as a separate entity. This is not to say the action plan does not need to involve changes that the recipient of the feedback needs to own and make, but it will be a lot easier for them to hear the criticisms and be open to change.
Try not to beat around the bush when starting a conversation. While it’s fine to start off by pointing out something you appreciate about the team member to show that you are not only full of criticisms, it is important to somewhat quickly get to the point of the meeting. Chances are the individual has picked up on clues that this conversation was coming so they will also appreciate you jumping to the point.

Be sure to be a part of the solution, and offer up ways to help the individual improve. Ask how they would prefer to receive feedback moving forward— in the moment, or later in the day (or the next day). Come up with a follow up plan to meet again and discuss how matters have progressed. Even if everything seems to improve quickly, to maintain this change in behavior long-term, it’s important to still touch base and keep the conversation present in the individual’s mind. The positive reinforcement will help make these behaviors a permanent part of their work personality and ethic.
Leading from the middle: Non-positional leadership

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It's always hard to explain to a team member that they didn’t get the promotion to a managerial position. However, it's important to remind staff that leading is not accomplished by virtue of a title. It’s about the way you interact with your coworkers and go about each work day. Attitude and enthusiasm go a long way. When an individual stays positive, even when everyone knows they are likely frustrated or disappointed in the situation, this helps their team members rise up and stay motivated. This person is leading through influence with their enthusiastic, positive outlook.

Regularly showing up on time and ready to work, may seem like a small task, but it’s one that many struggle with, and it matters a lot. It proves accountability and leads to respect among your team. If the team knows they can count on you, they are much more likely to listen to your opinions and view you as a leader within the team. This also shows you are able to lead yourself. Individuals whose lives seem to constantly welcome drama and issues tend not to be the best leaders as they are more focused on keeping their heads above water than helping others. Part of accountability also relates to following through on what you say you are going to do. If you make a commitment to your team or any individuals on your team, you need to follow through with this responsibility no matter how much work ends up being involved. This will help gain trust among your teammates.

In addition to reliability, it’s important to show support to coworkers. Leaders let go of their control issues and try to serve their team. They want to know how they can best contribute to the team and what they can do to help each individual succeed. Even when team members fail and make mistakes, they are there to help them out, whether it be through trying to mend an error or offering advice for the future. Just be sure this advice is provided from an appropriate position if you are not the “boss”. It can be helpful to approach a situation like this by saying, “I know, I made a similar mistake awhile back and what I did to help myself moving forward was to…”.

To become influential, it’s important to exhibit flexibility. Leaders cannot be stubborn and unwilling to try out different methods to get from point A to point B. They should be open to creative, different approaches from their team members that they might not have come up with themselves. They are also always looking for ways to make things better. They don’t settle for the office functioning smoothly as being the end of the road. There is always a new way to wow a client, to provide better patient care, to boost revenue, or even boost morale.

Leaders, title or not, are great active listeners. They seek opportunities to learn from others by truly hearing out their ideas. You need to genuinely care about what your team members are saying and what is important to them. Leaders are inherently people who enjoy helping and interacting with others.

It's also important to not always try to be in the limelight. When team members accomplish great things, recognize this and provide apt credit. You may be kicking yourself, thinking “I wish I thought of that!”, but being humble enough to acknowledge others’ successes shows maturity. You may also find you learn something new from another team member's ideas and perspectives, which helps you grow your own concepts.

Find your own strengths, within your career and personal life, and draw upon those. Ensure healthy habits and a lifestyle that allows you to function at 110% each day you come into work. It’s very difficult to be struggling and disorganized in one aspect of your live and thrive in the other. It’s also important to constantly be seeking self-improvement. Find opportunities for continuing education and career growth, and never stop pursuing these above and beyond activities, which will demonstrate your passion not only for leadership, but also for self-growth, to the higher ups within your organization.

Remember, if you are to wait to receive a title to start leading, it will likely never come to fruition. Hiring managers look for individuals who have proven themselves on the floor to embody the essence of
a true leader, before they are promoted. An individual who has already shown themselves to be reliable, helpful, supportive, flexible and positive, is a perfect fit for a leadership position (and title).
New kid on the block: Gaining respect and effectiveness as an entry-level leader

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When first entering a starter manager role, it’s important to get to know your team and make yourself approachable. It’s crucial to reach out to team members individually and ask- what did they like about previous management? What would they change? Aside from one on one pow-wows in the beginning, it may be helpful to send out an anonymous survey to get a pulse on the current office morale. Is everyone cautious of opening up to management due to being burned in the past? Are they really nervous about the change to come? This can help assess their concerns and address them directly with the group. Be sure to ask team members what their ideal team environment looks like, and under what conditions they work best at work.

Another way to pull the team together and motivate and engage staff is to set up team building opportunities. During team meetings, be sure to add a fun game in the end as a bonding strategy. This will also help humanize a new manager so they are more approachable. It’s important to demonstrate supportiveness and a down-to-earth approach in the beginning so that team members feel comfortable and develop trust. Getting started on the wrong foot in the beginning is much harder to correct later on.

Part of developing trust and being approachable is to communicate, and do it a lot- even if it seems excessive, the last thing you want is for team members to not feel listened to. The complaint usually heard from staff is that they are not communicated with enough, rarely is it that they wish their manager would check on them less.

When making decisions early on, it’s important to have transparency in these decisions and explain how they correlate to the company’s (and your own) core values. Make expectations of the team and individuals very clear from the beginning so there are no surprises and they know what their role is upon boarding this new ship. Also clearly explain protocols and processes so you do not risk individuals not feeling confident in how to go about their job, or being hesitant to contribute. When developing goals, try to incorporate the team’s opinions and set high-level, but achievable goals. Periodically provide updates on where you are at as a company (incorporate key performance indicator data, along with soft data from the team’s experiences). It can be helpful to find an easier-to-reach goal in the beginning and work towards achieving this first, as small wins early on will help build your team’s confidence in your abilities. This helps build team momentum and motivates the group as a whole. One way to find projects to get things started is by asking your team if there is anything they feel they should stop doing or is a waste of time, and is there anything they feel would really make their job easier (& clients happier)?

Remember, you will make mistakes- this is natural, especially for a new leader. How you handle this bump in the road is what matters. Try not to focus on explaining away why you made the mistake as this can come across as being overly defensive. Instead, acknowledge the error, own it, and explain how you will make sure it never happens again. This attitude will be much more readily embraced by your team.

Be sure to utilize your team and trust them. You may think you will impress a new group by solving all the problems on your own and doing all of the work for them. Approaching your work this way will not only likely lead to errors and failure on your end, but it will demotivate your team as they will no longer feel empowered to accomplish tasks on their own. It also can appear that you lack the necessary trust to relinquish these duties.

When demands become too high and the team is becoming overworked and burnt out, stick up for your team. Talk to higher ups and owners and be the voice of the staff. Explain their concerns, and make them your own. Come in with ideas for solutions to these problems, and be sure to keep your team in the loop as to what changes you are trying to implement on their behalf.
In addition to monitoring when the team is feeling exhausted, it's important to acknowledge their achievements every day. Try to set up a rewards system to regularly acknowledge their hard work. You can also ask the team how they prefer to be recognized as this may vary between team members (some may prefer public recognition, while others like private kudos, etc).

Always try to be learning, especially in the early stages. If there is another manager you know—whether it be at another hospital or within your company, utilize their advice and support as a mentor. There will be tough situations you will encounter that you likely do not know how to handle, but they may have previous experience and wisdom to draw upon to help you out. Join the VHMA (Veterinary Hospital Manager’s Association) for networking opportunities as well as continuing education leads. For new leaders, communication and emotional intelligence CE tend to prove the most useful.
Who runs the world? Top issues faced by female leaders and how to overcome them

Ori D. Scislowicz, BS, LVT, aPHR

While we as women have made huge progress in the world of leadership, there are still unique obstacles that typically women face in the workplace. Part of the movement needs to revolve around working for and supporting those organizations who value inclusion and diversity. The rest of our struggles become less overwhelming in a supportive, inclusive environment.

Remaining confident in the face of those who may try to doubt your abilities or knock you off track is vital. Women are more likely to try to be overly polite and cautious in their suggestions. Eliminate passive language such as “just” (such as “I was just thinking...”) or “if that’s okay” from your vocabulary. Understand your purpose and goals, and don’t be afraid to voice them. Speak up during meetings and do not allow yourself to be interrupted. There is a tendency for women to be interrupted more often than men, whether the offender is male or female. Interruptions can serve as a dominance tactic, but there are ways to stop these behaviors. Instead of bowing down to the interrupter, continue your thought, and louder if necessary so the interrupter eventually forfeits. Techniques such as employing a talking stick can help make it clear that interruptions will not be tolerated and there is an additional visual cue to remind individuals not to do so.

Many women use neutral language not only out of a lack of confidence, but also out of fear of coming across as a “bossy” female leader. There are ways to express dominance as a female leader in more subtle ways that do not create a perception of bossiness. Standing tall, taking a ‘power stance’ where you physically take up more space, can make a person appear dominant without coming across as threatening.

When feeling like you are being pushed aside or put down in the workplace, it’s important to take steps to ensure you are an expert in your area. Regularly partake in continuing education, and come up with innovative ways to improve your organization. It’s much harder to belittle someone’s opinions when they have proven themselves and their level of expertise. It also helps to build female allies in the field, which will help boost confidence and provide a sounding board for concerns and issues. Align yourself with decision makers in the field and within your organization. Voice your opinions with confidence in these circles.

Imposter syndrome is also plaguing our female leaders. We often play down and even doubt our accomplishments and have a persistent fear that we will be exposed as not being cut out for our position. It helps to start by learning how to accept and believe compliments that are handed to you. Self-motivation can help reframe the habit of negative internal commentary. Reminding yourself of the positive consequences if you can meet a certain goal, or journaling recent successes on a weekly basis can help re-train the mind. A mentor can help provide support and encouragement as well, and is highly recommended for any newer leader.
Financially, women need to not be timid or afraid to ask for what they feel they are worth. Especially in this field, we are trying to catch up to the salaries in comparable jobs, whether it be a human nurse or an HR manager in a non-veterinary facility. If more individuals refuse to accept lower salaries, the standards will change over time. Sometimes women will shy away from selling themselves for fear of being perceived as conceited or too boastful. We should instead stand in our success and do not be afraid to recognize that we have earned our positions and success.
How to talk to doctors about clients

Often veterinary professionals needlessly struggle with how to have productive discussions with clients about concepts where the perspective gap between the veterinary professional and the client differ widely. In these instances, if the veterinary professional is going to be successful in maintaining the relationship with the client, move the client to action, and help the pet, then the veterinary professional must learn how to guide the conversation in such a way so that a shared solution is created.

In these conversations, the goal should be understanding the client’s perspective, expressing empathy, creating trust and a new relationship with the veterinary professional, whether it be a technician or a veterinarian, and ultimately, moving the client to action on behalf of the pet.

It starts with attunement, otherwise known as perspective taking. If we are going to be able to help the client, we need to understand why they are in the exam room in the first place. The better we know the client’s core values, and their needs, wants, and desires, then the better we can help guide their perspective.

Often the first person to speak with the client is a receptionist or technician. It is important for technicians to use the time wisely and not only obtain a presenting complaint and history but also use active listening and open ended questions (continue reading for examples) to help paint a clearer picture. Often these clients come in scared because of the health status of their furry family member. When they are scared they can be more reactive but also forget details. Be as empathetic as possible when speaking with clients who appear visibly upset.

Unfortunately, veterinary professionals can be so full of their own perspective that there is no room for the client’s. In these situations, the pets lose. There is a reason for this: power causes an individual to anchor too firmly to his or her perspective. In a series of social experiments by Adam Gallinsky et al in 2008, he found that across the board, the person with the most power in a relationship, any relationship, the person
with the most power had the more difficult time taking the perspective of the person with perceived ‘lesser power’. Historically speaking, veterinary professionals have held all the power in the veterinary-client relationship. We have the stethoscopes, the caduceus symbol, the white coats, the knowledge, and ultimately, control of the drugs. In the past, when information was asymmetrical and clients had no way of researching or understanding medical concepts, this worked out fine, but in the age of information symmetry (a.k.a. Dr. Google), this no longer serves the veterinarian or the client. Instead of resisting Dr. Google, we could use it to our advantage, and realize that the internet has led to better educated clientele who want a transparent, honest discussion with their veterinarians that includes respect and understanding from both sides.

Gaining perspective can be difficult if you aren’t used to doing it. Using open ended questions that invite story from the client is a good way to start. Questions could include:

Tell me what you are thinking.
Tell me more about…
What have you researched?
What do you think about the plan moving forward?
What are you thinking?
How do you feel about the plan moving forward?
What do you know about the condition?
What are your concerns?
Help me understand…

It is very important that once the client starts talking you shut your mouth and actually LISTEN. A recent study showed that clients are interrupted on average 15-18 seconds after they start talking during an office call.

Another barrier to productive client communications over disagreements or conflicts is reactive behavior. Discussions about things like researching medical conditions or veterinary recommendations on google, breeder recommendations, and vaccines can certainly evoke reactive behavior if not handled properly. If technicians learn how to recognize reactive behavior in their clients that indicate that the client is triggered into fight, flight, or freeze, then the technician can adjust communications as necessary to maintain a productive, professional relationship that benefits all parties involved, including the pet.

When you have obtained a history and are going to give this information to your doctor, be as detailed as possible. Also, pay attention of your body language. If you
are visibly upset with the client or show annoyance, you are conveying this information to your doctor without even knowing.

**How to talk to doctors about behavior change**

Sometimes, vets can be a real pain in the ass to talk to, especially when the situation is critical of something in regards to the veterinarian. If you need to talk to your doctor about a behavior that they need to change, it is best to utilize non-violent communication techniques. The steps to express your needs, as presented by Marshall B. Rosenberg, PhD in his book ‘Nonviolent Communication’ include the following 4 components:

1. **Observe** the behaviors in the doctor that are impacting your well-being or work environment
2. **Notice** the feelings that arise as a result of the doctor’s behavior, either good or bad.
3. **Honestly** share how you feel in relation to the observations you have made.
4. **Clearly request** a concrete action in order to enrich your life or improve your work environment.

In contrast, if your doctor shares a need for a behavior change from you using the same four components, your job is to empathetically receive this information and modify your behavior to improve the situation.

A good tool to use with the nonviolent communication style is ‘I feel’ statements. Feel free to use the following skeleton to frame your requests to your doctors:

```
When I notice (fill in the blank undesirable behavior) it makes me feel (insert feeling here) because I need (insert need here). Would you be willing to (insert very specific request here).
```

Things that can block communication flow between doctors and technicians:

1. Making judgements or global statements about the other’s character. “You are lazy.” “Your behavior is inappropriate.”
2. Making comparisons to other staff members.
Tweakers and tokers: CNS intoxicants (Part 1)
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ASPCA Animal Poison Control Center, Urbana, IL

Toxins that affect the nervous system can vary in clinical signs depending on the dose and route of exposure. The brain is susceptible to injury as it receives a high percentage (20-25%) of cardiac output, requires a continual high level of oxygen and nutrient supply. The blood-brain barrier and blood-cerebrospinal fluid barrier help protect the brain from toxins. See table 1 and 2 for a list of some CNS toxicants.

Amphetamines
Amphetamines can be found in both prescription ADHD and weight loss medications (Ritalin®, Adderall®, Vyvanse®, Concerta®), as well as illicit substances (methamphetamine, crack). Amphetamines are sympathomimetic alkaloids. They stimulate alpha- and beta-adrenergic receptors, causing the release of endogenous catecholamines at synapses in the brain and heart. This stimulation causes peripheral vasoconstriction and cardiac stimulation resulting in hypertension, tachycardia, ataxia, agitation, tremors, and seizures. Amphetamines also increase serotonin and dopamine release and act directly on dopamine receptors.

Peak plasma concentrations of amphetamine occur 1-3 h after ingestion of immediate release or illicit products. Amphetamines are highly lipid soluble and readily cross the blood-brain barrier. Concentrations in the cerebral spinal fluid can be 80% of those found in the plasma. Methamphetamine has increased partitioning to the CNS compared to other amphetamines.

Amphetamines undergo significant hepatic metabolism. The two major pathways are hydroxylation and deamination. Deaminated products can be oxidized and conjugated to glycine. Amphetamine and its metabolites are excreted primarily in the urine. About 30% of an amphetamine sulfate dose is excreted unchanged in the dog. Urine excretion rate is significantly affected by urine pH. Amphetamine is almost completely eliminated within about 6 h in dogs with an average urinary pH of 7.5 and in 3.3 h if the urinary pH averages around 6.

Asymptomatic animals may have emesis induced and activated charcoal administered. Fluid therapy is important to enhance elimination and maintain CV stability. Agitation and hyperactivity responds best to phenothiazines. Diazepam can worsen dysphoria. Because part of the syndrome is related to serotonin excess, cyproheptadine has been used to manage some of the CNS effects. If tachycardia persists, propranolol may be used. Signs may last up to 48-72 hrs in severe cases.

Marijuana
Over the past few years there has been an increase in the number of marijuana intoxicated pets presented to veterinary clinics. It is unknown if this is truly an increase in cases, if people are more willing to seek veterinary care due to changing attitudes about marijuana or if more potent forms of marijuana are prompting pet owners to seek medical attention.

Marijuana (Cannabis sativa) is used both recreationally and medicinally by people. It is thought to be the most commonly used illegal substance worldwide, with nearly half the population in the United States reporting at least one time use. Marijuana has been used as an anti-emetic, analgesic, anticonvulsant, muscle relaxant, appetite stimulant and to decrease intra-ocular pressure in glaucoma. Currently in the United States, there are 33 states that legally allow cannabis for medical use and eleven states plus the District of Columbia, Guam and Puerto Rico, that have legalized small amount of cannabis for recreational use by adults age 21 years of age and older. However, it is still a Schedule I controlled substance under the US Controlled Substances Act.

The main toxic principle of marijuana is a resin called tetrahydrocannabinol (THC), but the plant contains over 60 cannabinoids and cannabinals. The amount of these resins will vary with plant variety, sex of plant, "sensemilla" more toxic, geographic location, and growing season. THC acts via stimulation of cannabinoid receptors throughout the body. With inhalation, absorption of THC approaches 50% and clinical signs occur within 6-12 minutes, but GI absorption is erratic in humans and dogs. Blood concentrations obtained by ingestion are 25-30% of those obtained by smoking in humans. Onset of clinical signs is usually delayed at least 30 minutes and up to several hours after ingestion. THC is highly lipid soluble and is rapidly distributed to the brain and other tissues. Within the brain, THC
accumulates in the neocortical, limbic, sensory and motor areas. The plasma half-life of THC is short because of the rapid tissue distribution.

Cannabinoid receptors found in the pain pathways of the brain and spinal cord mediate the analgesic effects. The antemetic properties are thought to be secondary to the effect of cannabinoid receptors within the central nervous system. d-9-THC also affects dopaminergic, cholinergic, noradrenergic, serotoninergic, and GABA sites. There are 2 main cannabinoid receptors, CB1 and CB2 that have been identified in rats, Guinea pigs, dogs, monkeys, pigs and humans. CB1 receptors, primarily found in the CNS, are associated with psychoactive effects, and peripheral CB2 receptors are associated with the immune system, responsible for the immunomodulatory effects of cannabinoids. CB1 receptors are located with lipid membranes of presynaptic neurons and coupled to G-proteins. They inhibit cAMP and stimulate mitogen-activated protein kinases to modulate control of ion channels, particularly voltage-activated calcium ion channels and potassium channels. This inhibits neurotransmitter release, both excitatory and inhibitory. The endogenous ligand for cannabinoid receptors, known as endocannabinoids, are derived from arachidonic acids and closely related to prostaglandins. CB2 receptors are absent in the CNS but found in the peripheral nervous system (PNS) and immune system where they play a part in inflammation and pain regulation.

THC is rapidly metabolized by the mixed-function oxidase system in the liver. The significant first-pass effect likely accounts for the lower blood concentrations associated with ingestion versus inhalation. Between 65 and 90% of a dose of THC is excreted as the parent compound or conjugated active metabolites through the feces. In the past, most pet exposures to marijuana were ingestions of plant material from baggies or joints. This has changed and now edibles (cookies, brownies, etc.) and concentrates (oils, waxes, shatters) have become more popular. Through selective breeding, THC levels have become higher than ever. The University of Mississippi Potency Monitoring Project has reported that THC levels have more than doubled over the last 25 years. THC levels in plant material ranges from 1-8%, extracts 28%, and hash oil up to 50%. Another change has been the increase in marijuana butter based edibles. THC butter is made by heating marijuana in butter to extract the lipophilic THC. This butter is then used to make the baked goods. While both dogs and cats willing ingest plant material, dogs are the most likely to consume edibles. Many of the edibles also incorporate chocolate and this can increase the toxicity.

Another issue with THC containing products is quality control. In one study, 75 products were evaluated to determine the amount of cannabidiol and THC found in the various products. The results indicated that 17% of products were accurately labeled, 23% were under labeled and 60% were over labeled with respect to THC content.

The most common clinical signs after ingesting marijuana are ataxia, lethargy, and urinary incontinence. However, about 25% of patients may present stimulated instead. Hyperesthesia and disorientation are also frequently seen along with bradycardia, hypothermia, mydriasis, and tremors. Animals that get into concentrates or THC butter products may become comatose and hypotensive. Clinical signs can be seen as soon as 30 minutes after oral ingestion and may last up to 72 hours.

Urine drug screening tests have not been validated for use in dogs. Most over-the-counter urine drug tests will give a false negative result for marijuana (THC) in dog urine. This is thought to be due to different metabolites produced by dogs when compared to humans (8-OH-Δ²-THC produced by dogs vs 11-OH-Δ²-THC in humans). These different metabolites may also explain the urinary incontinence that is seen in dogs and not in other species.

As marijuana is an anti-emetic, inducing emesis may not be successful but can be tried with recent (< 30 minutes) oral exposures if the animal is asymptomatic. Activated charcoal is generally not needed. Intravenous fluid administration should be started and adjusted if dehydration or hypotension develops. Diazepam or low dose acepromazine (if normotensive) can be used for agitated patients. Monitor blood glucose levels in young animals. Many cases with plant material ingestion can be managed at home with confinement and monitoring the ability to ambulate.

For more symptomatic animals, monitor respiratory function, heart rate, blood pressure and body temperature. Keep animal warm, quiet, minimize sensory stimuli, and rotate body position q 4 hours if the animal is recumbent. Intralipids (20% solution) may be helpful for severely affected (comatose) animals because THC is lipid soluble but results have been variable. No specific CBC or chemistry profile abnormalities are expected. THC is highly protein bound (97% to 99%) and has a large volume of distribution (10 L/kg, with high lipophilicity), and thus dialysis or hemoperfusion have no theoretical benefit.
Toxicity is dose-related, however, there is a wide-range of variability among individuals. Patients with hepatic impairment may be more sensitive. A lethal dose has not been established in dogs or cats, but it only takes a small amount to cause clinical signs. Fortunately death is rare. There are published reports of two dog deaths after ingesting edibles and a 12-week-old ferret after ingesting plant material. If appropriate treatment is implemented, the prognosis is good and no permanent effects should be anticipated.

Bromethalin

Bromethalin is a neurotoxic rodenticide. It uncouples oxidative phosphorylation in CNS mitochondria. This results in lack of adequate ATP concentration and insufficient energy for maintaining Na⁺-K⁺ ion channel pumps. Loss of pump activity results in cerebral and spinal cord edema, retention of water in the myelin lamellae, separation of myelin lamellae, and demyelination injury to long nerves.

Bromethalin is rapidly absorbed from GI tract. Bromethalin is metabolized in the liver, by N-demethylation forming desmethylbromethalin. Cats are far more sensitive to this agent than are dogs. Dogs seem to have both a low-dose and a high-dose syndrome. Dogs under 16 weeks of age appear to be more at risk. With lower doses, signs may not appear for 72-96 hours, and include hind limb ataxia and paresis, decreased proprioception, loss of deep pain response, vocalizations, patella hyper-reflexia, CNS depression progressing to coma, vomiting, and fine muscle tremors. At or above the mean lethal dose, signs can begin within 12-24 hours and include severe tremors, hyperthermia, extreme hyperexcitability, running fits, hyperesthesia and seizures.

Treatment of clinical signs is directed to controlling cerebral edema, and is mostly frustrating and non-productive. Mannitol, corticosteroids and diazepam may be used. Continued swelling of the lamellae results in a dramatic increase in intracranial and cerebrospinal fluid (CSF) pressure that is typically unresponsive to therapy. On necropsy, spongy degeneration (diffuse vacuolation) of the white matter of the CNS is noted. Early decontamination is the best preventative care available. Animals with sub-lethal doses will require good nursing care.

5-Fluorouracil

5-Fluorouracil (5-FU) is in the antimetabolite class of antineoplastic agents. The topical creams and solutions (Efudex, Fluoroplex, Adrucil) are extremely toxic if ingested. 5-FU causes severe vomiting, seizures and bone marrow aplasia.

Fluorouracil is metabolized to thymidine. Thymidine blocks the methylation reaction of deoxyuridylic acid to thymidylic acid, creating a thymine deficiency. Lack of thymine interferes with DNA and (to a lesser extent) RNA synthesis and results in cell death. Rapidly growing cells, such as those in the bone marrow and intestinal crypts, absorb fluorouracil rapidly, resulting in severe GI upset and bone marrow aplasia. 5-FU also has several active metabolites, such as 5-fluorouridine-5′-triphosphate (FUTP). FUTP is slower to cross cell membranes than fluorouracil, resulting in delayed clearance in the bone marrow. Another metabolite, fluorocitrate inhibits the tricarboxylic acid cycle (TCA cycle), which blocks the gamma-aminobutyric acid (GABA) shunt and causes low levels of GABA in the brain, leading to seizures. Fluorouracil and its metabolites also result in laminar splitting of the myelin sheaths in nerve cells, leading to vacuolization of the myelin.

The onset of clinical signs usually occurs within 0.5 to 5 hours following ingestion. 5-FU rapidly distributes to the total body water and it is absorbed by all cells. In dogs that survived, signs lasted from 18 hours to 14 days. The minimum lethal oral dose for the dog is 20 mg/kg, but signs of toxicosis are seen as low as 8.6 mg/kg. Often signs begin with vomiting (with or without blood) and progress to tremors and seizures within a few hours. The vomiting isn’t always seen before seizures, nor are seizures seen in every case. Seizures may require care for more than 24 hours. If given enough time (5-20 days) and if the animal survives, 5-FU can destroy bone marrow stem cells resulting in leukopenia which can progress to a pancytopenia.

Emesis, activated charcoal and cathartic can be started if the animal is asymptomatic and the ingestion was recent (less than 1 hour). Hemodialysis and peritoneal dialysis enhance elimination. Seizures and tremors are rarely controlled with diazepam. Levetiracetum, barbiturates, gas anesthetics (isoflurane), and propofol have been used successfully. GI protectants and antiemetics should be started. IV fluids, thermoregulation, antibiotics, and pain control are very important parts of the therapy. If animals live through the severe vomiting and seizures, WBC's could start to decline in 5-20 days.
Filgrastim (Neupogen) may be given for neutropenia (5-6 mg/kg SQ). Prognosis is guarded to poor once signs occur. Sixty-four percent of dogs ingesting 5-FU die or are euthanized.

Note: Visturonidine (uridine triacetate) is used in human medicine as an antidote. However, it is not used in veterinary medicine due to expense (~$74,000) and the fact that it has to be administered before CNS signs are seen. Under the orphan drug laws has to be special ordered, which typically takes 2-3 days. No known cases of its use have been reported in cats and dogs.

**Ethanol**

Ethanol is a CNS depressant. It enhances the inhibitory effects of gamma-amino butyric acid (GABA) at the GABA-A receptor and competitively inhibits the binding of glycine at the N-methyl-d-aspartate (NMDA) receptor (disrupts excitatory glutaminergic neurotransmission). Ethanol also releases other inhibitory neurotransmitters, such as dopamine and serotonin. While we mostly think of ethanol (ethyl alcohol) as an alcoholic beverage, it is also found in other places: liquid medications, cosmetics, hand sanitizers, perfumes, colognes, mouthwashes, food flavorings (eg, vanilla extract), and fermenting yeast bread dough. The amount of ethanol needed to cause intoxication will vary depending on the type of alcohol ingested (see table 3). For alcoholic beverages, the proof is double the percentage of alcohol.

There are no published minimum lethal oral doses of ethanol in dogs.

All alcohols are rapidly absorbed orally; dermal absorption can also occur. Inhalation, particularly of concentrated fumes in a confined area, can also cause systemic signs. Peak plasma levels occur 30 minutes to 2 hours post ingestion, but can be delayed after larger doses or in the presence of food. Ethanol is metabolized in the liver (95%) by ADH (alcohol dehydrogenase enzyme) to acetaldehyde and then to acetic acid. The elimination half-life is not meaningful due to saturation of the metabolizing enzymes. In animals, most recover within 12-24 hours. Signs develop rapidly, often within 30-60 minutes, and include vomiting, ataxia, tremors, hypothermia, hypoglycemia, acidosis, aspiration pneumonia, respiratory depression, and coma.

Alcohol is directly irritating to the stomach and causes vomiting. High ethanol blood levels will also stimulate emesis. The concern with vomiting while intoxicated is that at high blood ethanol concentrations the muscles that control the epiglottis become slow to react or even paralyzed. This increases the risk of aspiration. Ethanol intoxication reduces peripheral oxygen delivery and metabolism and causes mitochondrial oxidative dysfunction, potentially resulting in shock or hypoxia in the acutely intoxicated patient. Hypothermia is secondary to peripheral vasodilation, CNS depression, ethanol interference with the thermoregulatory mechanism, and/or impaired behavioral responses to a cold environment.

Due to rapid onset of signs, decontamination should be performed only within the first 30 minutes following ingestion. Activated charcoal is not indicated as it binds poorly to ethanol. Also as ethanol toxicity is characterized by vomiting, the risk of charcoal aspiration is high. Bathe the animal if a dermal exposure has occurred. Heart rate, blood pressure, ECG and body temperature should all be monitored. Monitor for acidosis and hypoglycemia. If dietary and hepatic (glycogenolysis) sources of glucose are exhausted, hypoglycemia may result. During the oxidation of ethanol, there is an increase in the NADH/NAD ratio, which creates an increase in the conversion of pyruvate to lactate. The lack of the key intermediate, pyruvate, halts gluconeogenesis, and hypoglycemia ensues.

Intravenous fluids do not accelerate ethanol clearance in intoxicated patients, but isotonic solutions should be started for supportive purposes. Sodium bicarbonate should be added to combat metabolic acidosis. Any arrhythmias should be treated symptomatically (atropine for bradycardia, lidocaine for VPCs). Seizures, not related to hypoglycemia, can be controlled with diazepam. Pass an endotracheal tube and position the patient to prevent aspiration in a comatose animal. In severe intoxications, monitor oxygen saturation and be prepared to mechanically support the animal's breathing. Yohimbine, atipamazole or naloxone can be tried to reverse severe CNS depression or coma. This effect does not appear to be predictable or consistent in animals. In severe cases, hemodialysis may be considered. Hemodialysis can eliminate ethanol approximately 3 to 4 times more rapidly than liver metabolism.

While ethanol levels may be determined from blood, serum, plasma, and urine, they are seldom performed in pets. In humans, blood ethanol concentrations between 150 and 300 mg/dL (32.6 to 65.2 mmol/L) will generally cause obvious signs and symptoms. Death is usually associated with blood ethanol levels greater than 400 mg/dL (86.8 mmol/L). If you are unable to get a blood ethanol level, serum or plasma osmolality may be used to estimate blood ethanol levels. Blood alcohol content: BAL
(g/L) = osmolal gap/27. In most cases of ethanol ingestion, the prognosis is good. Cases complicated by aspiration of gastric contents, co-ingestants, or preexisting disease have a more guarded prognosis. Intoxicated animals are also predisposed to traumatic injuries.

Table 1. CNS stimulants in dogs and cats

| Pharmaceuticals                          | 5-fluorouracil          | 5-HTP          |
|                                         | Amphetamines            | Antihistamines |
|                                         | Cocaine                 | Decongestants  |
|                                         | Isoniazid               | MAOIs (monamine oxidase inhibitors) |
|                                         | Metronidazole           | Novel antidepressants |
|                                         | SNRIs (serotonin and norepinephrine reuptake inhibitors) | |
|                                         | SSRIs (selective serotonin reuptake inhibitors) | |
|                                         | TCAs (tricyclic antidepressants) | |
| Pesticides                              | 4-aminopyridine         | Anticholinesterases (organophosphates, carbamates) |
|                                         | Bromethalin             | DEET           |
|                                         | Metaldehyde             | Pyrethrins     |
|                                         | Sodium fluoride (1080)  | Strychnine     |
| Metals                                  | Lead                    | Sodium         |
| Mycotoxins                               | Tremorgenic mycotoxins  |                |
| Plants                                  | Brunfelsia sp.          | Milkweed       |
|                                         | Water hemlock           |                |
| Other                                   | Alpha lipoic acid       | Methylxanthines (caffeine, chocolate) |

Table 2. CNS depressants in dogs and cats

<p>| Pharmaceuticals                          | Antihistamines          | Baclofen         |
|                                         | Barbiturates            | Benzodiazepines  |
|                                         | Bromide                 | dl-methionine    |
|                                         | Opioids                 | Progesterone     |
| Household/Industrial                    | Alcohols (methanol, ethanol, isopropyl, ethylene glycol, propylene glycol) | Essential oils |
|                                         |                         | Turpentine       |
| Pesticides                              | Amitraz                 | Avermectins (ivermectin, moxidectin, selamectin, milbemycin) |</p>
<table>
<thead>
<tr>
<th></th>
<th>Bromethalin</th>
<th>Imidacloprid</th>
<th>Piperazine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metals</td>
<td>Lead</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plants</td>
<td>Marijuana</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>Coral snakes</td>
<td>Any liver toxin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mohave toxin pit vipers</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Isoxazole mushrooms</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thiaminase (raw fish)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Ethanol concentrations in alcoholic beverages and other household products.

<table>
<thead>
<tr>
<th></th>
<th>Proof</th>
<th>Percent ethanol v/v</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light Beer</td>
<td>5-7</td>
<td>2.5-3.5</td>
</tr>
<tr>
<td>Beer</td>
<td>8-12</td>
<td>4-6</td>
</tr>
<tr>
<td>Ale</td>
<td>10-16</td>
<td>5-8</td>
</tr>
<tr>
<td>Wine</td>
<td>20-40</td>
<td>10-20</td>
</tr>
<tr>
<td>Mouthwash</td>
<td></td>
<td>14-27</td>
</tr>
<tr>
<td>Amaretto</td>
<td>34-56</td>
<td>17-28</td>
</tr>
<tr>
<td>Aftershave</td>
<td></td>
<td>19-90</td>
</tr>
<tr>
<td>Schnapps</td>
<td>40-100</td>
<td>20-50</td>
</tr>
<tr>
<td>Coffee Liqueurs</td>
<td>42-53</td>
<td>21-26.5</td>
</tr>
<tr>
<td>Brandy</td>
<td>70-80</td>
<td>35-40</td>
</tr>
<tr>
<td>Bourbon</td>
<td>80-90</td>
<td>40-45</td>
</tr>
<tr>
<td>Rum</td>
<td>80-82</td>
<td>40-41</td>
</tr>
<tr>
<td>Cognac</td>
<td>80-82</td>
<td>40-41</td>
</tr>
<tr>
<td>Vodka</td>
<td>80-82</td>
<td>40-41</td>
</tr>
<tr>
<td>Whiskey</td>
<td>80-90</td>
<td>40-45</td>
</tr>
<tr>
<td>Tequila</td>
<td>80-92</td>
<td>40-46</td>
</tr>
<tr>
<td>Gin</td>
<td>80-94</td>
<td>40-47</td>
</tr>
<tr>
<td>Cologne/perfume</td>
<td></td>
<td>50</td>
</tr>
<tr>
<td>Hand Sanitizers</td>
<td></td>
<td>60-95</td>
</tr>
</tbody>
</table>
Pyrethrins
Pyrethrins are common insecticides. The low concentration products rarely cause much more than GI upset or drooling (the exception is bifenthrin in dogs). The concentrated products, especially when used on cats can cause tremors and seizures by binding to the sodium channels on nerves, causing them to remain open and repetitively fire.

Permethrin is a synthetic type I pyrethrin. Permethrin is found in shampoos, dips, foggars, spot-ons, and sprays. Permethrins appear to be relatively safe in dogs. Smaller dogs seem to have a greater risk of toxicity and skin hypersensitivity reactions to the spot-ons. Skin reactions can be treated with bathing +/- antihistamines or steroids. Cats are more sensitive to the toxicity of pyrethroids. The low concentration products (sprays, foggars) contain 0.05-0.1% of permethrin and do not seem to cause the signs that the concentrated (45-65% permethrin) spot-ons do. Permethrin toxicity usually occurs when the owner applies the dog product to the cat; however, cats which actively groom or engage in close physical contact with recently treated dogs may also be at risk of toxic exposure. Clinical signs of permethrin toxicity in cats include hypersalivation, depression, muscle tremors, vomiting, anorexia, seizures, and possibly death. Onset of clinical signs is usually within a few hours of exposure but may be delayed up to 24 hours. The severity of clinical signs varies with each individual. Treatment recommendations include bathing with liquid dish washing detergent and controlling the tremors. Methocarbamol works best to control the tremors. If no injectable methocarbamol is available, the oral form may be dissolved in water and given rectally. If the cat is actively seizuring, barbiturates or inhalent anesthesia may need to be used. Permethrins appear to have no direct action on the liver or kidneys, but fluids may be needed to help protect kidneys from myoglobin break-down products in actively tremoring cats. Prognosis for mildly tremoring cats is usually good, but treatment may last 24-48 hours.

Bifenthrin is commonly found in lawn insecticides. It will cause the same clinical picture in dogs that permethrin causes in cats.

Avermectins
Avermectins include ivermectin, milbemycin, selamectin, doramectin, abamectin and moxidectin. In nematodes and arthropods, avermectins bind to glutamate-gated chloride channels causing hyperpolarization by enhancing the movement of chloride ions into the cell. This results in paralysis. In mammals, avermectins cause CNS effects by potentiating the release and binding of GABA in the central nervous system. Doses of ivermectin and moxidectin in heartworm medications are safe for even MDR1 (ABCB1) deficient dogs (Collie-type breeds, Australian Shepherds, etc). Problems arise when owners are giving large amounts to treat dermatologic disorders or give the equine product to their pets. In general, young animals are considered more sensitive to the effect of avermectins due to a less developed blood brain barrier. Ivermectin is well absorbed orally and the half life in the non-sensitive dog is as long as 2-3 days. Enterohepatic recirculation is suspected based on the long half life and extent of fecal excretion (98%) of ivermectin. With the ‘non-sensitive’ breeds of dogs signs may be seen at 2000 mcg/kg, but only 150 mcg/kg is needed in the ‘sensitive’ breeds to cause signs. Cats have demonstrated clinical signs at the "therapeutic dose" of 200 mcg/kg. Moxidectin is a semi-synthetic avermectin that is much more lipid soluble than ivermectin. Therapeutic levels of moxidectin have been measured 30 minutes post oral exposure. Moxidectin has a wide margin of safety in dogs when given orally. Doses of up to 300 times the therapeutic dose (300 mcg/kg) resulted in little to no side effects. Most problems are encountered when dogs ingest horse dewormer.

The most common clinical signs of avermectin toxicosis include: depression, weakness, recumbency, ataxia, and coma. Other reported signs include tremors, seizures, transient blindness, bradycardia, and hyperthermia. If the exposure has just occurred and the animal is asymptomatic induce vomiting (if an oral overdose) or consider surgical debridement if given SQ and can localize injection site in massive overdoses. If the animal is symptomatic, treatment is mostly supportive care and repeated dosages of activated charcoal. Activated charcoal/cathartic should be given q 8-12 hours (sorbitol 70% - cathartic of choice) until normal. Intralipids can be given, however efficacy is greater with moxidectin due to its higher lipid solubility. Treatment can take days to several weeks. Supportive care is very important (fluids, parenteral nutrition, frequent turning, etc.). Physostigmine can be given, but it is not an antidote.
Physostigmine has a very short beneficial effect (arousal for 30-90 minutes) and should only be used in severely non-responsive dogs (not recommended for cats). The recommended dose is 0.05 mg/kg IM or IV (very slow, over 5 minutes). Prognosis depends on the speed of onset of clinical signs, the faster the onset, the worse the prognosis.

**Spinosad**

Spinosad is a tetracyclic macrolides anti-parasitic. It can cause vomiting and ataxia, however, if spinosad is given in conjunction with high dose ivermectin, avermectin toxicosis can develop.

**Essential oils**

Essential oils have been used for flea control. D-limonene is a derivative of citrus pulp. This essential oil has minimal to moderate efficacy to control fleas. If diluted properly, this product has a high margin of safety. Application of the undiluted product can cause skin and oral irritation, lethargy, vomiting, salivation, ataxia and muscle tremors. Essential oils can penetrate the skin and cause peripheral vasodilatation leading to hypotension and hypothermia. Melaleuca oil is an essential oil from the Australian tea-tree, *Melaleuca alternifolia*. It does have antibacterial and antifungal properties but the efficacy of this agent to repel or kill fleas has not been established. Inappropriate application of products not intended for topical use may result in ataxia, weakness, tremors and depression. Pennyroyal oil is derived from the leaves and flowers of the pennyroyal, squaw mint, or mosquito plants. Pennyroyal oil contains a volatile compound called pulegone, which is responsible for the toxic effects of the plants. The effectiveness of pennyroyal oil to kill fleas is unknown; however, toxicity has been reported. Exposure to pennyroyal oil may induce depression, vomiting, hepatic necrosis, diarrhea, epistaxis, seizures, and death.

Toxicity is dose-related and the possibility of severe signs is more likely if the pure oil is applied to the pet. Cats appear to be more sensitive than dogs to any of the essential oils. Treatment recommendations include bathing with liquid dish washing detergent, activated charcoal with cathartic, pain control if needed, body temperature regulation and fluids. Most essential oils have long half lives (days) due to enterohepatic recirculation.

**Baclofen**

Baclofen is a centrally acting skeletal muscle relaxant that mimics γ-aminobutyric acid (GABA) within the spinal cord and causes a flaccid paralysis of skeletal muscles. At oral therapeutic levels, baclofen has virtually no CNS effects due to its poor ability to cross the blood brain barrier, but in overdose situations, CNS effects are common. The most common clinical signs of toxicosis are vomiting, ataxia and vocalization/disorientation, but the most life threatening signs are dyspnea, respiratory arrest and seizures. Dyspnea and respiratory arrest are secondary to paralysis of the diaphragm and intercostal muscles.

The onset of clinical signs varies in dogs with signs occurring anywhere from 15 minutes to 7 hours post exposure (average of 1.9 hr). Duration of clinical signs vary from several hours to several days. Signs can continue long after serum baclofen levels have returned to normal due to the slow clearance from the CNS. Dog doses as low as 1.3 mg/kg can cause vomiting, depression and vocalizing. There are no established lethal doses in animals, but per the APCC data base, deaths in dogs have occurred at doses as low as 8 mg/kg.

Due to the rapid onset of clinical signs, emesis should be considered in only the asymptomatic, recently exposed patient. Gastric lavage may be considered with large ingestions, but care must be taken to ensure that anesthesia does not compound CNS depression. Short acting induction agents such as propofol followed by inhalent anesthesia with a protected airway is preferred. All asymptomatic cases should receive activated charcoal with a cathartic. Avoid magnesium-based cathartics (Epsom salts), as they may worsen CNS depression. Exposed animals should be monitored for 12 hours for development of clinical signs.

Ventilatory support is a prime concern and endotracheal intubation and positive pressure mechanical ventilatory support may be needed for an extended time in severe cases. Diazepam is the drug of choice for centrally acting skeletal muscle relaxant induced seizures. Propofol or isoflurane may be considered in cases that are refractory to diazepam. Long acting barbiturates or other agents that produce profound or prolonged CNS depression should be used with care. Cyproheptadine (1.1 mg/kg PO or rectally) has been used successfully to reduce the vocalization/disorientation seen in some animals. Fluid diuresis is used to enhance elimination and maintain blood pressure. Intralipids have been
used successfully in early intoxications. The use of CNS respiratory stimulants are of questionable value and experimental studies have failed to consistently produce positive outcomes when flumazenil was used and have potential to cause serious adverse effects (seizures). Prognosis is variable, and can depend on the availability of ventilatory support for depressed patients. Prognosis is more guarded if seizures develop.

**Opioids and Opiates**

There are many opioids and opiates used in human and veterinary medicine. Opioids and opiates are synthetic or natural compounds derived from the opium poppy, *Papaver somniferum*, and are generally classified (agonist or partial agonist) by their ability to exert effects at the different opioid receptors (mu, kappa, delta, sigma). Partial agonists are agonists at one (or more receptors) and antagonists at others. Opioids act centrally to elevate the pain threshold and to alter the psychological response to pain. Most of the clinically used opioids exert effect at the mu receptor (mu₁ subtype mediates analgesic effects, mu₂ mediates respiratory depression).

Opioids are well absorbed from the GI tract, but bioavailability is variable as some opioids have a large first pass effect (i.e. fentanyl). These opioids are administered in other manners (CRI, buccal, transdermal) to reach therapeutic blood levels. Metabolism varies, but opioids generally undergo hepatic metabolism (conjugation, hydrolysis, oxidation, glucuronidation, or dealkylation). This glucuronidation may account for the sensitivity of cats (who are deficient in glucuronyl-S-transferase) to opioids.

In dogs, CNS signs include depression, ataxia, and seizures. Respiratory depression, vomiting, bradycardia, and hypotension may be seen. Cats may show excitatory behavior and urinary retention. Detection of opioids can be made from urine or serum samples.

Treatment in an asymptomatic animal may include emesis if the ingestion is recent. Activated charcoal with cathartic should be administered and the patient monitored for up to 12 hours. If the animal becomes symptomatic, naloxone (0.1-0.2 mg/kg IV, IN, IM or SQ) can be administered. As the duration of action of naloxone is much shorter than that of the opioids, repeat dosages may be necessary. Partial agonists/antagonists (i.e. butorphanol) may be used to partially reverse pure agonists if no naloxone is available. Monitor temperature, cardiac function and blood gases. Treatment times will vary with the half life of the opioid. If respiratory and cardiovascular function can be maintained then prognosis is good. For those cases that are seizuring, prognosis is guarded.

<table>
<thead>
<tr>
<th>DRUG</th>
<th>ACTIVITY</th>
<th>Mu</th>
<th>Delta</th>
<th>Kappa₁</th>
<th>Kappa₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>Agonist</td>
<td>+++</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Agonist</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Butorphanol</td>
<td>Agonist/antagonist</td>
<td>P</td>
<td>NA</td>
<td>+++</td>
<td>NA</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>Agonist/antagonist</td>
<td>P</td>
<td>NA</td>
<td>--</td>
<td>NA</td>
</tr>
<tr>
<td>Naloxone</td>
<td>Antagonist</td>
<td>---</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>

*(P = partial)*

**Fentanyl**: Fentanyl suckers, lozenges and transdermal patches are becoming more frequently used in both human and veterinary medicine. The lozenges or suckers contain fentanyl citrate in a sucrose and liquid glucose base and are attractive to animals. The patches have poor absorption from the GI tract, but can be absorbed transmucosally while the animals are chewing on them. Signs are similar to other opioids with depression, bradycardia, hypotension, weakness, and pallor predominating. Treatment is as for other opioids.

**Zinc Phosphide**

Zinc phosphide is an old rodenticide posing as a new one. The phosphide salts are unstable in an acid environment. At gastric pH they degrade rapidly to form phosphine gas. Phosphine gas, when inhaled, results in acute non-cardiogenic pulmonary edema. When absorbed systemically, it is thought to block cytochrome C oxidase, leading to formation of highly reactive oxygen compounds. It is these reactive compounds which cause most of the tissue injury, most severe damage is in tissues with the highest oxygen demand – brain, lungs, liver and kidney.

Lethal doses for dogs and cats range between 20-50 mg/kg. For a 55 pound (25 kg) dog, that would be between 10 grams (0.35 ounce) and 25 grams (just under an ounce) of 5% bait. Severely
Poisoned animals may die in 3-5 hours. Those who survive longer than 48 hours have a pretty good chance.

Initial signs may vary by species, as well as by the dose. Onset of signs is normally between 15 minutes to 4 hours post ingestion. Vomiting, often with blood, is common. Dogs may show lateral recumbency with whole body tremors and salivation. Other signs may include anorexia and lethargy. Rapid deep breathing may signal the onset of the pulmonary changes. Abdominal pain, ataxia, and weakness leading to recumbency may follow. Hyperesthesia and seizures may develop that resemble the signs of strychnine toxocosis.

Metabolic acidemia ensues. Other biochemical changes may include depressed serum calcium and magnesium. If there is survival beyond 48 hours an elevated blood urea is common. Hepatic and renal damage often may be detected 5-14 days later.

Initial decontamination is tempered by the wish to keep the stomach pH as high as possible to prevent the formation of phosphine gas. If there has been no spontaneous vomiting, it may be better to induce emesis with apomorphine rather than hydrogen peroxide. Giving food, commonly done in order to improve gastric emptying and the response to peroxide, will trigger release of gastric acid and increase the rate of production of phosphine. If you are going to perform gastric lavage, add an alkalizing component like a magnesium and aluminum hydroxide gel to your lavage liquid. Also consider mixing into your activated charcoal preparation.

Supportive care includes IV fluids to maintain blood pressure renal perfusion, and gastroprotectants. Seizures may respond to diazepam, or may require barbiturates or full anesthesia. Since the most severe injury is probably due to action of the oxygen radicals, use of an antioxidant may be useful – consider vitamin C or n-acetylcysteine.

Caution: Phosphine gas released from vomitus or stomach washings can cause significant illness in veterinary personnel assisting animal. Phosphine has been describes as having a spoiled fish or garlic odor. It is detectable at 1-3 ppm in air; maximum allowed in air in occupational situations is 0.3 ppm, so if you can smell it, you are being exposed to a concentration that can be harmful.

**Chocolate**

There are a wide variety of chocolate and cocoa products to which pets may be exposed, including candies, cakes, cookies, brownies, and cocoa bean mulches. The active (toxic) agents in chocolate are methylxanthines, specifically theobromine and caffeine. Methylxanthines stimulate the CNS, act on the kidney to stimulate diuresis, and increase the contractility of cardiac and skeletal muscle. The relative amounts of theobromine and caffeine will vary with the form of the chocolate (see table).

The LD50’s of theobromine and caffeine are 100-300 mg/kg, but severe and life threatening clinical signs may be seen at levels far below these doses. Mild signs have been seen with theobromine levels of 20 mg/kg, moderate signs have been seen at 40-50 mg/kg, and seizures have occurred at 60 mg/kg. Clinical signs occur within 6-12 hours of ingestion. Initial signs include polydipsia, bloating, vomiting, diarrhea, and restlessness. Signs progress to hyperactivity, polyuria, ataxia, tremors, seizures, tachycardia, PVC’s, tachypnea, cyanosis, hypertension, hyperthermia, and coma. Death is generally due to cardiac arrhythmias or respiratory failure. Because of the high fat content of many chocolate products, pancreatitis is a potential sequela.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Theobromine</th>
<th>Caffeine</th>
</tr>
</thead>
<tbody>
<tr>
<td>White Chocolate</td>
<td>0.25</td>
<td>0.85</td>
</tr>
<tr>
<td>Milk Chocolate</td>
<td>58</td>
<td>6</td>
</tr>
<tr>
<td>Semi-sweet Chocolate chips</td>
<td>138</td>
<td>22</td>
</tr>
<tr>
<td>Baker’s Chocolate (unsweetened)</td>
<td>393</td>
<td>47</td>
</tr>
<tr>
<td>Dry cocoa powder</td>
<td>737</td>
<td>70</td>
</tr>
</tbody>
</table>
Management of chocolate ingestion includes decontamination via emesis. Activated charcoal may be given in some instances. Intravenous fluids at twice maintenance levels will help maintain diuresis and enhance urinary excretion. Because caffeine can be reabsorbed from the bladder, placement of a urinary catheter is recommended. Cardiac status should be monitored via EKG and arrhythmias treated as needed; propranolol reportedly delays renal excretion of methylxanthines, so metoprolol is the beta-blocker of choice. Seizures may be controlled with diazepam or a barbiturate. In severe cases, clinical signs may persist up to 72 hours.

Moldy Food (Tremorgenic mycotoxins)

Tremorgenic mycotoxins produced by molds on foods are a relatively common, and possibly under-diagnosed, cause of tremors and seizures in pet animals. Because of their relatively indiscriminate appetites, dogs tend to be most commonly exposed to tremorgens. These toxins are produced from a variety of fungi that grow on practically any food, including dairy products, grains, nuts, and legumes; compost piles may also provide a source of tremorgens.

Clinical signs include fine muscle tremors that may rapidly progress to more severe tremors and seizures. Death generally occurs in the first 2 to 4 hours and is usually secondary to respiratory compromise, metabolic acidosis or hyperthermia. Other signs that may be seen include vomiting (common) hyperactivity, depression, coma, behavior alterations, tachycardia, and pulmonary edema.

Asymptomatic animals exposed to moldy foods should be decontaminated via emesis or lavage followed by activated charcoal and cathartic. In symptomatic animals, control of severe tremors or seizures has priority over decontamination. Seizures may respond to diazepam, tremors respond best to methocarbamol (55-220 mg/kg IV to effect). Supportive care should include intravenous fluids, thermoregulation, and correction of electrolyte and acid-base abnormalities. In severe cases, signs may persist for several days, and residual fine muscle tremors may take a week or more to fully resolve.
The liver is the largest organ in the body and serves many vital functions. The liver metabolizes almost every drug or toxin introduced into the body. There are various mechanisms of hepatic injury: disruption of the hepatocyte, disruption of the transport proteins, cytolytic T-cell activation, apoptosis of hepatocytes, mitochondrial disruption, bile duct injury, chemical induced hepatic damage, direct or intrinsic or predictable drug reactions, indirect or unpredictable idiosyncratic drug reactions. The liver can respond to toxic insults with hepatic lipidosis, hepatocellular necrosis (centrilobular, midzonal, periportal, massive), nodular regeneration, pigmentation, megalocytosis, biliary system damage, neoplasia and liver atrophy. Necrosis is the predominant form of cell death in most toxic insults. Degenerative changes including ballooning degeneration, hyaline degeneration and the presence of Mallory bodies, may precede necrosis. Cells lose osmotic homeostasis and swell. Energy production fails due to loss of calcium homeostasis. Cellular membranes rupture and leak cell contents, including cytosolic enzymes such as alanine transaminase and sorbitol dehydrogenase and mitochondrial enzymes like aspartate transaminase. Depending on the extent of liver necrosis, overall liver function may or may not be affected. Serum glucose levels and coagulation status should be monitored very closely. See table 1 for a list of hepatotoxicants in dogs and cats.

**Xylitol**

Xylitol is a sugar alcohol used as a natural sweetener. It is sourced from corn cobs or birch trees. Xylitol is used in sugar-free products from gums, candies, pudding, high protein peanut butter, toothpaste, quick dissolve and chewable medications, as well as for baking. In humans, it doesn’t cause significant increases in blood glucose or insulin and helps prevent dental caries. However, in dogs, xylitol causes a rapid, dose-dependent insulin release (2.5-7x that of glucose) followed by potentially significant hypoglycemia. Signs can include vomiting, weakness, ataxia, depression, hypokalemia, seizures, and coma. Some dogs have developed liver dysfunction or failure following ingestion of xylitol although the mechanism has not been established. The working theory is that xylitol uses the pentose phosphate pathway (PPP) instead of the TCA (Kreb’s cycle) for metabolism. Studies in both rats and guinea pigs have shown this to be true, but there have been no studies performed in dogs. The use of the PPP means that energy is channeled into reducing potential not ATP production. Hypoglycemia can start at 100 mg/kg and liver effects around 400 mg/kg.

Treatment of xylitol ingestion by dogs should include emesis if asymptomatic. Dogs can show signs of hypoglycemia in as few as 30 minutes or it may be delayed for several hours. Activated charcoal is not efficacious for decontamination. If clinical signs of hypoglycemia develop, dextrose should be given (bolus +/- CRI). Hypokalemia, likely secondary to insulin-induced movement of potassium into cells, should be treated if significant. Treatment should continue until blood glucose normalizes. Dextrose decreases the need for gluconeogenesis and may be liver supportive. SAMe (precursor for glutathione, methyl donor) and n-acetylcysteine (antioxidant, precursor of l-cysteine that increases glutathione production) may be started but it is unknown how efficacious they are. With liver necrosis, monitoring of coagulation values and plasma transfusions may be needed.

**Iron**

Iron is an essential part of oxygen delivery to tissues (hemoglobin, myoglobin), enzymatic processes, and oxidative metabolism within the body. Accidental ingestion of iron supplements, molluscsicides, and hand-warmers may cause corrosive gastroenteritis and hepatic injury.

Iron absorption from the gastrointestinal tract is highly regulated by the body. Typically only 5-15% of ingested iron is absorbed out of the GI tract. In the small intestine, iron is absorbed by enterocytes in the ferrous (Fe2+) form and transferred to the serum where it is converted to the ferric (Fe3+) form. Serum iron is primarily bound to transferrin with lesser amounts bound to ferritin. Iron uptake from the liver sinusoids is receptor mediated and sequestration occurs through binding with iron storage proteins. In the liver, iron is either utilized or stored in small amounts as ferritin or as in larger amounts as hemosiderin. Ferritin and hemosiderin are protective in that they keep cellular iron in a bound form. High hepatic free iron concentrations initiate intracellular oxidative damage through electron donation leading to reactive oxygen species (ROS) affecting zone 1 hepatocytes. The generation of ROS
in excess of cellular oxidant defenses results in lipid peroxidation of membranes, protein and DNA cross linking and cell death. Hepatic damage may be massive and severe, as in the case of a large acute iron overdose, or it may be chronic and fulminating, resulting in cirrhosis of the liver over time.

Clinical signs of acute iron toxicosis include bloody vomiting and/or diarrhea, abdominal pain, weakness, shock, collapse and death. Animals that survive may subsequently develop signs of acute hepatic failure. Dyspnea and exercise intolerance may be seen if cardiac injury is present. Elevated liver enzymes occur within 24-48 hours. A high serum iron, can aid in determining whether iron toxicosis is likely. Due to the limited ability of the body to excrete iron, treatment of acute iron toxicosis entails stabilizing the animal (oxygen, blood replacement as needed) and possible chelation with deferoxamine.

**APAP**

Acetaminophen (Tylenol®, non-aspirin pain reliever, APAP) is a synthetic non-opiate derivative of p-aminophenol. It is available in tablets (80-650 mg) and liquid preparations. APAP acts primarily in the CNS to increase the pain threshold and may also inhibit chemical mediators that sensitize the pain receptors to mechanical or chemical stimulation. The antipyretic activity of APAP is achieved by blocking the effects of endogenous pyrogens by inhibiting prostaglandin synthesis. There is some thought that the KCS rarely seen in dogs is due to inhibition of COX-3.

Acetaminophen is rapidly and almost completely absorbed from the GI tract. Peak plasma levels are seen at 10-60 minutes (60-120 min for extended release). APAP is distributed into most body tissues with the highest concentrations in the peri-portal zone of the liver and the renal medulla. The metabolism of acetaminophen occurs primarily in the liver. There are three major pathways: direct glucuronide conjugation, direct sulfate conjugation, and oxidation mediated by cytochrome P450 enzymes.

Due to APAPs available hydroxyl group, immediate phase II conjugation is the primary route of metabolism in most species, and involves glucuronide and sulfate. Cats unfortunately have only about one tenth the acetaminophen biotransformation ability of dogs due to decreased microsomal UDP-glucuronosyltransferase enzyme activity. In dogs and humans, glucuronide conjugation accounts for 50-60% of a dose of acetaminophen. In cats, after an oral dose of 20, 60 and 120 mg/kg acetaminophen, only 1%, 5% and 16% undergo glucuronide conjugation, respectively.

Sulfate conjugation is less important than glucuronide conjugation in the metabolism of acetaminophen in most species aside from cats. Dogs metabolize only about 10-20% of a given dose of APAP via sulfate conjugation. After cats were dosed with 20, 60 and 120 mg acetaminophen/kg, 92%, 78% and 57% of the acetaminophen underwent sulfate conjugation, respectively. The sulfate conjugation pathway can be saturated due to the limited availability of inorganic sulfates.

Acetaminophen-induced hepatotoxicity and nephrotoxicity is due to the formation of the oxidative metabolite, N-acetyl-para-benzoquinoneimine (NAPQI). Cytochrome P450 oxidation increases as phase II pathways become saturated. In dogs, approximately 5% of a dose of acetaminophen normally undergoes oxidation by cytochrome P450 in dogs. When cats are dosed with 20 mg/kg acetaminophen, 5% undergoes oxidation, but this number increases to 10% at doses of 60-120 mg/kg. The reaction that produces NAPQI also generates superoxide anions as a by-product. NAPQI itself also acts as an electrophile, targeting mitochondria in particular, forming covalent adducts with protein thiol groups and other cellular macromolecules. Interaction of NAPQI with other cellular molecules generates more ROS, leading to oxidative stress. Adenine nucleotides and plasma membrane proteins involved in calcium homeostasis are also targeted. Cytoskeletal damage/activation of endonucleases and DNA fragmentation are proposed causes of cell death. Zone 3 (centrilobular) hepatocyte degeneration is most common due to the concentration of P450s in that area. The role of Kupffer cell activation has also been implicated as contribution to acetaminophen-induced liver injury through the production of reactive nitrogen species. If glutathione (GSH) is available, NAPQI will conjugate with reduced GSH, forming an inactive product. GSH stores become depleted 16-24 hours after exposure to acetaminophen, increasing the risk of cellular damage.

A second byproduct of acetaminophen metabolism, via phase 1 deacetylation, is para-aminophenol (PAP), it is implicated in the hematotoxic effects seen in cats and dogs. PAP is rapidly conjugated to GSH or acetate in laboratory rodents, but cats have reduced capacity for n-acetylation and dogs lack the hepatic n-acetyltransferase enzyme. GSH becomes depleted in erythrocytes, and hemoglobin is oxidized to methemoglobin, which cannot carry oxygen. Methemoglobin values increase within 2-4 hours, followed by Heinz body formation.
Clinical signs include depression, weakness, hyperventilation, icterus, vomiting, chocolate colored gums, hypothermia, facial or paw edema, death, cyanosis, dyspnea, and hepatic necrosis. Liver necrosis is less common in cats than in dogs. Clinical signs of methemoglobinemia may last 3-4 days. Hepatic injury may not resolve for several weeks.

Early decontamination is most beneficial. Activated charcoal adsorbs APAP. Monitor liver values and for the presence of methemoglobinemia. ALT, AST and bilirubin may rise within 24 hours after ingestion and peak within 48 to 72 hours. Symptomatic patients need initial stabilization, including oxygen if dyspneic. Antidotal therapy involves the use of sulfate sources to bind the active metabolites and enhance glutathione production.

N-acetylcysteine (Mucomyst®, NAC) is a precursor in the synthesis of glutathione, provides sulfhydryl groups to be used for phase II sulfate conjugation or for glutathione production, and can be oxidized to organic sulfate which provides sulfhydryl groups that bind with APAP metabolites to enhance elimination. NAC also decreases half-life of methemoglobinemia in cats from 10 to 5 hours. An initial loading dose of 140 mg/kg (dilute to 5% in dextrose or sterile water) is given, followed by 70 mg/kg PO QID for 7 treatments. Continue treatment until normal. Ascorbic acid provides a slow-acting, nonenzymatic reduction of methemoglobin back to hemoglobin. For hepatic injury, s-adenosylmethionine (SAMe, Denosyl-SD4®) at 20 mg/kg/day shows a positive effect for treatment of APAP toxicosis. The use of SAMe for prevention of methemoglobinemia in cats has been inconsistent. Prognosis is good if the animal is treated promptly. Animals with severe signs of methemoglobinemia or with hepatic damage have poor to guarded prognosis.

Blue-green Algae
Accumulation of large amounts of blue-green algae (cyanobacteria) can be found in many lakes, ponds and rivers. Macroscopically they appear as a “scum” on top of the water. Toxic blooms occur following warm, sunny weather and are seen more frequently in ponds that get runoff from heavily fertilized fields or from feed lots or pastures bearing significant numbers of animals. The most important toxin-producing genera of fresh and brackish water blue-green algae include Oscillatoria, Anabaena, and Nodularia. The primary toxic effects of blue-green algae in animals include acute hepatotoxicoses, peracute neurotoxicoses, and gastrointestinal disturbances. Oscillatoria, Anabaena, and less often Nodularia may produce cyclic heptapeptide hepatotoxins called microcystins. There are more than 100 variants of microcystin. When ingested, the acidic environment of the stomach can result in the release of microcystins from the algae. Hepatocytes are the target of microcystin, which enters the cell through a bile-acid transporter. Microcystins cause hepatic damage by several different mechanisms. Microcystin covalently binds to serine/threonine protein phosphatase, leading to the hyperphosphorylation of cytoskeletal proteins. The deformation and loss of function of the cytoskeleton produces cholestasis by preventing the normal pulsatile contraction that moves bile through the canalicular system to the bile ducts. Microcystins cause severe hepatomegaly and progressive centrilobular hepatocyte rounding, dissociation and necrosis. Microcystins also induce apoptosis of hepatocytes via induction of free radical formation and mitochondrial alteration. Breakdown of the sinusoidal endothelium and intrahepatic hemorrhage ultimately result in death.

Clinical signs in animals include weakness, stupor, prolonged capillary refill time, pallor of mucous membranes, bloody diarrhea, and cardiovascular collapse. Clinical signs are usually observed within 12 hours after exposure. Death may occur within a few hours to a few days. Death often is preceded by coma, muscle tremors, and seizures. Death usually results from intrahepatic hemorrhage and hypovolemic shock. For recent exposures, decontamination measures such as emesis and activated charcoal may be useful. Monitor liver function for 48 hours. Treat symptomatically with fluids, anticonvulsants, and blood transfusions as needed. Many animals will die before receiving any treatment.

Amatoxins
Amanita, Galerina and Lepiota sp. mushrooms have been associated with hepatic dysfunction. These mushrooms have a wide distribution throughout the U.S.. They contain three different groups of cyclopeptides: the amatoxins, phallotoxins, and virotoxins. Amatoxins are bicyclic octapeptides and include the amantitins (α-, β-, γ-, and ε-amantitins), amanin, amanullin, and proamanullin. Severe mushroom poisonings and lethality are mainly attributable to the amantitins. The bicyclic heptapeptides phallotoxins were once thought to be the cause of gastrointestinal signs; however, they are no longer
believed to exert any acute toxicity. They can cause signs when injected, but not by the oral route. Virotoxins are also not considered to have toxic effects after oral exposure.

Amanitins inhibit nuclear RNA polymerase II, interfering with DNA and RNA transcription, thus inhibiting ribosomal protein synthesis. Cells with high metabolic rates (hepatocytes, intestinal crypt cells) are most sensitive. Humans are the most susceptible, then dogs. α-amanitin is taken up by cells in the GI tract, where the first effects are seen. Following systemic absorption, α-amanitin is taken up into hepatocytes via OATP1B3, an organic anion-transporting polypeptide. The toxins bind tightly to actin filaments and preventing cytoskeletal disassembly, causing deformation and loss of cytoskeletal function. The decrease in mRNA and associated decrease in protein synthesis result in hepatocyte necrosis.

There are three phases in amatoxicosis. A latent phase of approximately 6-24 hours is followed by a GI phase with abdominal pain, vomiting and diarrhea (lasts 2-3 days). The hepatic phase begins 36-48 hours after ingestion. Jaundice progresses to coma, coagulopathies and anuria. Many humans require liver transplantation. UC Davis offers a urine or serum test for amatoxins, but turn-around time may make this only useful for confirmation.

Emesis and activated charcoal is recommended in witnessed exposures. Because amatoxins are cleared from the serum in the first 24 to 48 hours either by the kidneys or via uptake into hepatocytes, extracorporeal elimination techniques are not likely to be useful. Monitor serum glucose, liver and renal values, acid/base, electrolytes, PCV/TS, and coagulation parameters. Significant amounts of amatoxin are eliminated in the urine during the first 48 hours. Therefore, high urine output should be achieved over the first 48 hours. Silymarin (milk thistle) is used extensively in Europe, but is not available in the U.S. (yet). Silibinin reduces the uptake of amanitins into hepatocytes. Historical treatment has been with acetylcysteine (APAP treatment dosages), penicillin G (interferes with enterohepatic recirculation of amatoxins) and cimetidine. Ultrasound-guided percutaneous bile drainage has been used successfully in a handful of cases. Prognosis is guarded and hepatic injury may be permanent.

Aspirin

Aspirin (acetylsalicylic acid, ASA) is the salicylate ester of acetic acid and is a weak acid derived from phenol. It is available as tablets and capsules (65, 81, 325, and 500 mg), powders, effervescent tablets and oral liquid preparations. Aspirin reduces pain and inflammation by reducing prostaglandin and thromboxane synthesis through inhibition of cyclooxygenase. At very high doses, aspirin and other salicylates uncouple oxidative phosphorylation leading to decreased ATP production. Salicylates also affect platelet aggregation. Aspirin is rapidly absorbed from the stomach and proximal small intestines in monogastrics. Aspirin is metabolized in the liver and excreted through the urine. Cats are deficient in glucuronyl transferase and have prolonged excretion due to decreased metabolism. Elimination is also slower in neonates and geriatric animals.

Signs may include vomiting (+/− blood), hyperpnea, respiratory alkalosis, metabolic acidosis, gastric hemorrhage, central lobular liver necrosis, and bleeding diathesis. Fever and seizures may be seen due to the uncoupling of oxidative phosphorylation. In dogs, single doses over 150 mg/kg can cause GI ulcers, doses over 400 mg/kg liver effects, seizures and acidosis.

Emesis can be performed in the asymptomatic animal, unless contraindicated. Activated charcoal adsorbs aspirin and repeated doses may be used with large ingestions. Liver values, glucose, acid base status and electrolytes should be monitored. Maintain hydration and start GI protectants. Gastric protectants should be continued for 5-7 days, longer in the symptomatic patient. Alkalization of the urine results in ion trapping of salicylate in the kidney tubule and increases its secretion. Ion trapping should only be used in cases where the acid base balance can be monitored. Assisted ventilation and supplemental oxygen may be required if the animal is comatose. Seizures should be treated with diazepam. Fluids and whole blood may be needed to control hypotension and hemorrhage. Hyperpyrexia can be treated conservatively. Prognosis is good if the animal is treated promptly and appropriately. The development of hepatic necrosis is considered to have a poor prognosis. With hepatic damage, treatment may need to be continued for weeks.

NSAIDs

Hepatotoxicity can be seen with any NSAID and is thought to be an immune mediated reaction. Most cases are reversible with supportive care.
Sago palms

Sago palms (*Cycas* and *Macrozamia* sp.) are ornamental plants found in tropical to subtropical climates, but they can also be grown as houseplants. There are three toxins in cycads: cycasin (hepatic necrosis, GI hemorrhage), B-methlamino-L-alanine (neurotoxin) and another unidentified neurotoxin. The seeds contain the highest amount of cycasin, but the entire plant is toxic. Cycasin causes centrolobular and midzonal coagulative hepatic necrosis along with GI hemorrhage. GI signs begin within a day and laboratory values (ALT, bilirubin, Alk Phos) become abnormal in 24-48 hrs. The most common signs are vomiting (+/- blood), depression, diarrhea, anorexia, and seizures. **Decontamination is emesis, followed by activated charcoal.** Monitor liver enzymes for 48 hours, or until levels return to normal. Blood or plasma transfusions may be necessary if coagulopathies develop. Prognosis is good if caught early, but guarded in cases where the animal is already showing signs. Mortality rate is about 30%.

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<th>Table 1. Hepatotoxicants in Dogs and Cats</th>
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<td>Arsenical antihelmintics</td>
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<td>Sulfonamides</td>
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<td>Household/Industrial</td>
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<td>Microcystin (blue-green algae)</td>
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Myths and misconceptions in veterinary toxicology
Tina Wismer DVM, MS, DABVT, DABT
ASPCA Animal Poison Control Center, Urbana, IL

Myths, or ‘old wives tales’ have always been prevalent in veterinary medicine. We hear these commonly in practice (I know he’s sick ‘cause his nose is warm. My dogs are brother and sister, they would never have puppies together.). However, with the rise of the internet, rumors and misinformation can spread like wildfire.

Internet rumors are probably the most modern form of folklore (handed-down beliefs, stories, and customs). These stories are written to be as believable as possible, and often contain precautionary advice on how to avoid harm to your pet. These tales also tend to evolve in time due to embellishment and repetition; internet rumors in particular have a way of being resurrected months or years after the initial distribution, often with adjustments made to make them more plausible.

Milk is the universal antidote
Many times owners will call after a pet has ingested something it shouldn’t have and they will tell you they have already given milk. Milk can help to dilute caustic substances (acids, alkalis) and irritants (detergents), along with dissolving the insoluble calcium oxalate crystals found in some plants. The calcium in milk may also decrease the absorption of certain pharmaceuticals (bisphosphonates, tetracyclines). Unfortunately, milk does not treat all toxins.

Atropine is the universal antidote
Atropine can be used to reverse the muscarinic signs (salivation, lacrimation, urination, dyspnea, drooling, emesis) from organophosphate and carbamate insecticides. Atropine however, does not treat all toxins. This misconception probably arises from many years ago when most insecticides were organophosphates or carbamates. Today, these compounds are rarely encountered.

Burnt toast is the universal antidote
This myth arises from the use of activated charcoal in poisonings. Unfortunately, the black bits off of burnt bread are not absorbents like activated charcoal.

Weak tea is the universal antidote
Tea does contain tannins, which can be helpful in treatment of some toxicants (rhododendrons, azaleas), but it does nothing to treat other poisons.

Febreze kills pets
When Febreze first came out on the market in 1999 there was an extensive internet email rumor that implicated it in the deaths of many dogs. The email occasionally is sent around again. Febreze can cause respiratory issues in birds and allergic reactions in some dogs, but it has not been linked to any dog deaths.

Swiffer wet jet kills pets
Another internet rumor started in May of 2004, which stated “Swiffer wet jet contains a compound which is ‘one molecule away’ from antifreeze and caused liver failure and death in a German shepherd dog.” Nothing in the ingredients of the Swiffer liquid poses risk of hepatotoxicity. If antifreeze or a closely related glycol were involved, we would expect renal, not liver damage. Any molecule is ‘one molecule away’ from antifreeze.

Ultra Clorox bleach kills pets
Per the internet, Ultra Clorox bleach poses danger to pets and should not be used in households with pets because it contains sodium hydroxide, which is “LYE,” which is not present in ‘regular’ bleaches. The truth is that all bleaches contain lye (sodium hydroxide) and compared to other bleaches, Ultra Clorox does not pose additional hazard to pets when used as directed.

Almonds are poisonous to pets
Per the myth, almonds contain cyanide and will kill your pet. This myth does have some truth behind it. Bitter almonds do contain cyanide. However, we eat sweet almonds which contain no cyanide. Bitter almond essential oil may be purchased but because it has been processed, there is no poisoning risk.

**Pistachios are poisonous to pets**
This myth is all over the internet if you google ‘pistachios and dogs.’ There is no basis to this myth. Nuts can certainly cause GI upset but are not considered poisonous.

**Chocolate gives dogs worms**
Per this misconception, you shouldn’t give your dog chocolate, not because it is poisonous, but because it will give them worms. This myth may have resulted from the fact that dogs who eat chocolate may vomit or have diarrhea, and the roundworms (already present in the dog) may be seen in the vomit or stool.

**If you give your dog water after it eats mouse poison it will explode**
Per the pest control operator, ‘This poison causes the mice to leave the house looking for water and once they drink, they explode.’ This is not true. None of the rodenticides cause mice to explode. This myth is perpetuated as the pest control operator doesn’t want you to know that the mice are dying inside the walls of your house.

**Rimadyl (or insert pharmaceutical name here) kills pets**
If you look on the internet, any drug used in veterinary medicine is blamed for multiple problems.

**Pot scrubbing sponges manufactured by Proctor & Gamble contain dangerous derivative of agent orange**
The myth is that an owner cleaned out aquarium with a new sponge and when he placed the tropical fish back into aquarium, they died. He concluded that the Pot scrubbing sponges manufactured by Procter & Gamble contain a dangerous “derivative of 2-4-D, more popularly known as Agent Orange” that can kill pets. This rumor is wrong on many levels. First of all, Proctor & Gamble doesn’t make sponges. 2,4-D is not Agent Orange, it is a safe herbicide. Most likely the fish died after being replaced in the aquarium following cleaning and not completely rinsing out of aquarium, or destruction of the biological filter.

**Tennis balls can explode and kill a dog**
This myth is unfortunately true. In 2000 a dog in Portland, OR picked up a tennis ball during a walk and it exploded killing the dog. The ball had been made into a bomb. Per the Portland police, tennis ball bombs are not uncommon and information on how to make them can be found on the internet. They caution people to leave found tennis balls alone, especially if they are wrapped in electrical or duct tape.

**Tennis balls contain lead**
Tennis balls themselves do not contain lead, but there have been inks used to print logos on the balls that have tested high in lead.

**Paper towel tubes contain zinc**
This myth states that the glue used in the cardboard found in the middle of paper towel rolls and toilet paper contains enough zinc to poison an animal. This is untrue.

**Dogs die after eating children’s stuffed toys containing flame retardant materials.**
Per the circulating email; "The dog ate a child's teddy bear and was very sick. When the vet opened the dog up to remove what she thought was an intestinal obstruction she found a huge gelatin type mess inside and the dog's intestines were black and the tissue dead. The dog will die no surgery can fix him up there was no living intestine left from stomach to colon. This was not an obstruction, so the vet called the manufacturer of the Teddy Bear on a quest to find out what the gel was and what killed the dog. Turns out the stuffing in children's toys contains ingredients for flame retardants and mite control! It
Rawhide manufactured overseas is poisonous to dogs

“Rawhides from overseas can kill dogs.” There is some truth to this, but it is somewhat of a misconception. There was one batch of rawhides from Thailand that were contaminated with arsenic, but this was many years ago. However, rawhide, no matter the source, can be contaminated with salmonella or other bacterial toxins.

To prevent heartworm in dogs, once a year you give two copper pennies by mouth

Per this myth, the copper in the pennies is attracted to the heart and will kill the worms. Pennies cause zinc toxicity so this is a very dangerous practice.

Ice cubes will cause your dog to bloat on a hot day

Internet rumor that a person gave their dog ice to cool off on a hot day and the dog developed bloat and died. Dogs do not bloat from drinking ice water on hot days.

Garlic is a natural way to get rid of fleas

Wanting to use a safe flea control on pets is understandable, but owners may equate natural with safe, which is not always the case. The simple answer is no, garlic is not efficacious for treating fleas. Garlic can however cause hemolytic anemia and methemoglobinemia. For dogs, 10 g/kg of fresh garlic (or its equivalent) may be a problem.

Ingesting mouse bait will cause an animal to seek water, and when the animal drinks the water it will activate the poison

Water is not needed to activate mouse poison. The three most common types of bait in the United States (anticoagulants, bromethalin, cholecalciferol) all work on their own when ingested. This myth likely came about because no one wants to think that mice may die inside their home. Hence the pest control officer tells people the mice leave the house to find water.

Hostas make bubbles & bloat in a pet’s stomach

The theory is that since Hostas contain saponins, and saponins are used to make soap, if a pet ingests the plants they will make soap bubbles in the pet’s stomach. That, according to the rumors, leads to bloat. It’s true that saponins are used to make soap, but it’s not true that soap or soap bubbles are produced in a pet’s stomach if plants containing saponins are ingested. Pets will commonly vomit after ingesting Hostas.

Cats and dogs are small fuzzy people

Drugs that people take without any problems (Aleve, acetaminophen) can cause serious problems in pets. However, many human medications (levothyroxine, benzodiazepines) are tolerated at much higher doses in animals.

Many factors come into play when assessing domestic dog and cat exposures to various toxins. For years veterinarians have witnessed wide inter-individual variation in response to drug therapy. Some patients respond well, others fail to respond, and still others experience idiosyncratic toxicity to routine doses of a particular drug. The same things can be noted when we try to predict animal responses to human medications.

Dogs make up the largest percentage of calls to the ASPCA Animal Poison Control Center. This is due to many reasons. Dogs are low to the ground, they investigate with their mouths and they have indiscriminate eating habits. Cats have more selective eating habits than dogs, however, their fastidious grooming habits make almost all dermal exposures into oral exposures also.

Metabolic processes have evolved over time to allow individual species to handle various components of their diets. Animals that are ‘true carnivores’ (e.g. cats) have a more restricted diet and have evolved fewer biotransformation pathways than those with more diverse diets (e.g. herbivores, omnivores). This can be a problem when animals encounter a xenobiotic that requires a biotransformation pathway they do not possess. The N-acetylation pathway is used for the metabolism of sulfonamides, procainamide, dapsone, isoniazid, and hydralazine. This pathway is absent in all dogs due to a loss of both N-acetyltransferase genes. Cats are ‘defective’ in glucuronidation (pseudogene vs
functional gene) so they cannot glucuronidate phenols, naphthols, and morphine. This makes cats highly susceptible to xenobiotics that require glucuronidation for metabolism (acetaminophen, aspirin, etc).

Dogs and cats are more sensitive to RBC oxidative damage than humans (4 vs 8 vs 2 sulfhydryl groups on hemoglobin, respectively). Our pets can easily develop Heinz bodies and methemoglobinemia when exposed to oxidative agents such as aniline dyes, onions/garlic, acetaminophen, and benzocaine.

As dogs and cats are carnivores, they tend to have acidic urine. This can influence the rate of elimination of xenobiotics. Dogs poorly excrete organic acids which increases susceptibility to phenoxy herbicides.

Genetic differences exist not only between people, dogs and cats, but also among different breeds of dogs and cats. Purebred patients represent distinct gene pools and breed specific variations in drug response are recognized. A few CYP pathways have been shown to be polymorphic in dogs. CYP2D15 metabolizes the COX-2 selective NSAID celecoxib in dogs. The half-life varies from 1.5 hr for extensive metabolizers to 5 hours for poor metabolizers. CYP2B11 is the enzyme responsible for propofol metabolism. Its activity varies at least 14-fold in mixed breed dogs, while greyhounds have particularly low activity. This corresponds to reduced clearance of propofol, higher blood concentrations and delayed recovery in greyhounds when compared to mixed breeds.

Some breeds of dogs are deficient in a P glycoprotein (MDR-1 or ABCB1) that keep xenobiotics out of the brain. This is an autosomal recessive trait in some collies, Australian Shepherds, and others and confers an increased sensitivity to avermectins (antiparasitics), loperamide (antidiarrheal), acepromazine (sedative), butorphanol (analgesic) and vincristine, vinblastine, and doxorubicin (chemotherapy agents). "Normal" therapeutic doses of these medications have an exaggerated effect in these animals.

Aging rates vary with breed and age can play a role in the sensitivity to xenobiotics. Young animals tend to have a more permeable GI tract and blood brain barrier (BBB), decreased GI motility, lower levels of metabolic enzymes, lower glomerular filtration rates (GFR), higher caloric requirements/intake and have an increased risk from fat soluble compounds (milk diets, yolk sac). Comparatively, aged animals tend to have decreased GFR, decreased metabolic activity, concurrent degenerative/disease processes and decreased GI motility. Pre-existing diseases can alter intestinal or dermal barriers, alter plasma protein status, affect RBC numbers, decrease function of liver, kidney, other organs and alter the BBB.

Especially in dogs, there is a wide variation in size and body type. For example, sighthounds have a low percentage of body fat. These dogs may also have an increased sensitivity to organophosphorous pesticides and barbiturates. With just these few examples of genetic differences, it is easy to see that managing toxicoses in different species can be challenging.
Ten decontamination techniques: The old and the new
Tina Wismer, DVM, MS, DABVT, DABT
ASPCA Animal Poison Control Center, Urbana, IL

The practitioner is occasionally presented with a situation where it is suspected, either by the owner or the veterinarian, that an unidentified “poison” has intoxicated the patient. Although identification of the agent involved is often extremely helpful in determining proper treatment and prognosis, it is important to remember that the majority of these types of cases are managed without the offending agent ever being identified. Just because the identity of the toxicant remains a mystery does not mean that the veterinarian cannot deliver appropriate and effective treatment. Whether the toxic agent is known or not, it should always be remembered that the goal of the practitioner is to “treat the patient, not the poison.”

ASSESS THE PATIENT

Upon initial examination, evaluation of the animal for immediate life-threatening problems such as seizures, apnea/dyspnea, hemorrhage, cardiac arrhythmias, and hyperthermia is essential. A brief history may be obtained from the client while the examination is taking place—more detailed history may be obtained after the animal is stabilized. Additional important information that should be obtained includes duration of signs, age and prior health status of the animal, and any initial signs that are no longer apparent.

STABILIZE THE PATIENT

Stabilization is a priority in animals presenting with severe clinical signs. Animals should be intubated and/or provided with supplemental oxygen as needed. If possible, obtain venous access and draw blood for laboratory profile and potential diagnostic testing (3 cc EDTA tube, 2 serum tubes are ideal), prior to administration of other medications. Standard anticonvulsants such as diazepam or barbiturates may be used to control seizures. Anticonvulsants, particularly benzodiazepines, should be administered slowly IV, as rapid administration may induce a dysphoric effect and temporarily exacerbate the situation. If the standard anticonvulsant therapy does not have any effect, consider inhalant anesthesia or a propofol CRI to allow for initial management of the patient.

Life-threatening cardiac arrhythmias should be treated as needed (atropine, propranolol, or lidocaine prn); arrhythmias not deemed immediately life-threatening can be treated after a better history has been obtained. Intravenous fluids and blood or blood replacement agents should be administered as needed. Body temperature should be normalized as needed, however aggressive cooling measures should be undertaken with care. Any electrolyte or acid/base abnormalities should be corrected. Once the patient has been fully stabilized, a more comprehensive physical examination may be performed.

OBTAIN HISTORY

Once the animal is stable, further questioning of the owner should be performed in an attempt to narrow down the possible causes for the animal’s signs. Questions to consider include how long since the last time the animal appeared normal to the owner, whether the onset of signs was gradual or sudden, the location of the animal in the last few hours prior to the development of clinical signs, and any history of administration of medications/herbal products/flea or tick control products to this animal or other animals in the household in the past 24 hours. The type of environment in which the animal lives (e.g. indoor only vs. indoor/outdoor vs. fenced yard vs. roaming) will help to determine the next lines of questions to ask.

For indoor animals, information that may be useful includes the areas to which the animal has access, the types of medications/herbal products (human and veterinary, prescription, illicit and OTC) available, whether there have been recent visitors who may have dropped medication, the types of houseplants in the home, whether there are children or teenagers in the household, presence of rodenticides or insecticides, and whether other pets in the house appear normal. In cases where illicit drugs are involved, or where owners have inappropriately administered medications or other products to their pets, the veterinarian may notice some reluctance on the part of the owner to provide the requested information. Tactful questioning may aid in obtaining the desired information. In other cases, it may be helpful to mention that without knowledge of the agent involved, more intensive (and expensive) diagnostics and treatments may be necessary.
For outdoor animals confined by fences or other means, identification of potentially toxic agents in outbuildings, garages or sheds to which the pet may have access is important. Other potential hazards found in yards include compost piles, plants, mushrooms, and yard treatments (especially some systemic insecticides and crabgrass killers). For free roaming animals, the challenge is much greater as the number of potentially toxic agents available is quite large. Determining whether the animal is in an urban, suburban, or rural environment and identifying the nature of the animal’s immediate surroundings (e.g. wooded areas vs. parks and lawns) may help in narrowing down the agents to which the roaming animal may have been exposed. The presence of livestock in the pet’s environment should stimulate questioning to determine the pet’s access to the barns or feed bins, whether medicated feeds, fly baits or feeds with growth promotants in them are present, whether the livestock have recently been medicated or dewormed, and if any livestock have recently been euthanized and buried on the property.

FORMULATE RULE-OUT LIST
Armed with a thorough physical examination and as much history as is obtainable, the clinician should then formulate a list of differential diagnoses. It is important not to become so caught up in the certainty that the causative agent is a poison that one loses sight of potential etiologies of infectious, metabolic or other “non-toxic” origin. For instance, although a variety of toxicants may cause acute onset of seizures, other potential etiologies to consider include encephalitis, idiopathic epilepsy, hypoglycemia, head trauma, hypoxia, hepatic failure, acid/base abnormalities, etc.

ANCILLARY SUPPORT
General supportive care includes maintaining hydration, ensuring adequate urine output, monitoring of respiratory, cardiac and neurologic status, and managing clinical signs as they develop. Recumbent or comatose animals require careful monitoring and thermoregulation. Gastrointestinal protectants or anti-emetics may be required (e.g. NSAID overdosages). Management of secondary hepatic or renal injury is imperative.

PREVENT TOXICANT ABSORPTION
Decontamination should be instituted only after the animal has been fully stabilized. If there could be possible legal action, seal with tape and initial/date sample. It is important to maintain records of chain of custody of samples (vomitus, carcass, etc.).

Ocular Exposure
Ocular exposures may cause irritation or corrosion of the ocular tissues depending on the substance, the concentration, the exposure time and the sensitivity of the patient. With any ocular exposure, the eyes should be flushed repeatedly with tepid water or saline solution for a minimum of 20-30 minutes. An eyedropper may be used for smaller patients. With a larger patient, fill a plastic cup and slowly pour over the ocular area, or a medicinal syringe may be used. Patients may be given a mild sedative prior to flushing if needed and if the health of the patient will allow. If not sedated, the patient should be allowed to rest at regular intervals during the flushing to minimize stress. Fluorescein staining should be performed after flushing and repeated at 12 – 24 hours post-exposure to check for corneal ulceration. Treatment with lubricant ointments should follow staining, and topical medications applied as indicated.

Dermal Exposure
Dermal exposures may occur to a large variety of substances including petroleum products, pesticides and insecticides, corrosive or irritating materials and substances that are sticky (tar, asphalt, sap and glue). Removal of dermal substances may be less stressful if the patient is sedated. Sedatives should only be used if the health of the patient will allow. If not sedated, the patient should be allowed to rest at regular intervals during the bathing to minimize stress.

Bathing
For dogs and cats, bathing in a mild liquid dishwashing detergent (e.g. Dawn) and warm water is recommended. Baths may need to be repeated to completely remove the toxicant. Afterwards, the animal should be rinsed well with warm water and towel dried to prevent chilling. These patients should be kept in a warm environment away from drafts until completely dry. For smaller patients that resent
being sprayed with water, the bucket technique may be helpful. Fill a bucket with soapy warm water. Hold the patient, supporting the hind legs and dunk into the bucket up to the neck. Remove the patient and continue bathing. Use a fresh bucket with plain warm water to rinse well.

Dermal substances can be removed from very small animals such as birds, reptiles or rodents by misting with room temperature water in a warm environment. Misting should continue until the product can no longer be detected on the coat or feathers by odor or touch. If misting is insufficient at removing the product, a liquid dishwashing detergent (e.g. Dawn) should be diluted in the misting bottle and applied, making sure to avoid the eyes. After removal of the substance, the animal should be rinsed via misting with clear water until all soap is removed. With heavy exposures, the animals may be bathed with liquid dishwashing detergent and rinsed well, with care taken not to over-stress the animal. After misting or bathing, the animal should be wiped with a dry towel and kept in a warm environment away from drafts until completely dry.

**Sticky substances**

When dealing with sticky substances (e.g. gum, glue traps, tar, etc), the use of solvents should be avoided as solvents may cause dermal irritation or burns. Any remaining exposed sticky material should be covered with paper towels, baby powder or vegetable oil to avoid further entrapment of the animal with the agent. To remove sticky substances from mammals, trim the fur to remove as much of the substance as possible. Then work a small amount of vegetable oil, mineral oil, mayonnaise or peanut butter through the rest of the substance until it breaks down into "gummy balls". Afterwards, wash with liquid dishwashing detergent as described above. For birds, do not trim the feathers, just utilize vegetable oil, mineral oil mayonnaise or peanut butter and then mist as described above.

**Oral Exposure**

**Dilution**

Dilution with milk, water, or liquid from water-packed tuna fish is recommended in cases of ingestion of corrosive or irritant products, exposure to toad secretions, or taste reactions due to topically applied products (e.g. “foaming kittens” following flea spray application). Dilution with milk may also aid in relief of oral discomfort secondary to chewing on plants that contain insoluble calcium oxalates in their leaves (e.g. *Philodendron* spp.). For birds and reptiles, juicy fruits and vegetables can be fed to accomplish dilution.

**Emesis**

Emetics generally empty 40-60% of the stomach contents and are assumed to be more beneficial than gastric lavage. Dogs, cats, ferrets, and potbelly pigs are examples of animals that can vomit. Induction of emesis is contraindicated with ingestion of corrosive agents or hydrocarbons. Pre-existing conditions (e.g. seizure disorders, severe dyspnea) may also cause use of an emetic to be contraindicated. Emesis should not be attempted if the animal has already vomited or is exhibiting significant clinical signs. Potential complications from emesis induction may include aspiration, persistent gastritis, and transient bradycardia (due to vagal stimulation).

Emesis is most productive if performed within 2-3 hours post-ingestion. In some cases, such as ingestion of chocolate, large numbers of sugar-coated tablets, grain-based rodenticides, or plant material, emesis may be effective even after 2-3 hours due to formation of boluses of product in the stomach (chocolate, tablets) or delay in gastric emptying (grain-based products, plants). Feeding the animal a small meal prior to inducing vomiting can increase chances of an adequate emesis.

Three percent (3%) hydrogen peroxide is a preferred emetic, especially if emesis is to be induced at home by the owner. Peroxide is readily available (needs to be “fizzy,” not flat), easy to administer, and often highly effective, especially in dogs. The dosage is 1 teaspoon/5 lbs body weight, not to exceed 3 tablespoons. Vomiting usually occurs within 10-15 minutes and the dose can be repeated once if not initially successful. In the process of foaming (which triggers the vomiting) the peroxide is converted to water and oxygen, so if no vomiting occurs there is no concern about adverse effects from the retained peroxide. Overdosing with hydrogen peroxide should be avoided, as it may result in gastritis that may take days to resolve.

Apomorphine hydrochloride may also be utilized as an emetic in dogs. The recommended dosage is 0.04 mg/kg IV, SQ. Reversal of the CNS depression from apomorphine may be accomplished through the use of naloxone. An alternative route of administration is to instill apomorphine conjunctivally.
The eye should be rinsed well after the animal has vomited. Anecdotally, the latter method results in less CNS depression than injection. Xylazine or dexmedetomidine can be used as an emetic in cats. It will cause significant hypotension, bradycardia and CNS depression, but these effects can be reversed with yohimbine or atipamezole.

Other emetics have been used including salt, liquid dishwashing liquid, syrup of ipecac and powdered mustard. Salt that is not vomited up may result in hypernatremia, causing severe neurological derangements. Syrup of ipecac generally has a delay in onset of action of up to 40 minutes in dogs and if not vomited up can cause myocardial depression and hypotension; the FDA has withdrawn ipecac as an emetic for human use due to questions of efficacy and safety. Powdered mustard does not appear to be an effective emetic in dogs or cats.

Adsorbents

Activated charcoal adsorbs toxicants and facilitates excretion via the feces by capturing the toxicant molecules in its micro-porous matrix. Activated charcoal is available in powder, liquid, gel and capsule forms. Activated charcoal capsules are not uniformly broken down in the GI tract of animals, and many will pass through the digestive tract intact. For this reason, if capsules are to be given, they must be cut open and the charcoal from multiple capsules pooled and then mixed with liquid to be administered. Activated charcoal tablets used for breath freshening or “anti-gas” are not appropriate forms of charcoal for decontamination.

Activated charcoal is contraindicated in animals that have ingested caustic materials. Chemicals that are not effectively adsorbed by activated charcoal include ethanol, methanol, fertilizer, fluoride, petroleum distillates, most heavy metals, iodides, nitrates, nitrites, sodium chloride, and chloride.

The recommended dose of activated charcoal for all species of animals is 1-3 gms/kg body weight. Repeated doses of activated charcoal every 4-8 hours at half the original dose may be indicated when enterohepatic recirculation of the toxicant is known to occur, or if ingestion of sustained release medications has occurred. See precautions regarding electrolyte disturbances under Cathartics below.

Kaolin-Pectin (Kaopectate) has also been recommended as an adsorbent in some instances. Kaolin is a form of clay (hydrated aluminum silicate) and pectin is a purified carbohydrate derived from fruits. Unfortunately, many of these kaolin-pectin products have recently been reformulated to contain salicylates, which makes their use in small animals less desirable. Another clay, bentonite (colloidal hydrated silica) has been used historically, but in most instances activated charcoal is a superior absorbent to the clays.

Cathartics

Cathartics enhance elimination of substances, including activated charcoal, by moving them through the gastrointestinal tract. Without cathartics, the toxicant bound by activated charcoal can eventually be released and absorbed by the GI tract. Cathartics are not to be used if the animal has diarrhea or is dehydrated. There are saline, osmotic and bulk cathartics.

Caution: Saline and osmotic cathartics may result in electrolyte disturbances (most notably hypernatremia) if overdosed or used in small, dehydrated or debilitated animals. Occasionally, hypernatremia may develop in animals with no apparent predisposition. Animals developing tremors, fasciculation, disorientation or other neurologic signs within 1-3 hours of receiving activated charcoal should have their electrolytes evaluated.

Bulk cathartics increase stool volume. These are used to ease passage of packaging.

Osmotic cathartics, like sorbitol, pull electrolyte-free water into the gastrointestinal tract. Sorbitol is commonly combined with activated charcoal in prepared products. The dose is 3ml/kg. Osmotic cathartics can be utilized in mammals, birds and reptiles.

Saline cathartics include sodium sulfate (Glauber's salts) and magnesium sulfate (Epsom salts). Saline cathartics act by stimulating gastrointestinal motility. The dose is 250 mg/kg mixed in water or activated charcoal. Saline cathartics should not be used in birds or reptiles.

Enemas

Enemas can be helpful when elimination of toxicants from the lower gastrointestinal tract is desired. The general technique is to use plain warm water or slightly soapy warm water. Enemas are not recommended for birds. In reptiles, enemas may be useful since ingested materials often lag for prolonged periods in the colon.
**Lavage**

*Gastric lavage* is used in mammals to remove recently ingested toxicants. Gastric lavage should not be used to remove caustic substances or hydrocarbons. Rabbits have very thin stomach walls so use great caution when performing gastric lavage in this species. Gastric lavage is generally considered to be less effective than emesis in removing toxicants from the stomach, but may be indicated in cases where induction of emesis has been ineffective or is not possible (e.g. seizing animals). General anesthesia must be maintained when performing gastric lavage. A cuffed endotracheal tube should be in place to prevent aspiration. Body temperature water or physiologic saline (preferred for small patients) should be instilled via gastric tube at 10 ml/kg BWT. Use only gravity to instill and to drain the liquid, repeat until lavage fluid runs clear. Inclining the patient head-down at 20-degree angle will facilitate fluid removal, and use of large bore tubes and multiple flushes may yield better success. Body temperature should be monitored closely, as animals may become hypothermic even when body temperature water has been used. Potential complications from gastric lavage may include gastric or esophageal perforation, aspiration, and hypothermia.

*Enterogastric lavage* is sometimes recommended when potentially lethal oral exposure has occurred and there is need for evacuation of more than just the stomach. Gastric lavage is performed as directed above and an enema administered. A pre-anesthetic dose of atropine (0.02 mg/kg), if not already given for anesthesia and not contraindicated, may aid in intestinal relaxation and prevent abdominal distention. With the stomach tube left in place, the enema tube is attached to a water faucet and digital pressure around the rectal orifice is used to seal the tube in place. Low pressure, body temperature water is allowed to slowly fill the intestinal tract until water flows from the stomach tube; gently massaging the intestines may via abdominal palpation may enhance the water passage. Once the water from the stomach tube flows clear, the process is complete. Potential complications from this procedure include intestinal rupture, profound hypothermia, and gastroenteritis.

*Gastrotomy* may be indicated for agents that will not readily pass through the gastrointestinal tract on their own. This may include ingestion of pennies and iron supplements (both of which tend to adhere to gastric mucosa), raw yeast bread dough, expandable polyurethane wood glues, lead objects, and ingestion of large amounts of toxic plant material, pill vials or medication tubes.

*Crop lavage* is used in birds to remove recently ingested toxicants. Frightened and fractious birds should be anesthetized prior to crop lavage. An endotracheal tube should be placed to prevent aspiration. The crop should be flushed gently with warm saline and aspirated. This should be repeated 3 – 4 times. Crop lavage should not be performed in cases of caustic or petroleum distillate ingestion.

**Lipid therapy**

The use of lipid emulsions to treat toxicoses has really taken off in the past five years in both human and veterinary medicine. Lipid emulsions have been historically given intravenously to patients who were unable to get enough fat in their diet. In 1998, Weinberg et al demonstrated that an infusion of lipid emulsion protects against cardiac arrest secondary to a bupivacaine (local anesthetic) overdose in rats. This paper led to the idea that lipids may potentially be used to treat many other toxins.

The exact mechanism of action is unknown, but there are three theories. The first is the ‘lipid sink/lipid shuttle’ theory. In this theory lipophilic drugs are redistributed (bound) in the expanded plasma lipid volume and this decreases free (active) drug levels. The second theory is that because fatty acids can increase calcium concentrations in cardiomyocytes, lipids may cause an increase in inotropy that overcomes the depressive effects of the intoxication. The third theory is that local anesthetics inhibit carnitine acyltransferase, which is needed to transport fatty acids across myocardial mitochondrial membranes for oxidative phosphorylation. Lipids may overcome the decreased fatty acid transport via mass action or an unknown mechanism. All three of these theories may work for local anesthetic overdoses, but theory one is the prevailing thought for lipids working on non-local anesthetic intoxicants.

It is thought that after administration, the lipids are cleared from the blood by striated muscle, viscera, myocardium and subcutaneous tissues. The lipids are metabolized into free fatty acids and glycerol. What happens to the toxin is unknown.

The lipid emulsion of choice for treating toxicoses is the 20% solution. This may be given through a peripheral catheter (no central line is needed). Veterinary dosing guidelines are taken from the human literature and are considered extra-label. Dosing is a 1.5 mL/kg initial IV bolus. In humans this is given very quickly (over 1 minute) as many of these patients are in asystole. The bolus is followed by a
constant rate infusion (CRI) of 0.25 mL/kg/min for 30-60 minutes. The CRI can be repeated in 4-6 hours if no lipemia is present and the animal is still symptomatic. A total limit of 8 mL/kg/d has been suggested in the human literature.

In human medicine, use of lipids is reserved for severe toxicoses and life threatening conditions after conventional therapies have failed. In veterinary medicine as we have no 'standardized protocols' to follow lipids have been tried more frequently. The published human papers are mainly case reports. Lipids have been used successfully in local anesthetic (bupivacaine, mepivacaine, ropivacaine), beta-blockers (propranolol, carvedilol), calcium channel blockers (verapamil, amlodipine) bupropion, haloperidol, quetiapine, carbamazepine (anticonvulsant), hydrochloroquine (antimalarial), flecainide (class Ic antiarrhythmic), thiopentone and serotoninergic drug (sertraline, doxepin) overdoses. Theoretically, lipids will work best on more lipid soluble toxins. Studies in laboratory animals have confirmed the efficacy of lipid emulsions for treating local anesthetics, chlorpromazine, cyclosporine, clomipramine and verapamil. Papers published in the veterinary literature include using lipids to treat moxidectin toxicosis in a puppy, lidocaine intoxication in a cat and ivermectin toxicosis in a cat.

The use of lipids unfortunately has not been shown to be effective in all cases of lipophilic drug toxicosis. In the human literature there have been failures when treating verapamil, amlodipine and tricyclic antidepressants. The veterinary literature also has a case report where lipids were unhelpful in treating ivermectin toxicosis in a group of ABCB1–1 mutant status dogs (MDR-1). Anecdotally, using lipids to treat baclofen intoxication in dogs sometimes works well and other times has no effect. In both of these situations the toxin can easily cross the blood-brain barrier (drug itself or genetic defect). It is possible that once these drugs are in the CNS, lipids will not help.

Adverse effects of lipids are uncommon but have been reported. Bacterial contamination of the product can occur as the emulsion is nutrient rich. Make sure sterile technique is followed. A bag should only be used for 24 hours and then discarded. The unused portion between doses should be stored in the refrigerator and still discarded after 24 hours. Rarely, an animal can have a reaction to the emulsion, which can cause an anaphylactoid-like reaction within 20 minutes of administration. Allergic reactions to the egg or soybean oil content can also occur. Side effects can include lipemia, hypertriglyceridemia, immunosuppression (immune cell dysfunction), hemolysis (oxidative damage), phlebitis, thrombosis, and hepatic lipidosis. It is unknown if dogs have a higher risk of pancreatitis and seizures secondary to hypertriglyceridemia in these situations. Fat overload syndrome is delayed and is caused by excessive volumes or high administration rates overwhelming the endogenous lipid clearance mechanisms. Fat overload syndrome is associated with hyperlipidemia, fat embolism, hepatomegaly, splenomegaly, thrombocytopenia, jaundice, increased clotting times and hemolysis. Lipids can also bind antidotes or other therapies that are being used to treat the patient. This can cause a worsening of the clinical condition.

The use of lipids to treat intoxications appears to be a safe therapy, but more research is needed. If there is an effective therapy or antidote available, then the traditional therapy is recommended. If the toxin is lipid soluble and traditional therapies have failed or are too expensive, then using lipids is an option. The more lipophilic the toxin, the better the lipids may work. Some animals will experience complete resolution of their signs, while others may have no or minimal improvement. Appropriate supportive care is still needed in these patients. Fortunately, adverse events are rare.

Cholestyramine

Cholestyramine is an anion exchange resin available by prescription only. It is used to lower cholesterol in patients who have not responded to normal therapies. Cholestyramine has been used in human medicine to aid in the treatment of toxicoses (amiodarone, digitoxin, chloroquine, methotrexate, piroxicam, vitamin D, warfarin, blue-green algae, indomethacin). It binds with bile acids in the intestine, preventing their reabsorption. This stops enterohepatic recirculation. Cholestyramine is not absorbed out of the digestive tract, so it has no systemic effects, but constipation and mild liver enzyme elevation may be seen. The dose is 0.3 – 1 g/kg TID for several days (depends on toxin ingested). For our patients, the powder should be given or mixed with canned food. Cholestyramine is cost effective with a price around $50-80 for 240g.

"THE ANTIDOTE"

If, after stabilizing the animal and obtaining an adequate history, the toxic agent has been identified, specific antagonists may be indicated (e.g. Vitamin K for rodenticides). It is important to
remember that the vast majority of toxic agents have no specific antidote, so the treatment will be, by necessity, symptomatic and supportive. Even in cases where antidotes do exist for the specific toxicant, there are often barriers to their use in veterinary medicine, including high cost and lack of availability (e.g. pamidronate for cholecalciferol or calcipotriene toxicosis).

ANALYTIC TESTING

Unfortunately, there is no one test that will “screen” for all known toxicants, and multiple tests for specific agents can become costly. In general, one needs to have an idea of the general type of agent that may be involved before analytical testing is attempted. For suspected human medication ingestion, human hospitals may be willing to run tests for illicit drugs, antidepressants, cardiac drugs, acetaminophen, etc. on a STAT basis. Alternatively, there are now available in many human pharmacies, OTC home drug testing kits that might be considered; these bench-top tests, though technically not validated for non-humans, are quick, easy and cost-effective in cases of suspected exposure to certain human medications. For suspected rodenticide, insecticide, or heavy metal exposure, most veterinary diagnostic laboratories offer basic screens. Some diagnostic laboratories offer specialty screenings, such as “convulsant” screens that might detect agents such as bromethalin, tremorgenic mycotoxins, strychnine, etc. In many cases, the results from these tests may not be obtained for days, at which point the patient may be either recovered or dead. Therefore, the veterinarian should still be prepared to manage the case using appropriate symptomatic and supportive care.
There are many toxicants that can directly and indirectly affect the kidneys (see table 1). The kidney is susceptible to toxicants because of its high relative blood flow (~25% of cardiac output) compared with its low percentage of total body weight. Blood borne toxicants are delivered at the highest rates to cortical tissues, whereas the medullary and papillary regions are exposed to the highest luminal concentration of toxicants and for longer period of time. Different portions of the kidney vary in their susceptibility to toxicant damage. The proximal tubule is highly involved in active transport of substances and is the most sensitive portion the nephron to both hypoxia and toxicosis. The kidney also has enzymes for detoxification, some ability to regenerate and considerable reserve capacity.

The renal response to toxicosis is limited to only four categories: glomerular lesions, nephrosis, mineralization and papillary necrosis. Glomerular lesions are rarely reported with toxicosis (primarily associated with immune disease). Acute tubular necrosis (tubular nephrosis) is the most commonly recognized form of toxicant-induced renal damage. Mineralization or nephrocalcinosis results from excessive amounts of vitamin D or its analogs. Papillary necrosis, or ischemic necrosis, occurs secondary to inhibition of prostaglandin synthesis.

**Cholecalciferol (Vitamin D) and its analogs**

Vitamin D and vitamin D analog toxicoses are on the rise. In addition to prescription strength vitamin D capsules (50,000 IU), and psoriasis creams (Dovonex®, Taclonex®), there has also been an increase in cholecalciferol containing rodenticides (D-Con®, Quintox®, Rampage®, etc.). These products cause elevations of serum Ca++ and P leading to soft tissue mineralization and renal failure.

Cholecalciferol is absorbed out of the gastrointestinal tract and transported in blood bound to carrier proteins. The major carrier protein is vitamin D-binding protein. Cholecalciferol is metabolized to 25-hydroxycholecalciferol (calcifediol) and then to 1,25-dihydroxycholecalciferol (calcitriol = active metabolite). Cholecalciferol and its metabolites exert their effects by binding to vitamin D receptors in tissues. Calcitriol is the most metabolically active form, with a 500 times greater binding to vitamin D receptors than that of calcifediol and 1000 times greater binding than that of cholecalciferol.

Calcitriol increases intestinal absorption of calcium, stimulates bone resorption, and increases renal tubular reabsorption of calcium. Elevations in P can be seen within 12 hours, and Ca++ elevations within 24 hours. Animals usually begin to exhibit vomiting, depression, polyuria and polydipsia by 12-18 hours post ingestion. Anorexia, bloody vomiting and diarrhea may also be seen. Renal failure can occur within 24-48 hours. Problematic doses can be as low as 0.1 mg/kg. Elevated blood calcium causes heart issues, and bleeding secondary to mineralization of the vessels, kidneys, stomach wall and lungs. Histopathologic lesions include: proximal tubular degeneration and necrosis, and mineralization. Renal ischemia occurs due to vasoconstriction and mitochondrial calcification secondary to hypercalcemia.

Emesis is recommended if pills/creams have been ingested for less than 30-60 minutes, with bait ingestion, emesis can be induced within 4 hours. Decontamination should continue with one dose of activated charcoal, followed by cholestyramine (300 mg/kg q 8 h for 4 days). Obtain baseline (< 8 hr post exposure) Ca++, P, BUN, and creatinine for future comparison. Monitor Ca++ daily for 4 days. If no elevations of calcium are seen by 4 days, no further treatment is needed.

If Ca++ is rising, start animal on intravenous 0.9% NaCl. Sodium competes for reabsorption with calcium in renal tubules. If the Ca++ values rise quickly (within 24 hours) consider calcitonin (inhibits osteoclastic bone resorption and reduces tubular reabsorption of calcium), pamidronate or zoledronate (inhibits osteoclastic bone resorption). Other treatments that can be used include furosemide and prednisolone (adjust fluids accordingly). Steroids reduce bone resorption, decrease intestinal absorption, and increase renal excretion of calcium. Phosphate binders should also be administered. The animal should be maintained on a low calcium diet (K/D, U/D, S/D, or macaroni and lean ground beef).

Monitor blood values frequently for 5-6 weeks. Treatment may be prolonged because of the long half-life of calcifediol (16-30 days). Prognosis decreases with prolonged elevations in Ca++. Lesions from soft tissue mineralization (renal, cardiac, GI) are poorly reversible and may result in long term sequelae or sudden death.
Ethylene glycol

Ethylene glycol (EG) is present in automotive radiator antifreeze, brake fluids, aircraft deicers, condensers, heat exchangers, home solar units and portable basketball goal post bases. Ethylene glycol may also be used to winterize toilets in RVs and summer homes in colder latitudes. Cats, rabbits and humans are the most sensitive to EG, with dogs, cattle, pigs and rodents having an intermediate sensitivity. Unfortunately, reliable toxic doses of EG have not been established for most animals. Much of the acute toxicity data available is based on lethal doses and do not take into account the fact that many animals may survive the initial stages of toxicosis only to succumb to kidney failure days later. Because of this, any suspected exposure of an animal to EG should be considered a potential toxicosis. When doubt exists, treat as if potentially toxic.

It is important to remember that EG is a potent alcohol and many of the early signs of toxicosis will relate to severe alcohol intoxication. Before metabolism starts, EG is no more toxic than ethanol, although EG is a more potent CNS depressant than ethanol. Because of the different mechanisms involved in EG toxicosis, clinical signs frequently change throughout the course of the toxicosis.

EG is initially oxidized to glycoaldehyde by alcohol dehydrogenase (ADH), and glycoaldehyde is then oxidized to glycolic acid, and then to glyoxylic acid. Glyoxylic acid is primarily converted to oxalic acid but may follow other metabolic pathways (end products include glycine, formic acid, hypuric acid, oxalomalic acid, and benzoic acid). EG and its first metabolite, glycoaldehyde, are mainly responsible for the initial CNS signs. The accumulation of glycolic acid and glyoxylic acid lead to metabolic acidosis. Acidosis will alter consciousness and increase cerebral damage. Glycolic acid accumulates because the lactic dehydrogenase enzyme that metabolizes glycolic to glyoxylic acid becomes saturated. Renal tubular injury is from the direct nephrotoxic action of glycoaldehyde and glyoxylic acid on the renal tubules as well as mechanical injury and obstruction by calcium oxalate crystals.

It is sometimes easier to break the clinical signs into 3 different stages, although considerable overlap between these stages may be seen and some animals will not experience each stage; death can occur at any stage. The stages are 1) neurologic—the initial inebriation due to the effects of alcohol on the CNS, 2) cardiopulmonary—due to severe acidosis and electrolyte disturbances, and 3) renal—due to renal tubular injury from calcium oxalate crystals and EG metabolites.

The neurologic stage generally begins within 30 minutes of exposure and lasts up to 12 hours. In some cases, this stage may pass quickly and may not be noted by the pet owner. Animals are initially ataxic, disoriented, stuporous, PU/PD (more pronounced in dogs) and hypothermic (especially cats). They then may appear to recover. By 6-12 hours, the neurologic status may worsen due to severe metabolic acidosis from EG metabolites. The cardiopulmonary stage generally occurs from 12-24 hours following exposure. Tachypnea, tachycardia, depression, and pulmonary edema may be seen. The renal stage can be seen as early as 12 hours, especially in cats, but is generally seen within 24-72 hours following exposure. Clinical signs include azotemia, depression, anorexia, vomiting, abdominal pain, oral ulcers, oliguria/anuria and seizures. Urinalysis shows low urine SG, glucosuria, and possibly calcium oxalate crystals (absence of crystalluria does NOT rule out EG toxicosis). BUN and creatinine become elevated but usually not before 12 hours post exposure; therefore BUN and creatinine are of minimal benefit in diagnosing early exposures.

Diagnosis is based on history, clinical signs, and confirmatory laboratory testing. In dogs, the ethylene glycol test kit can be an invaluable aid to determine whether an exposure is significant enough to warrant treatment. There are two available patient side ethylene glycol tests: VetSpec (Catachem) and Kacey. The VetSpec test is a colormetric qualitative test. It will be positive for any cis-1-diol (ethylene glycol, propylene glycol, glycerol, sorbitol, etc). It has both canine and feline tests. The Kacey strip test will be positive for any alcohol (see above, plus ethanol, methanol, etc.). It has both canine and feline tests on the same strip.

It is important to remember that some forms of activated charcoal and most diazepam injectable products contain propylene glycol that may interfere with the interpretation of the test. For this reason blood for testing should be taken prior to administration of propylene glycol-containing activated charcoal (check the label) or diazepam.

Other means of diagnosing EG exposure in pets include having EG levels run at a human hospital. Levels of 50 mg/dl or greater in dogs would be considered significant. In cats, any level above zero should be treated. Measuring anion gap (> 25 mEq/L) or serum osmolality (> 20 mOsm/kg) may assist in diagnosing EG toxicosis. Observation, via Wood’s lamp, of fluorescence in urine, stomach
contents or on paws/muzzle may suggest exposure (fluorescein dye is added to automotive antifreeze to help in detecting radiator leaks).

Treatment of EG toxicosis must be timely and aggressive. Failure to institute appropriate therapy within the first several hours may result in irreversible renal damage or death of the animal. For recent (within 45 minutes) exposures and asymptomatic animals, induce vomiting or perform gastric lavage. The use of activated charcoal is somewhat controversial, as aliphatic alcohols are not thought to be well adsorbed by charcoal. Based on exposure history and/or diagnostic test results, the use of either fomepizole or ethanol infusion is indicated.

Symptomatic animals should be stabilized as needed. IV fluids are extremely important, high infusion rates of crystalloids are necessary to correct dehydration and hypoperfusion. Treat acidosis and renal failure as needed. Oliguric or anuric animals may require peritoneal dialysis. IV ethanol and fomepizole (4-MP, 4-methylpyrazole) are used to delay the breakdown of EG to its more toxic metabolites. Best results with either of these treatments require initiation of treatment as soon as possible following ingestion. Ethanol has the advantages of being inexpensive and readily available, but it has some serious drawbacks, including worsening of metabolic acidosis and CNS depression. Fomepizole will not cause these signs and in contrast to ethanol, which is administered every 4 hours or as a CRI, fomepizole is administered every 12 hours for 36 hours. The main drawback with fomepizole is the cost and the need to have it compounded.

Treatment should be continued until animals are clinically normal and have had at least 24 hours with normal renal function and acid base parameters. Alternatively, for dogs, a negative EG test indicates that ethanol/fomepizole treatment may be discontinued (although treatment may need to be continued for any residual renal impairment). The prognosis for recovery depends on degree of exposure, length of time between exposure and treatment, and aggressiveness of treatment. The presence of oliguria/anuria indicates a grave prognosis.

On necropsy, histopathologic renal changes include: proximal tubular damage, tubular degeneration and necrosis, and intratubular calcium oxalate crystals. The crystals are birefringent, light yellow, and arranged in sheaves, rosettes or prisms.

**NSAIDs**

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the most common type of drugs involved in accidental overdoses in dogs. NSAIDs are a mainstay of pain control in both humans and animals. Arachidonic acid is released from cell membranes by phospholipase A2 and phospholipase C when a cell is damaged. Arachidonic acid itself has little activity, it can enter two pathways: the cyclooxygenase (COX) pathway, which produces eicosanoids, or the lipoxygenase pathway, which produces leukotrienes. Oxidation of arachidonic acid by COX, and further metabolism by other enzymes, leads to the production of various prostaglandins (PGs) and the release of oxygen-free radicals. These PGs include PGH₂, PGE₂ and PGI₂. Prostaglandins promote inflammation that is necessary for healing, but also results in pain and fever.

NSAIDs inhibit prostaglandin synthesis by blocking the conversion of arachidonic acid to prostaglandins by inhibiting cyclooxygenases. NSAIDs bind the active site of COX, usually through competitive inhibition. There are three isoform of COX: COX-1, COX-2, and COX-3. COX-1 is found in almost all tissues, including the GI tract, platelets, endothelium and kidneys, it is continuously produced and functions in tissue homeostasis. Most of the adverse effects associated with NSAID use are due to inhibition of COX-1. COX-2 is produced by macrophages, fibroblasts, chondrocytes, endothelial cells, and some other cell types. This isoform only function intermittently, and is induced by cytokines in area of inflammation. Inhibition of this enzyme produces antipyretic, analgesic and antiinflammatory effects. Little is known about the function of COX-3, which is present in dogs but not functional in humans.

Decreased prostaglandin production means decreased pain but also increased production of gastric acid and pepsin, decreased secretion of the protective mucous layer in the stomach and small intestine. Vasoconstriction in the gastric mucosa impairs mucosal circulation leading to mucosal hypoxia and thrombosis. NSAIDs inhibit renal blood flow, glomerular filtration rate, tubular ion transport, renin release and water homeostasis. The loss of the vasodilative action of PGE₂ and PGI₂ in the kidneys through inhibition of COX-1 leads to hypoxic renal injury. The medulla and renal papillae are at increased risk for NSAID-induced hypoxic injury due to their low oxygenation and relatively slow blood flow that predisposes to accumulation of toxic substances. Dogs, rats, mice and pigs are thought to be most sensitive to NSAID-induced papillary necrosis.
NSAIDs have a narrow margin of safety. GI ulcers and renal failure can be seen after an acute ingestion. Cats are thought to be more sensitive than dogs due to their limited glucuronyl-conjugating capacity. Intravenous fluids and gastroprotectants are the mainstays of therapy. The onset of GI upset is generally within the first 2-6 hours after ingestion, with GI hemorrhage and ulceration occurring 12 hours to 4 days post ingestion. Renal failure often occurs within the first 12-48 hours after exposure.

**Ibuprofen**
In addition to gastric ulcers and AKI, ibuprofen can cause CNS signs, affect platelet aggregation and rarely hepatic function. Ibuprofen has a narrow margin of safety. Even at the therapeutic dog dosage of 5 mg/kg, ibuprofen may cause gastric ulcers and perforations with chronic use. In dogs, an acute exposure of 50-125 mg/kg can result in GI signs (vomiting, diarrhea, abdominal pain, anorexia), > 175 mg/kg can result in more severe GI signs (hematemesis, melena) plus renal damage (PU/PD, oliguria, uremia), > 400 mg/kg results in GI, renal, and CNS signs (seizure, ataxia, coma). Ferrets that ingest ibuprofen are at high risk for CNS depression and coma, with or without GI upset. Assisted ventilation and supplemental oxygen may be required if animal is comatose. Seizures should be treated with diazepam. Both naloxone and lipids have been used to reverse the coma seen in ibuprofen toxicosis with mixed results.

**Naproxen (Naprosyn®, Aleve®)**
Half life in the dog is 74 hours, as the drug undergoes extensive enterohepatic recirculation. Ulcerative gastritis is possible in dogs at 5 mg and doses of > 10 mg/kg can cause acute renal failure. Due to the prolonged half life, fluids need to be continued for at least 72 hours and GI protectants for 10-14 days.

**Carprofen**
Dogs can develop GI ulcers at 20 mg/kg and acute renal failure at 40 mg/kg. Cats develop ulcers at 4 mg/kg and ARF at 8 mg/kg.

**Deracoxib**
COX selectivity is lost in overdose situations. Dogs can develop GI ulcers at 15 mg/kg and acute renal failure at 30 mg/kg.

**Grapiprant**
Grapiprant is a prostaglandin E (PGE) EP4 receptor antagonist; a non-cyclooxygenase inhibiting, non-steroidal, anti-inflammatory drug. The therapeutic index is wider with grapiprant when compared to other NSAIDs, but GI ulcers and AKI can still occur. Dogs can develop GI ulcers at 55 mg/kg and acute renal failure at 125 mg/kg.

**Lilies**
Members of the *Lilium* and *Hemerocallis* genera (Easter lilies, tiger lilies, day lilies, etc.) have been incriminated in causing acute renal failure in cats. The water soluble toxic principle is unknown. Even minor exposures (bite on a leaf, ingestion of pollen) may result in toxicosis, so all feline exposures to lilies should be considered potentially life-threatening. It should be noted that not all plants with “lily” in the name are the ‘true lilies’ of the Liliaceae family. Affected cats often vomit within a few hours after exposure. Within 24 to 72 hours of ingestion, oliguric to anuric renal failure develops, accompanied by vomiting, depression, anorexia, and dehydration. Elevations in BUN, creatinine, P and K⁺ are detectable as early as 12 hours post ingestion. Creatinine elevations may be especially high. Abundant casts, proteinuria, glucosuria, and isosthenuria are usually detectable on urinalysis within 24 hours of ingestion, reflecting lily-induced damage to renal tubular cells. On histopathology there is proximal convoluted tubule degeneration and necrosis with denudation of basement membrane and filling of tubular lumen with cellular debris. In severe cases, death or euthanasia due to acute renal failure generally occurs within 3 to 6 days of ingestion. When initiated within 18 hours of ingestion, fluid diuresis at 2x maintenance for 48 hours has been effective in preventing lily-induced acute renal failure. Conversely, delaying treatment beyond 18 hours frequently results in death or euthanasia. Baseline renal values should be obtained upon presentation and then repeated at 24 and 48 hours. In severe cases, dialysis may aid in managing renal failure until tubular regeneration occurs (10-14 days or longer).

**Grapes/raisins**
There have been numerous well-documented reports of dogs developing polyuric/oliguric/anuric renal failure within 72 hours of ingesting grapes and raisins, usually in large quantities. At this time the
mechanism of action and toxic principle are unknown. Grapes and raisins have come from various sources. Analysis of grapes and raisins involved in some of these cases have tested negative for heavy metals, pesticides, and known mycotoxins. Histopathologic examination has shown proximal renal tubular degeneration and necrosis with the basement membrane remaining intact. The distal convoluted tubules are usually less frequently and less severely affected. The extensive sloughing of proximal tubule epithelium results in extensive necrotic debris within the tubular lumens.

The lowest documented grape dose leading to renal failure is 0.7 oz/kg and the lowest documented raisin dose leading to renal failure is 0.11 oz/kg, however, there are reports that as little as 1 grape/raisin has caused renal failure. Some dogs are exposed and never develop signs and some only developed mild GI signs and recovered. Vomiting usually begins within 6 hours of ingesting the grapes/raisins. BUN and creatinine begin to elevate in 12-18 hours. Treatment is the same as for lily toxicosis. Dogs developing severe oliguria or anuria generally were poorly responsive to attempts to increase urine production (mixed results with peritoneal and hemodialysis). If renal values are normal at 48 hours, the animal can be weaned off fluids and sent home. Symptomatic care for vomiting, diarrhea, or other signs may be required.

Table 1. Nephrotoxicants in Dogs and Cats

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<td>Alkylating antineoplastics</td>
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Recurrent UTI and Current Approaches to Manage Infections
Michael Wood, DVM, PhD, DACVIM (SAIM)
University of Wisconsin-Madison, School of Veterinary Medicine

The development of urinary tract infections (UTIs) is multifactorial, dependent on alterations in anatomical, environmental, and immunological competency. For many individuals with normal urinary defenses bacteria are cleared from the bladder without the need of medical care. Unfortunately, despite the bladder’s inherent resistance to bacterial colonization 14% of dogs are estimated to develop UTIs during their lives. Of these cases, the most problematic are recurrent urinary tract infections.

Recurrent infections pose a challenge to the clinician as treatment should be paired with an identification or understanding of the cause of the infection. If the primary reason for bacterial colonization is not identified and addressed the patient may be at risk for developing another UTI. Traditionally, treatment of recurrent infections has relied on characterizing the UTI as a persistent infection or re-infection.

Persistent (Refractory) Infections
Persistent infections occur when appropriate antimicrobial therapy fails to clear the UTI and are typically identified within 2 weeks after completing treatment for the initial infection. Provided that the prescribed antibiotic was administered at the appropriate dose/duration, persistent infections indicate that the infecting bacteria developed resistance, the immune system of the patient is compromised, or the antibiotic is unable to achieve adequate concentrations to eradicate the infection.

Over the last few decades as the incidence of multidrug resistant (MDR) UTI has increased there is a rising population of patients with persistent infections not susceptible to first or even second line antibiotics. These infections can be difficult to manage. Alternative therapies have been proposed that are largely supported by individual case reports and anecdotal evidence. The use of high dose amoxicillin/clavulanic acid is one technique that uses the urine concentrating ability to treat MDR infections that otherwise appear resistant on susceptibility testing. Another approach that has been described to treat resistant organisms is directly instilling aminoglycoside antibiotics into the bladder to attempt sterilization.

Persistent infections can also be caused by inadequate antibiotic tissue concentrations and this can occur for a number of reasons. Decreased intestinal absorption of oral medications, altered perfusion of infected tissue, altered drug metabolism, or reduced urinary concentrating ability all may result in treatment failure. For these reasons, persistent infections require screening for systemic disease including an assessment of the function of the immune, gastrointestinal, hepatic, and renal systems. If the pathology resulting in a persistent infection cannot be corrected then antibiotic doses or route of administration should be adjusted to maximize tissue concentrations or an alternative antibiotic should be chosen.

A subset of persistent infections is termed “relapse”. Relapse differs from persistent infection in that during relapse urine can be cleared of infection but bacterial reservoirs remain within the body allowing for urine recolonization with the same organism within a few days to weeks. Sites that may harbor bacterial colonies include the kidneys, prostate, uroliths, vagina, and possibly the urinary bladder wall. The treatment goal for relapsing infections is to identify the site of infection so that eradication of the reservoir infection is ensured. For suspected tissue infections, bacterial sensitivities should be determined based upon the achievable plasma concentrations and tissue penetration of the antibiotic. Therapy is traditionally prolonged with general recommendations of treatment for 4-6 weeks. However the optimal treatment duration to clear these infections is largely unknown. It is possible that in certain circumstances treatment duration could be safely reduced. Relapse due to the formation of intracellular bacterial communities is a unique example of complicated UTI of unknown clinical importance in dogs and cats. In humans and mice small collections of intracellular bacteria can form quiescent intracellular reservoirs that reseed the bladder weeks to months later. Antibiotic therapy even with prolonged treatment duration is unable to clear QIR and currently effective therapy is not known.
Re-infections

Reinfection occurs when there are abnormal host defenses allowing new bacterial strains to colonize the urinary bladder weeks to months after an initial UTI. In both reinfection and relapse a time period exists when the patient’s urine is sterile making it challenging to differentiate between the two when similar bacterial species are isolated in subsequent infections. Re-infections require an investigation as to the urinary tract pathology that may be causing the failure in treatment otherwise continued reinfection is likely. This includes a complete systemic evaluation of the patient including advanced diagnostics such as radiographs/contrast studies, ultrasound and cystoscopy. Treatment of reinfection should be paired with urine culture and sensitivity to guide therapy and if clinical signs are mild to absent at the time of UTI diagnosis it is prudent to wait for antibiotic susceptibility results before starting therapy. In instances when treatment must be instituted immediately a first line antibiotic should be chosen. Since reinfection is caused by recolonization with a different bacterial strain each time, treatment duration is similar to the patient having numerous single infections making long antibiotic courses usually unnecessary.

Unfortunately in 25% of dogs the defect allowing re-colonization is not identified resulting in a frustrating cycle of repeat infections and treatment. These repeated infections lead to multidrug resistance and treatment failure. For these reasons a treatment plan focused on prevention of UTI is ideal. Unfortunately, there is little clinical evidence proving the long-term efficacy of nearly all preventative treatments in dogs and cats. Despite this a number of them are used clinically given the lack of alternative options.

UTI Prevention?

Of note: The following therapies are considered UTI preventatives and not treatment. If the patient has a UTI these options should not be used as a sole therapy to clear the infection. Also, nearly all of these options stem from their use in human medicine with variable efficacy and recommendation. In veterinary medicine there is currently a lack of clinical trials proving efficacy of all of these preventatives.

The first group of preventatives is the drugs that work by anti-adherence with cranberry extract and proanthocyanidins (PACs) being one of the most commonly used in dogs and cats. PACs isolated from cranberries can prevent P-fimbriated E. coli from binding to urothelial cells. Unfortunately, these molecules do not appear to have a similar effect on many other uropathogenic bacteria (including other E. coli) limiting their efficacy. There is data suggesting that the consumption of cranberry juice rather than PACS can block additional bacteria, but the mechanism by which consumption of the juice acts differently than the PACs is not known. The oral administration of D-mannose is a second anti-adherence therapeutic that may block the ability of type 1 fimbriated bacteria to bind to the bladder wall although clinical evidence of efficacy in dogs and cats is lacking. A third anti-adherence preventative is the use of glycosaminoglycans (GAGs). During E. coli UTI bacteria can injure the protective GAG barrier overlying the urothelium. GAG therapy contends that exogenous GAG will adhere to the urothelium or bind to invading bacteria preventing bacterial induced injury. In people, numerous independent studies have demonstrated that direct instillation of the GAGs hyaluronic acid and chondroitin sulfate into the urinary bladder significantly reduces UTI recurrence rates. However in veterinary medicine GAG bladder instillations can be challenging and expensive. Current evidence indicates that orally administered GAG may minimally increase urinary GAG concentrations in dogs, but nowhere near the concentration used in intravesicular instillation and so the clinical benefit of orally administered GAG is questionable.

A second group of preventatives are the urinary antiseptics. This group includes methenamine and modified dose antibiotic prophylaxis. Methenamine salts are urinary antiseptics that produce formaldehyde in an acidic urine environment. Since a urine pH of 5.5 is required to convert methenamine to bacteriostatic concentrations of formaldehyde, vitamin C or other urine acidifiers are co-administered to achieve this low pH. In people the long-term benefit of this medication is questionable and is generally only recommended for periods of a week or less. In addition it should be avoided in patients with metabolic acidosis and renal disease. In veterinary medicine there is not evidence supporting its use. In contrast modified dose antibiotic prophylaxis is used in veterinary medicine particularly in some animals suffering from recurrent infections with
severe clinical signs. This treatment will not prevent infection once the antibiotics are discontinued and selective pressures applied to bacteria via repeated courses of antibiotics may lead to the development of multidrug resistant uropathogenic bacterial strains and treatment failure. For that reason, first line antibiotics such as cephalaxin, ampicillin, or nitrofurantoin (a urinary antiseptic antibiotic used in humans) are common choices. The basis of prophylactic antibiotic therapy is to provide \( \frac{1}{3} \) to \( \frac{1}{2} \) of the total daily dose of an antibiotic usually at night after the last void of the day. Antibiotics concentrate in the urinary bladder overnight preventing colonization of the urine. The daily treatments are continued for 6 months with monthly cultures to ensure a breakthrough infection has not occurred. After 6 months if the urine remains sterile the antibiotics can then be discontinued and the therapy repeated as necessary.

A final group of UTI preventatives is bacterial interference. This therapy uses the theory that colonization of the urinary bladder with nonvirulent organisms may outcompete and prevent infection with more virulent bacteria. This is also the basis for monitoring and not treating patients with asymptomatic bacteriuria (ASB) or subclinical bacterial colonization of the urinary bladder. In people the treatment of ASB is not recommended in many circumstances since ASB clearance does not prevent future infection and the presence of ASB may actually be protective. Evidence demonstrating the benefit of ASB in veterinary medicine is lacking. The decision to treat or monitor is a challenging one since subtle clinical signs and pyelonephritis can be missed, but in patients with recurrent resistant bacteriuria this option should be considered if the underlying cause of recurrent UTI cannot be addressed. Situations where cautious monitoring can be considered is with attentive owners that have pets lacking clinical signs of UTI and pyelonephritis while also lacking hematuria, pyuria, fever, a leukocytosis, and azotemia. In these patients I will have owners check their pets temperature and measure hematuria using a urine dipstick 1-2x per week. If they note a temperature increase, hematuria, or changes in clinical signs I will then institute therapy. This is by no means a foolproof method and hence must only be considered with a strong understanding of the patient’s overall health and after discussing the pros and cons of not treating the bacteriuria with owners.

Other forms of bacterial interference include the intravesicular instillation of nonpathogenic \textit{E. coli} to try and establish an ASB and the use of probiotics. Thus far nonpathogenic \textit{E. coli} instillations lack clinical studies in veterinary medicine, although they are ongoing. As for probiotics, \textit{Lactobacillus} and other lactic acid producing bacteria are theorized to decrease vaginal pH thereby inhibiting uropathogenic bacterial colonization. In female dogs the importance of \textit{Lactobacillus} as a vaginal colonizer is not known as it sporadically colonizes the vaginal vault of dogs with recurrent UTI and normal spayed female dogs. Orally supplementing female dogs with lactic acid producing bacteria fails to alter the vaginal population suggesting that oral supplements containing lactic acid producing bacteria are of limited utility for preventing reinfection in dogs. Similarly, the use of estrogens also helps to prevent reinfection in women by altering the urinary microenvironment by promoting vaginal \textit{Lactobacillus} growth, lowering vaginal pH, and restoring atrophic mucosa within the urethra. Vaginally applied and not orally administered estrogen supplements reduce the number of recurrent UTIs in postmenopausal women. It is unknown how this treatment translates to dogs and cats, however patients with subclinical USMI may benefit from estrogen therapy.
Incorporating Angiotensin Receptor Blockers into Chronic Kidney Disease Management
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In patients with chronic kidney disease (CKD) altered kidney perfusion and filtrate formation activates the renin-angiotensin-aldosterone system (RAAS) resulting in the retention of sodium and water, systemic vasoconstriction and efferent glomerular arteriole vasoconstriction. Together these processes promote intraglomerular hypertension leading to glomerular injury and subsequent proteinuria. Renal tubular epithelial cells process these leaked proteins promoting the activation of inflammatory/vasoactive processes and mesangial proliferation causing tubulointerstitial nephritis, fibrosis, mineralization, and cell death. For this reason monitoring and managing hypertension and proteinuria induced by RAAS activation is essential to slowing renal injury.

Angiotensin converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARBs) are two drug classes designed to reduce RAAS activation. While ACEi have been used routinely in veterinary medicine for decades, ARB use has slowly increased and a veterinary approved product is now available for managing feline hypertension in the United States. The purpose of this presentation is to discuss RAAS activation effects, the difference between the ACEi and ARBs mechanism of action, and the practical application of these drugs to kidney disease.

Renin-Angiotensin-Aldosterone System Activation

Through the actions of angiotensin II (AT-II) and aldosterone, RAAS maintains systemic homeostasis with regards to fluid balance, blood pressure, and electrolyte concentrations. Systemic AT-II production begins with the release of renin from kidney juxtaglomerular cells in response to reduced renal blood flow and tubular filtrate flow. Renin catalyzes angiotensinogen to angiotensin I (AT-I) which in turn is hydrolyzed by circulating and endothelial associated angiotensin converting enzyme (ACE) to form AT-II. Alternate pathways of AT-II generation also exists within certain tissues such as the heart and kidney using enzymes such as chymase. These ACE independent pathways provide a mechanism for producing AT-II even when ACE is inhibited. This is called ACE-escape.

Once formed AT-II binds to type 1 (AT1) and type 2 (AT2) receptors. Stimulation of the type 1 receptor (AT1) is primarily responsible for systemic vasoconstriction, constriction of the glomerular efferent arteriole, and aldosterone production by the adrenal glands. These AT1 effects increase systemic blood pressure, increase intraglomerular pressure and filtrate formation, and increase salt and water retention to increase blood volume. Stimulation via the AT2 receptor has many renoprotective effects including promoting vasodilation, natriuresis, anti-inflammatory and antifibrotic effects.

Renin-Angiotensin-Aldosterone System Inhibition

In CKD chronic RAAS activation has numerous detrimental effects including systemic hypertension, increased intraglomerular pressure, glomerular enlargement, and proteinuria. Although it is possible to block RAAS at many points along its pathway in veterinary medicine ACEi are commonly reached for to manage hypertension and proteinuria. ACEi such as enalapril and benazepril inhibit the ACE mediated conversion of AT-I to AT-II and ACE degradation of bradykinin. Systemically the primary effect of ACEi is vasodilation. Locally at the level of the kidney ACEi promote efferent arteriole dilation thereby reducing intraglomerular pressure, glomerular hyperfiltration, and proteinuria. Long-term use of ACEi in dogs with CKD is beneficial as it has been shown to slow the progression of glomerulosclerosis and tubulointerstitial lesions.

In contrast ARBs (telmisartan and losartan) block the action of AT-II specifically at the AT1 receptor. This alternate mechanism of RAAS inhibition has two benefits. First, by inhibiting only AT1 signaling the renoprotective AT2 receptor signaling is preserved. Second, since ARBs act at the level of receptor binding, they are able to block the action of AT-II regardless of whether it is produced systemically by ACE or locally by alternate enzymes. This may help prevent the ACE-escape seen with the use of ACEi however additional clinical studies in dogs and cats are required to prove superiority to ACEi. Regardless, the clinical benefits of telmisartan appears to
be similar to ACEi and in cats the telmisartan has been proven to be effective at reducing blood pressure and proteinuria.

**Practical Application of ACEi and ARBs**

Given their vasodilatory properties both systemically and at the level of the glomerular afferent arteriole, ACEi and ARBs are valuable tools in managing patients with hypertension and/or glomerular proteinuria. The expected antihypertensive effects can vary depending on the species and class of drug used, however a reduction in blood pressure of 10-15% is generally expected. In proteinuric patients (UPC >0.2 in dogs and >0.4 in cats) ARBs and particularly ACEi are standard of care. Depending on the underlying pathology causing the glomerular proteinuria or hypertension, the effect of RAAS inhibition can vary and hence treatment recommendations for cats and dogs also vary.

In dogs hypertension is commonly associated with CKD and proteinuria. Given the importance in controlling both of these pathologies in patients with CKD, the dual benefit RAAS inhibitors provide make ACEi and ARBs a good first choice. Historically ACEi have primarily been used given that studies have demonstrated that enalapril/benazepril successfully reduce proteinuria, reduce progression of tubulointerstitial lesions, and improve CKD outcome. Starting doses of 0.25-0.5mg/kg PO q12h of enalapril and benazepril are common with a titration to up to 2.0mg/kg/d possible to control hypertension. ARBs like telmisartan are now being used as a therapeutic alternative starting at a dose of 1mg/kg PO q24h. However, to date there has been limited studies in dogs demonstrating ARB superiority to ACEi for the treatment of hypertension and proteinuria.

In general, ACEi and ARBs are weak antihypertensive drugs. In dogs with evidence of end organ damage or systemic blood pressures >200mmHg monotherapy with an ACEi or ARB is unlikely to reduce blood pressure sufficiently. In these instances co-administration with amlodipine is recommended. Amlodipine is a calcium channel blocker that acts on vascular smooth muscle, particularly the arterioles, resulting in vasodilation, primarily of the afferent arteriole. Theoretically this effect could be dangerous because if pressures are not adequately controlled the dilated afferent vessel may become a conduit in which high systemic pressures can be directly imposed on the glomerulus furthering renal damage. Combination therapy of amlodipine with an ACEi or ARB is considered ideal since amlodipine dilates the glomerular afferent arteriole and ACEi/ARBs preferentially dilate the efferent arteriole thereby limiting the potential detrimental effect of one or the other on glomerular hydrostatic pressures.

In cats amlodipine remains the recommended first-line therapy for hypertension regardless of cause given its proven efficacy. Unlike in dogs, amlodipine is often given as a sole agent. The justification is related to limiting pill burden rather than being pathophysiological as amlodipine has not been proven to improve survival times and activates RAAS. For that reason, in cats, particularly those with proteinuria, combination therapy with ACEi or ARBs may be beneficial. Benazepril in particular has been demonstrated to reduce proteinuria and glomerular capillary pressures in cats with CKD.

Unfortunately, in naturally occurring hypertension ACEi have not been proven to be particularly effective agents in reducing hypertension in cats. Studies have revealed only marginal improvements in systolic blood pressure (~10mmHg) and hence ACEI use as antihypertensive agents is generally not recommended unless the goal of therapy is to reduce proteinuria. In contrast the ARB telmisartan at a dose of 2mg/kg/d significantly reduced systolic blood pressures in hypertensive cats with nearly ½ of patients reducing their blood pressure by >20mmHg. This study did exclude cats with blood pressures >200mmHg and hence combination therapy with amlodipine is recommended if a RAAS inhibitor is used to treat marked hypertension. Although the evidence is limited, combination therapy with amlodipine and telmisartan does appear safe and effective.

Alternatively, in some patients, particularly dogs with proteinuria, the combined use of an ACEi and ARB can be considered when ACEi dosages are maximized. Their combined use has not been extensively studied in cats and dogs and must be used cautiously as the risk of side effects may increase and in one human study combined ACEi and ARB use was associated with increased risk of death. The main concerns associated with ACEi and ARB use is that the resultant dilation of the glomerular efferent arteriole may decrease glomerular pressures. This
effect can lead to a decrease in GFR and may be accompanied by a mild increase in the magnitude of the azotemia in dogs and cats. For this reason initiating ACEi or ARB therapy in a patient that is dehydrated, hemodynamically unstable, or in patients with unstable renal disease is not recommended. That said, there has been proven a clinical benefit of using ACEi in dogs with CKD and hence the presence of stable azotemia should not dissuade one from using these drugs. In general, in cats and dogs with CKD ACEi or ARBs therapy can be utilized in stage 1 and 2 CKD patients with hypertension and/or proteinuria. In stage 3 and 4 CKD their use should be approached more cautiously utilizing a lower starting dose (typically ½ the normal starting dose). If upon starting an ACEi or ARB the azotemia worsens by 30% or more then the dosage is reduced or drug discontinued. Other side effects to consider are that the RAAS inhibition can induce hyperkalemia while the resultant vasodilation can result in hypotension, weakness, and lethargy.
Medical Management of Hyperphosphatemia, Hypokalemia, and Acidosis in Chronic Kidney Disease
Michael Wood, DVM, PhD, DACVIM (SAIM)
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Controlling metabolic derangements in patients with chronic kidney disease (CKD) such as hyperphosphatemia, hypokalemia, and metabolic acidosis are important to maintain a patient’s quality of life for an extended period. Paramount to this therapeutic plan is feeding a diet designed specifically for patients with kidney disease. As the disease progresses through Stage II to Stage III and IV CKD often diet alone is insufficient to control these electrolyte disorders and additional therapeutic interventions are necessary. This lecture will focus on enteral therapies beyond diet that are beneficial in treating these metabolic abnormalities.

Hyperphosphatemia

Increased serum phosphate concentrations have been linked to higher mortality in dogs, cats, and people with chronic kidney disease. The observed hyperphosphatemia occurs secondary to an imbalance in gastrointestinal phosphate absorption, bone formation/resorption, and renal phosphate clearance. This imbalance occurs in early stages of CKD and if unaddressed can lead to renal secondary hyperparathyroidism. In patients with stage II, III, and IV CKD target serum phosphorus concentrations should be 3.5-4.5mg/dL, 3.5-5.0mg/dL, and 3.5-6.0mg/dL respectively. In veterinary patients, limiting serum phosphorus concentrations is primarily achieved by restricting the dietary consumption of phosphorus and the administration of oral phosphate binders. Phosphate binder dosing is usually performed to effect with second agents added as a patient nears a binder’s maximal dose before toxicity is possible. Therapy is initiated at the lower end of each binder’s dosing range and titrated upwards every 4-6 weeks until the serum phosphorus concentration is controlled. The efficacy of each binder at achieving this goal is variable and dependent on a number of characteristics unique to each binder.

Phosphate binders primarily exert their therapeutic effect by ionic binding or ion exchange. The former class of phosphate binders is primarily metal-based and contains two elements that disassociate within the gastrointestinal tract. One of the elements binds phosphate with high affinity to create an insoluble complex that is not absorbed by the body. The freed second element is then available to bind cations within the intestine such as hydrogen and frequently acts as an acid buffer thereby exerting a secondary effect of acidosis control. For most ionic binders a portion of disassociated elements fails to bind phosphorus and is systemically absorbed increasing the metal’s accumulation within the body thereby eliciting toxic or occasionally beneficial effects depending on the binder used. Examples of ionic binding phosphate binders include aluminum hydroxide, calcium carbonate, calcium acetate, magnesium carbonate, lanthanum carbonate, ferric citrate, and sucroferri oxyhydroxide. Alternatively, ion exchange phosphate binders bind phosphate to a large cationic polymer by exchanging an ion such as chloride or bicarbonate for phosphorus. The insoluble polymer thus makes any bound phosphorus unavailable for absorption into circulation. Examples of ion exchange phosphate binders include sevelamer hydrochloride, sevelamer carbonate, and chitosan.

Aluminum hydroxide is one of the more common phosphate binders used in veterinary medicine since it binds phosphorus with a higher affinity than most other medications with the possible exception of lanthanum carbonate, is relatively inexpensive, and the toxic side effects preventing its use in human medicine are rare in dogs and cats at recommended dosages. Aluminum hydroxide is soluble at a wide range of pH allowing disassociation throughout greater lengths of the gastrointestinal tract compared with other drugs. The disassociation creates an aluminum ion capable of binding phosphorus as well as a hydroxide ion capable of reducing the body’s acid load thereby allowing aluminum hydroxide to have a dual benefit in patients with CKD.

Calcium carbonate is another common phosphate binder given its low cost and dual effect of the carbonate acting as an acid buffer. One drawback to this binder is that it has reduced efficacy at a low pH however the calcium carbonate tablets dissolve best in an acidic pH resulting in greatest drug availability in an environment suboptimal to maximize phosphate binding. This
also means that patients on gastric acid suppressors may have reduced availability of calcium carbonate. Another obstacle to its use is the potential induction of hypercalcemia. This side effect is exacerbated by the co-administration of vitamin D analogues and hence calcium salts should not be administered to hypercalcemic patients or patients on calcitriol.

Lanthanum carbonate (Fosrenol) is a relatively new to veterinary medicine calcium-free phosphate binder. It has the advantage of binding phosphorus within a wide pH range and in animal studies lanthanum was comparable to aluminum salts in phosphate binding capacity. Largely insoluble, 93.4% of the drug is excreted in the feces of dogs after oral administration and hence side effects of lanthanum accumulation within the body are not currently reported in our patients. The greatest argument against the use of lanthanum carbonate is its high cost with limited proven survival benefit over less expensive alternatives.

Sevelamer is a nonabsorbable cationic polymer that exists in two forms as sevelamer hydrochloride (Renagel) and sevelamer carbonate (Renvela). The hydrochloride formulation was used in veterinary medicine in the 90’s and 00’s before being removed from the U.S. market because of the acidifying effects of the hydrochloride. It can be found compounded. The alternative, sevelamer carbonate, replaced the chloride ions with bicarbonate thereby creating a dual effect of phosphate binding and bicarbonate production. Sevelamer’s peak binding pH is 7 and it has less phosphate binding capacity per gram than most other phosphate binders. This reduced potency is related to a number of factors. First, sevelamer is a non-specific ion exchanger hence as it moves through the GI tract phosphate can be displaced by other anions. Second, within the duodenum sevelamer couples with bile salts limiting its ability to bind phosphorus. An additional side effect of the bile salt binding is that sevelamer can limit the absorption of lipid soluble vitamins and drugs such as vitamin D, ciprofloxacin, and mycophenolate. Overall this requires a higher dose of sevelamer to achieve similar serum phosphorus reductions in CKD patients and also higher costs hence this phosphate binder is only rarely used in veterinary patients.

Chitosan is a glucosamine polymer similar to sevelamer in that the compound contains positively charged amine moieties capable of binding phosphorus. It is one of the phosphate binders found in Epakitin along with calcium carbonate. Given that chitosan has limited amine groups (estimated to be less than ½) when compared to sevelamer, chitosan is an inferior phosphate binder at the same dosage. In addition, since chitosan is a non-specific ion exchanger, it will bind other ions throughout the gastrointestinal tract thereby further limiting its efficacy.

Hypokalemia

Hypokalemia is another common metabolic abnormality occurring in 20-30% of cats with CKD. In dogs it is less commonly clinically detected. The observed hypokalemia is secondary to reduced dietary intake and increased losses from polyuria, increased RAAS activation, and vomiting. Even moderate reductions in serum potassium can be associated with clinical signs such as lethargy, decreased appetite, muscle weakness, polyuria/polydipsia, and potentially constipation. These clinical signs are also common consequences of uremia and CKD in general making it challenging to know when a patient is suffering from hypokalemia without routinely checking serum potassium concentrations.

Potassium supplementation is recommended when dietary therapy is unable to maintain serum potassium concentrations within the normal reference range. Supplementing even when slight reductions are noted may be beneficial since there is circumstantial evidence that concurrent acidosis and hypokalemia may further reduce renal function. In addition hypokalemia is not noted until total body depletion of potassium occurs since the acidosis associated with CKD can cause cellular shifts of potassium resulting in potassium wasting not apparent on blood chemistry measurements. These observations have been further substantiated in cats where normokalemic cats with CKD have been shown to have reduced muscle concentrations of potassium. Despite these findings no studies have demonstrated that prophylactically supplementing potassium when serum concentrations are within the normal range improves survival. However many clinicians will aim to achieve serum potassium concentrations >4mg/dL in patients with CKD to prevent hypokalemia before clinical signs are apparent.

Although some potassium can be supplemented in subcutaneous fluids as potassium chloride (up to 30meq/L), in general enteral supplementation is recommended for otherwise
hemodynamically stable patients given that adverse effects are generally low. Potassium gluconate and potassium citrate are the two oral supplements used in veterinary medicine to increase serum potassium concentrations. Potassium gluconate is available as a palatable powder (Tumil-K or Renal K+) as well as a flavored gel (Renal K+) and tablet. Given this relative increased ease of administration it is a common first choice. However in patients where acidosis is also a concern potassium citrate (Polycitra K) is an excellent choice given the alkalinizing benefits of citrate. Oral formulations of potassium chloride also exist, however these are generally avoided in patients with CKD given their acidifying nature. RAAS antagonists such ACEi and ARBs also have the effect of increasing serum potassium, but are rarely used as the primary treatment of hypokalemia in patients with CKD. After initiating supplementation clinical signs including muscle weakness typically improve within 1-5 days and serum potassium concentrations should be measured every 1-2 weeks until the serum potassium concentration is achieved and clinical signs have resolved.

Metabolic acidosis

Greater than 50% of cats with advanced stage (stage IV) CKD are acidotic, however this metabolic abnormality is only present in <10% of cats with mild disease (stage II). The observed acidosis in chronic renal disease is multifactorial, but the decreased ability of the diseased kidney to secrete hydrogen ions by a myriad of mechanisms is most influential. While metabolic acidosis may have a direct effect on reducing renal function, perhaps more clinically important is how acidosis affects the patient systemically and drives catabolic processes. Problems including protein wasting, negative potassium balance, calcium loss, bone demineralization, and uremia paired with the clinical signs of muscle loss, weight loss, lethargy, weakness, and anorexia may ensue. Alkalization therapy can reverse these and should be instituted if patients continue to have persistent bicarbonate levels below 15mEq/L despite adequate fluid balance and appropriate nutritional management. As with the other metabolic abnormalities discussed above, changing to a renal specific diet may correct mild acidosis as renal diets are neutral to slightly alkalinizing. If after a few weeks of feeding a renal diet the acidosis persists then drug therapy consisting of sodium bicarbonate or potassium citrate can be implemented, with the latter having the added effect of potassium supplementation in hypokalemic animals.

When possible the total daily dose of an alkalinizing supplement is best administered over 2 or 3 separate administrations to prevent large fluctuations in blood pH. Sodium bicarbonate is readily available and inexpensive however as a powder it is not very palatable. Compounding the powder into capsules or administering tablets is generally preferable to mixing the powder in food. Potassium citrate is a good alternative treatment since its use can have the dual effect of correcting hypokalemia. Upon initiating therapy serum bicarbonate concentrations or blood pH should be checked in 2-4 weeks and dosages adjusted as necessary. Target bicarbonate concentrations of 18-25mmol/L in dogs and 15-22mmol/L in cats are recommended with the serum concentrations checked before bicarbonate administration.
Urethral Sphincter Mechanism Incompetence: Current Therapies
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Urethral sphincter mechanism incompetence (USMI), also called hormone-responsive, estrogen-responsive, and spay incontinence, is the most common cause of acquired urinary incontinence in dogs with females overrepresented when compared to males. While the exact etiology is unknown, USMI has been associated with hormonal, structural, and functional changes to the urethra.

Hormonally, USMI has been hypothesized to occur secondary to decreased estrogen concentrations as estrogen is an important sensitizer of the urethra to catecholamines. However, since not all dogs respond to estrogen and USMI is possible in both intact and neutered female and male dogs other etiologies must also be at play. Increased gonadotropin concentrations and decreased prostaglandins in spayed dogs are proposed alternate mechanisms, although additional evidence supporting these causes of USMI is necessary. Structurally and functionally, the urethra does undergo some histologic changes post ovariectomy (OE) with greater collagen to muscle ratios and an overall shorter length. These changes may reduce urethral tone and change the conformation of the lower urinary tract promoting incontinence. Despite these findings an association between age of OE and development of USMI remains unproven. Multiple reports reveal no difference in USMI rates and OE age. Others suggest OE prior to the first heat cycle is protective and yet others indicate that spay prior to 3 months is associated with a higher USMI rate. Early OE may be particularly important in dogs with an expected body weight >25kg since a recent retrospective study associated earlier neutering in this patient group with an increased risk of developing USMI.

USMI develops on average 2-4 years after OE regardless of timing (although can occur anytime from immediately to 10 years post-op). USMI is more common in breeds >15kg, with Doberman Pinscher, Giant Schnauzer, Old English Sheepdog, Weimaraner, Irish setters, Rottweiler and Boxer breeds over represented. Historically the urine leakage commonly occurs while the dog is lying down or sleeping due to increased intra-abdominal pressure and increased parasympathetic tone. However, these signs are not unique to USMI and a full physical/neurologic exam including and assessment of urine residual volume is required to rule out neurologic and paradoxical obstructive causes of incontinence. Definitive diagnosis is achieved by completing urodynamic studies including urethral pressure profiles. Given that these tests require specialized equipment and are challenging to interpret diagnosis is typically made based off of history, clinical signs, and response to therapy.

Pharmacologic Treatment:

The primary aim of USMI medical management is to improve urethral tone. Since activation of α-receptors within the muscle of the internal urethral sphincter controls urethral contraction historical therapies have included α-adrenergic agonists such as phenylpropanolamine (PPA). PPA controls incontinence in 86-97% of female dogs and 44% of male dogs however the therapy may become less effective with prolonged use. The traditional starting dose is 1.0-1.5mg/kg PO TID/BID, however in a retrospective study 8 of 9 dogs receiving once daily dosing at 1.5mg/kg became continent. Once a day dosing effectiveness has also been supported by urethral pressure studies. In general, after staring PPA improvement in clinical signs should occur within the first few weeks. If the signs remain or response is partial the dose may be titrated upwards towards 2mg/kg. Side effects are uncommon but include: aggression, hyperactivity, anxiousness, panting, diarrhea, vomiting, lethargy, inappetence, tachycardia and an increase in blood pressure. These effects are much more common when alternate α-adrenergic agonists such as pseudoephedrine is used. Pseudoephedrine is also not as effective as PPA and hence is not a recommended therapy for USMI.
Ephedrine is a third α-adrenergic agonists option with success rates at controlling incontinence reported to be close to 75%, however ephedrine needs to be dosed more frequently than PPA and hence PPA remains the standard therapy. In general, given the possible side effects α-adrenergic agonists should be used with caution in hypertensive and prehypertensive dogs.

When PPA is ineffective estrogens are commonly used as a second line therapy in spayed female dogs. Since estrogen acts by increasing the sensitivity of the urethral smooth muscle to catecholamines and other α-adrenergic agonists a treatment synergy is theoretically achieved by pairing estrogens with PPA. The most common estrogen formulations used in veterinary medicine are the long acting estrogen diethylstilbestrol (DES) and the short acting estrogen estriol (Incurin®). DES has historically been the estrogen of choice with a starting dose of 0.1-1mg/dog PO q 24 hr for 5-7 days tapered to a weekly dose. Monotherapy with DES controls incontinence in 60-65% of female dogs with an additional partial response in ~20%. However, DES is a long-acting estrogen and does carry some significant potential side effects including clinical signs of estrus (vulvar swelling and bleeding, mammary swelling, and attractiveness to males), perineal alopecia, bone marrow suppression (aplastic anemia, thrombocytopenia or neutropenia less common at low doses), and may increase the risk of mammary chain neoplasia. For that reason anecdotally there has been a gradual move towards using the short-acting estrogen estriol. Although estriol can still cause some of the reproductive signs including vulvar swelling, it is hypothesized to result in less bone marrow suppression with similar response rates when compared to DES (up to 82%). Given the potential side effects, titration to the lowest possible dose is recommended. A starting dose of 2mg PO q24 per dog is common with dose reductions every 2 weeks until the lowest effective dose is obtained. Anecdotally estriol can be used concurrently with PPA to achieve theoretical therapeutic synergy, although combination therapy has not been proven to be more effective than dosing estriol alone. Estrogen therapies should be avoided in male dogs, intact female dogs, and cats.

Other pharmaceutical therapies exist but are used rarely or in certain circumstances. For example, administration of gonadotropin-releasing hormone (GnRH) analogues and GnRH immunization reduce LH secretion and has been demonstrated to improve continence rates in dogs as these therapies are thought to increase urinary bladder threshold volume. However, high expense and/or limited availability limit the utility of these treatments. Testosterone cypionate is occasionally used in neutered male dogs that have a poor response to PPA. A recent case series describing the effects of monthly intramuscular testosterone injections reported that a median dose of 1.8mg/kg yielded an excellent response in 38% (3 of 8) of dogs and a slight response in 12% (1 of 8). In this study dogs receiving concurrent PPA was excluded and hence the benefit of combination therapy was not assessed. Although no adverse effects were reported in this study, prostatic hyperplasia and behavior changes have been reported in dogs with high circulating concentrations of testosterone.

**Interventional Techniques:**

When medical USMI management is unsuccessful interventional and surgical therapies are considered. Submucosal urethral injections of bulking agents such as bovine collagen, polydimethylsiloxane, and dextranomer/hyaluronic acid copolymer are one option. In this procedure injections are performed cystoscopically just distally from the vesico-urethral junction to diminish the urethral lumen thereby enhancing the sphincter function. They also increase muscle fiber length and thereby increase urethral closing pressures. Post procedure complete response rates of 50-80% can be achieved with collagen injection alone within 2-3 days. An additional 15-22% of patients can achieve continence with the addition of PPA. The bulking effect can last for 1–64 months (mean duration of 15-17 months) at which point additional injections are necessary. This treatment avoids daily medications but is expensive and bovine collagen has been more difficult to obtain in recent years. For that reason other
bulking agents such as polydimethylsiloxane and dextranomer/hyaluronic acid copolymer have been used with good success in clinical trials. A recent retrospective study comparing collagen to dextranomer/hyaluronic acid copolymer injections reported mean continence durations of 45.8 and 20.5 months respectively with 6-month success rate of 71 and 58%. In general, side effects from urethral bulking procedures are generally mild and transient occurring in roughly 15% of dogs. They include urine retention, stranguria, hematuria, vaginitis, urethral trauma and urethral obstruction.

**Surgical Management:**

In the last decade advances in the surgical treatment of USMI is arguably the most significant changes in managing USMI. Traditional surgical techniques such as colposuspension and urethropexy aim to increase the urethral pressure either by putting tension on the mid-urethra or by cranial repositioning of the bladder neck. Although these procedures provide reasonable improvement in continence in the short term, long-term these patients redevelop incontinence. Colposuspension initial response rate are about 50%, however, success rate decreases to <14% at 1 year post-surgery while with urethropexy initial response rates are about 87%, however, success rates decrease to <56% long-term.

An alternative to the surgical repositioning techniques is occlusion-based methods using the hydraulic occluder. This hollow horse-shoe shaped device placed around proximal urethra is connected via silicone tubing to a subcutaneously placed injection port. Continence is achieved by gradual expansion of the occluder by infusion of saline through the injection port. Simply placing the occluder may increase continence in 30-45% of dogs. Success increases to 61% when only placement is combined with medical management. If incontinence remains the occluder can then be inflated with saline thereby putting pressure on the urethra with reported continence rates >90%. Given that the pressure within the cuff can be increased and decreased as needed, these occluders also have good long-term success with continence extending >2y. Complications are common, but most are minor when the occluder is placed by an surgeon with expertise and include urinary tract infection, seroma formation, leakage of fluid from the occluder, prolonged urination, and recurrence of incontinence. Major complications may require device removal and include implant infection and urinary obstruction that can occur up to 23 months post operatively. These major complications are reported in 5-16% of dogs.

One final technique achieving more recent attention with a few clinical studies is the placement of suburethral tapes. In humans this procedure aims to restabilize the urethra after the loss of supporting structures post pregnancy. Although this procedure has been available for decades the initial approaches in dogs were extensive only achieving similar success rates to other available surgical techniques. More recently a less invasive transobturator vaginal tape inside-out technique (TVT-O) using an episiotomy approach reported continence in 7 of 12 dogs at 1y (58%) and 4 of 10 dogs at 4y (40%).
The challenge:
Trauma is in the top 3 causes of death in dog and cat patients and is responsible for 10-30% of animal visits to large tertiary veterinary hospitals and emergency visits to primary care veterinary teams.\(^1\) While survival to discharge is very good for animals with mild injury, animals with moderate to severe injury have a < 50% survival to discharge.\(^5\)

The opportunity:
There are a number of manifestations of trauma in canine patients that could serve as a translational model for improving both human and veterinary trauma patient care and outcome: e.g., traumatic brain injury, acute hemorrhagic shock diagnosis and intervention and resuscitation in the austere environment. In addition to helping inform go/no go decisions for drugs and devices being evaluated on the translational medicine pathway, research efforts could lead to improved care and outcome for veterinary clinical patients.\(^6\)

Why a trauma registry?
As human patient trauma systems developed and matured in an effort to improve trauma patient care and outcome in the 1970s and 1980s, pilot studies pooling trauma patient data were performed. The Major Trauma Outcome Study involved 139 North American hospitals collecting data on > 80,000 cases in a 5-year period of time.\(^7\) It was soon realized that trauma registries could be utilized to “describe injury epidemiology and suggest prevention strategies, to evaluate the performance of trauma systems and trauma centers, and to evaluate the efficacy of clinical interventions for trauma patients”.\(^8\) As a result of these efforts and findings, the American College of Surgeons worked collaboratively with trauma hospitals and systems to develop and utilize the National Trauma Data Bank (NTDB). The NTDB is utilized to create hospital benchmark reports, data quality reports and research data sets.\(^9\)

Cool idea, but can veterinarians build and sustain multi-hospital collaborations?
With the primary aim to improve trauma patient care and outcomes, the Veterinary Committee on Trauma (VetCOT) was formed to create a network of lead hospitals that will seed development of trauma systems. These hospitals will work collaboratively to define high standards of care and disseminate information that improves trauma patient management efficiency and outcome.\(^10\) Adapting successes from human trauma patient care, early implementation and utilization of a trauma registry that allows for continued advancement of trauma patient care was recognized.

In September 2013, nine inaugural Veterinary Trauma Centers (VTCs) were identified. In addition to ensuring resources and performance improvement processes within their hospital, these VTCs started entering data on all trauma cases presenting to their hospital into REDCap, a secure, web-based application for research studies.\(^11\) There are now 31 active private and university based VTCs that are responsible for the veterinary trauma registry that contains ~40,000 cases as of June 2019 (http://vetcot.org/index.php/home/the-veterinary-trauma-centers-leads-and-locations/vtcs-map/).

The VetCOT trauma registry
The goals of the VetCOT trauma registry are to inform improvement of veterinary and human trauma patient care and design clinical and pre-clinical trials that could inform go/no go decisions for interventional strategies and tools. The web-based digital interface of REDCap allows for hospitals to enter and track their data with continual access to their hospital’s data. There were 68 data fields in the original registry (Sept 2013-Mar 31, 2017).\(^12\) Quarterly reports summarizing all registry data are sent to VTC leads.

Steps to data capture
1. **Case identification** – each hospital creates a tracking process that is most effective for their system. Hospitals with electronic medical records (EMR) may tag cases electronically (e.g, required history question: “Is this animal presenting for traumatic injury?”, zero charge line item: “trauma case” or a check
box). Some hospitals have a printed Data Capture Form (DCF), frequently printed on a bright color of paper, that is labeled and follows the case during its initial evaluation/work-up (first 6 hours), then placed in a central local for Data Entry Personnel (DEP).

2. **Data completion** – there are 2 “forms” within the trauma registry: admission (data recorded in the first 6 hours from presentation) and outcome (data recorded for the entire visit). While the initial data is captured by most hospitals in “real time”, the outcome data typically requires personnel to evaluate records and determine final outcome data.

3. **Data entry** – VTCs are required to enter all cases on a quarterly basis by the end of the month following admission (i.e., cases seen Jan-Mar must be entered by April 30, cases seen April-June must be entered by July 31, etc.). Some VTCs have DEP enter “as they go”, utilizing time as available throughout the quarter, while other VTCs have assigned personnel with scripted time to enter cases. DEP at VTCs vary, but include students, house officers, Emergency veterinarians, veterinary nurses and staff specialists.

**Quality assurance and quality control-initial efforts**

Throughout the initial 2.5 years of data entry into the registry, DEP and VTC leads emailed suggestions regarding need for further clarification and areas of misunderstanding to Registry subcommittee lead. Additionally, during the annual veterinary trauma conference, data from the registry are presented, and discussion regarding areas of concern and opportunity for improvement occur. In July 2016, the VetCOT – Registry Subcommittee reviewed notes on registry “issues” and evaluated raw data from the database.

As a result of the review process, the registry was updated to include limit warnings, drop down menus and radio buttons (when appropriate) and improvement of visual appeal. Additional questions were added, and some questions (e.g., time of presentation) became “required” data point (including an option for categorical selection to be made). Finally, added options for drop down menus to decrease the amount of data being entered via textbox were created.

**How is data used?**

While individual VTCs are able to access and utilize their patient data at any time, utilization of data from the pooled registry is obtained through an application process outlined on the VetCOT website (http://vetcot.org/index.php/home/registry-use-materials/). One paper evaluating trauma scoring systems has been published in dogs (cats, *in press*).

**Registry vs. Data Base**

One important distinction to make here is that the trauma registry is capturing very specific data points for one “disease” process. This registry essentially represents a subset of each hospital’s entire patient dataset. As discussions move forward regarding enhanced collaboration and data-sharing between hospitals, multi-center databases need not replace registries, but serve to facilitate quality improvement and quality assurance checks. Additionally, registries allow for the added level of minimizing missing data fields as DEP are prompted to enter data on “required” questions.

**References:**


4. VetCOMPASS website: https://www.rvc.ac.uk/vetcompass


9. NTDB website: [https://www.facs.org/quality-programs/trauma/tqp/center-programs/ntdb](https://www.facs.org/quality-programs/trauma/tqp/center-programs/ntdb)

10. VetCOT website: [vetcot.org](http://vetcot.org)


High levels of student debt combined with modest incomes have created serious enough concerns that AVMA conducted a major symposium on solutions, and according to the 2018 Merck Wellbeing study, due to these concerns, only 41% of veterinarians recommend a career in veterinary medicine.

Basically, we have bought into the lie that in order to have a successful career in veterinary medicine, we must sacrifice financial well being and that you can’t make good money in this career.

We’re here to bust that myth, and get ourselves out of victim mode. If you are stuck in a financial rut, working full time and still barely making ends meet, then it is time to make a change.

In order to overhaul your wealth portfolio and manifest financial abundance, you must start with examining your own money mindset. Lack is a mindset, a fear of never having enough. A lack mindset stacks the cards against you from the beginning. To change that mindset, you must first be aware of it, know where it comes from (hint - your parents and society!), and consciously make different choices. Your mindset might be limiting you from creating wealth if you find yourself thinking, saying, or doing the following things:

- That’s too expensive.
- I can’t afford that.
- I didn’t go into vet med for the money.
- Veterinary professionals don’t make a lot of money.
- I’ll never pay off my student loan.
- I’ll take what I can for this patient, even if it isn’t the full estimate.
- I hate talking about money!
- I avoid high dollar stores because I don’t feel good enough to be there.
- I have jealousy or resentment of other people’s wealth.
- I Think that getting rich is evil.
- I don’t charge what I think I am worth because I don’t think others will pay it.

Your money mindset affects how you create estimates. Your staff’s money mindset affects how they present the estimates - if they don’t believe in the value or think that services are ‘too expensive’, that will translate to the client. Making sure everyone is on the same page when it comes to client communication about money is critical to your success.

Tips for success:
Avoid saying ‘it’s expensive’. That phrase throws up financial barriers immediately. Instead, utilize ‘it’s an investment in your pet’s health.

Overcommunicate value. If your client declines heartworm testing and prevention, but is wearing $300 jeans and driving a BMW, it isn’t that your services are too expensive, its that the client doesn’t understand the value.

Realize that you can’t please everyone, and that you will be truly ‘too expensive’ for some people. These people may not be your target market. If you choose to service low income clients, then you will need to get creative on how to provide financing or subsidy.

If you offer a free office exam to new clients, make sure that it is marketed as a ‘get to know you’ opportunity, and not just an opportunity to get free stuff.

Never discount on an individual client basis. This erodes your value, the practice’s value, and affects everyone.

If you must discount as part of your happiness quotient in vet med, then designate a charitable leg of your business. Never mix business and charity - it is confusing to your clients and your staff.

Knowing what you’re worth starts with knowing what you’re bringing into the practice, and to your own wallet. It’s easy to ignore the numbers, but by doing so, you’re shortchanging your practice and yourself. Familiarize yourself with what your contract gives you the ability to earn, how much you actually produce each month, and what your average transaction charge is. These aren’t numbers that you should use to judge your “success,” because success for some appointments means no treatments are needed, right? However, if you don’t know how much money you’re making for your clinic, you have no negotiating power. Bone up on some numbers just to keep in your back pocket. Most practice managers and practice management softwares are already calculating this- just ask for a copy.

Average transaction charge: Using PMS software, determine how many patients you personally saw in the last month, and all of their totals as well. Determine the average. Most practice management softwares should also be able to calculate this for you.

Production: determine what percentage of your production you earn according to your contract, or if you’re salaried, it’s still quantifiable data you can use to know your worth and aid in future negotiations. Your practice management software should be able to run period totals of any service and prescription charges under your name.

Compare your salary and/or production against local, regional and national benchmarks. Check the AVMA’s Veterinary Salary Calculator for comparison.

Tools/ideas to consider to increase production. Note: these are NOT intended to pad the bill, but rather to encourage compliance and make sure you’re not missing any services.

Scheduling rechecks before they leave
Forward booking annual appointments
Ensuring that they are up to date on all recommended services and parasite prevention products
Presenting estimates for all recommended services (e.g. dental cleanings)
• Calculate how much you “gave away” in the last month. It may surprise you. Work with your owners to determine what is an appropriate/acceptable amount to discount off each month (e.g. free nail trims, free ear cleanings, etc) and work to stay within that amount each month.

• Follow-up, personally, after illnesses, procedures, etc. to ensure all questions were answered and to increase client bonding.

• Consider checking your fee structure against benchmarks (e.g The Veterinary Fee Reference, Compensation and Benefits from AAHA Press). Making sure fee schedules are appropriate along with practicing a great standard of care lends itself to great production!

Now that you know what you are worth, lets look at building equity. It starts by first negotiating to get what you want and then making sure you are smart financially.

● Get a financial advisor
  ○ If you don’t have one - get one
  ○ What are your 1 year, 3 year and 5 year goals
  ○ Visualize what you want retirement to look like, when you would like that to happen
Title: Essential exchanges Part 1 and 2

Sarah J. Wooten, DVM, CVJ
Kimberly Ann Therrien, DVM

Conflict avoidance unfortunately manifests itself when people do not deal with the conflict at hand but instead use other tactics to avoid the issue.

Three Types of Conflict Avoidance
1. Ignore the problem
2. Divert attention away
3. Shut down

Why is it important to address conflict?

1. Emotional health
With conflict avoidance people find themselves constantly suppressing emotions which eventually surface elsewhere in the form of anxiety or anger and when least expected.

2. Creation of fear
Conflict avoidance teaches the brain, in a negative way and creates fear. Honestly, people are only putting off what needs to be done or said and the feelings of relief negatively reinforce avoidance. While it may feel good in the moment to avoid the conflict, in the long run it increases fears.

3. Creates missed opportunities
When avoiding conflict, people are in actuality stunting their own personal growth by missing chances that could offer them change, growth, development and stop them from become stagnant. These fears of conflict can become irrational and lead to phobias which then can be restrictive in everyday lives and stop others from living a fulfilling life.

Non Violent Communication Techniques
People often avoid conflict because they have not been taught to utilize nonviolent communication techniques. Most people unknowingly use automatic, reactive behaviors to deal with conflict because they are afraid of the conflict, and wish to get out of it in any way possible.

Conflict can be approach in a different way that reduces stress and allows people to consciously and compassionate express themselves and be heard. It requires a reframing of our old fear-based ways of handling conflict, which often include withdrawal, judgement, criticism, defensiveness, and attacking.

The steps to express your needs, as presented by Marshall B. Rosenberg, PhD in his book ‘Nonviolent Communication’ include the following 4 components:

1. **Observe** the behaviors in the doctor that are impacting your well-being or work environment
2. **Notice** the feelings that arise as a result of the doctor’s behavior, either good or bad.
3. **Honestly** share how you feel in relation to the observations you have made.
4. **Clearly request** a concrete action in order to enrich your life or improve your work environment.

In contrast, if your doctor shares a need for a behavior change from you using the same four components, your job is to empathetically receive this information and modify your behavior to improve the situation.

A good tool to use with the nonviolent communication style is ‘I feel’ statements. Feel free to use the following skeleton to frame your requests to your doctors:

When I notice (fill in the blank undesirable behavior) it makes me feel (insert feeling here) because I need (insert need here). Would you be willing to (insert very specific request here).

Things that can block communication flow between doctors and technicians:

1. Making judgements or global statements about the other’s character. “You are lazy.” “Your behavior is inappropriate.”
2. Making comparisons to other staff members.
4. Defending yourself.
5. Withdrawing or attacking
Title: 50 Shades of Greatness

Sarah J. Wooten, DVM, CVJ
Kimberly Ann Therrien, DVM

StrengthsFinder™

There are very few people around us who know or understand who they really are, how unique they are or how to engage this uniqueness in everyday life. A tool like StrengthsFinder™ can introduce a new way of looking at oneself – through the lens of strength. When you venture to discover what you naturally do best you encounter improved self-awareness and performance as well as a new way to explain who you are to others. In our current society, most of us have focused on fixing weaknesses or opportunities, thus it goes without saying that focusing on strengths requires a tremendous shift in thinking for us but also in how we engage others. It becomes about developing relationships and creates a sense of belonging. Studies show that when leaders focus on strengths among their team, associates are 6 times more likely to be engaged in their work. With increased engagement at work, most have greater productivity and satisfaction which in the end positively impacts their relationships at work, at home and within their community.

Discover your strengths

"There is no more effective way to empower people than to see each person in terms of his or her strengths." -Don Clifton

The book Strengths Finder2.0 from Gullpop offers you the opportunity to complete a Clifton strengths assessment that identifies an individual's unique sequence of 34 themes of talent as well as a more detailed Top 5 report. Once you have these, you can begin to better understand what your strengths are and how you can leverage them every day.

https://www.gallupstrengthscenter.com

Reframing Success and Developing an Internal Compass

“It is the set of the sails, not the direction of the wind that determines which way we will go.” - Jim Rohn

When you are feeling lost, your values work like a magnetized needle, carefully redirecting you back on course. Values lead you forward by reminding you not only of what you believe, but
they also remind you of *who you are*. Knowing these two things will determine the direction you will go.

**Hone in on what is important to you**

Creating a list of life values, who and what you want to be known as, is an important step in building greatness because it is a tool that you can use to define yourself on your terms. This is important because if you don’t define yourself, then you will let external circumstances define who you are or who you ‘should be’.

By creating a clear set of values, you are able to understand what in life is most meaningful to you, and consequently, run everything through that filter. Begin by asking yourself, what do I value most in life? Write down 5 words that come to mind. If you are drawing a blank, an extended list of life values is available at this link:

[http://www.theseedsofbeauty.com/en/content/extended-list-personal-values](http://www.theseedsofbeauty.com/en/content/extended-list-personal-values)

Out of those 5 values, pick the top three that most resonate with you. This isn’t a concrete choice - values can change over time, so don’t be afraid to make a decision because you can change it if and when you need to.

Next, write out a sentence or two about what each of your top three values mean to you. Keep your list of your top three life values in a place where you will see it often. This list will serve as a visible reminder to check yourself - are you thinking, acting, and/or feeling in alignment with your more desired values? Consider putting reminders on your phone to pop up during the day as visible reminders. These reminders will help develop your internal compass that is based upon the values that you choose for yourself.

If you feel your life isn’t in alignment with these values, what is one step that you can take to get closer to alignment?

How do you behave under pressure? Do you behave in accordance with your values, or in opposition? If so, what can you change?
Enlightened Rebels Make Shift Happen!
Next-Stage Practice Management & Leadership

We are at an important crossroads. Faced with rapid change, increased competition, economic challenges, increasing personal debt, and burnout, we need to consider new ways to preserve our practices and our people.

The future is becoming increasingly difficult to predict and change is needed for businesses of all kinds to remain relevant and effective in our rapidly changing and connected world. However, most veterinary practices still rely on a way of working designed over 100 years ago for the challenges and opportunities of the industrial age. It’s time to call into question some of the long-accepted workplace best practices that no longer serve us.

As a veterinary practice leader, what if you could learn to …

• Create a team that’s adaptable and responsive to change?
• Effectively engage people so they choose accountability and responsibility over an attitude of entitlement?
• Adopt an authentic leadership approach that doesn’t rely on chain of command or micromanagement, but a framework for dialogue & collaboration?
• Cultivate an inspired workplace environment, where you can dream and take risks to accomplish extraordinary things?
• Build an organization that achieves bottom-line results while rallying people around shared values and unity of purpose?
• Equip team members with abundant real-time information that increases the speed and accuracy of decision-making?

All this is possible when you honor six design principles that show up as common themes shared by progressive, “next-stage” organizations across a variety of industries. You must shift …

1. From an Operating System of Predict and Plan to One of Experiment and Adapt
2. From a Hierarchical Pyramid Structure to a Network of Teams Structure
3. From Directive Leadership and Centralized Authority to Collective Leadership and Distributed Authority
4. From Dependence on Extrinsic Motivators to Unleashing Intrinsic Motivation
5. From a Focus Purely on Profit to Purpose and Values
6. From Secrecy to Radical Transparency

Einstein’s definition of insanity – doing the same things over and over while expecting different results – is truer today than it has ever been. To deal with a future of rapid change, we need to reframe the way we think about work.
Six Design Principles for Next-Stage Veterinary Practices

Let’s take a look at short descriptions of each of the principles ...

1. From an Operating System of Predict and Plan to One of Experiment and Adapt: Progressive organizations understand that a long-term vision is essential but the ability to adapt and experiment in ever-evolving environments is superior to the ability to create rigid short-term plans.

2. From a Hierarchical Pyramid Structure to a Network of Teams Structure: Technology and connectivity has increased our ability to self-organize and collaborate more. Working as a network allows us to organize with many different kinds of connections, and increased autonomy.

3. From Directive Leadership and Centralized Authority to Collective Leadership and Distributed Authority: Circumstances and markets change rapidly as information flows faster. Rather than controlling through process and hierarchy, organizations can achieve better results by inspiring and empowering people at the edges to make decisions about how to accomplish their work, creating a high degree of freedom and responsibility.

4. From Dependence on Extrinsic Motivators to Unleashing Intrinsic Motivation: Next-stage organizations allow members the freedom to be their passionate, motivated, authentic, and diverse selves in the pursuit of tasks that fit individual interests, talents, and strengths.

5. From a Focus Purely on Profit to Purpose and Values: Organizations are changing their focus from profit alone to a long-term value based model. Aiming to do well by doing good creates a visionary purpose that brings together stunning talent, committed shareholders, partners and communities. This formula creates profit.

6. From Secrecy to Radical Transparency: Radical transparency and leveraging technology equip team members with abundant real-time information, increasing the speed and accuracy of decisions.

Learning Objectives
As a result of participating in this workshop, you will learn:

- What a progressive, 21st century veterinary practice looks like, including how to apply the six key design principles in a veterinary context.
- What you need to do differently to implement next stage practice management and leadership.
- How you may be getting in your own way by using outdated management practices.
- Where you and your practice should focus right now to make the shift from business as usual to next-stage veterinary practice management and leadership.
**Learning Facilitators**

**Jeff Thoren** is the founder of Gifted Leaders, LLC, an established executive and team coaching company based in Phoenix, AZ, serving clients nationwide. Jeff is committed to building engaging and innovative workplace cultures. He understands the mindset required to effectively lead and influence others in a business environment that is increasingly uncertain, complex, and ambiguous. His goal is to accelerate the shift from traditional hierarchical leadership (where a few leaders at the top exert control) to collective leadership (where leadership emerges as a collective capacity from everyone).

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**Peg Thoren** is a Team and Leadership Coach with Gifted Leaders. She coaches and consults with companies in the areas of enhancing positive team dynamics, increasing essential communication, identifying individuals’ gifts and strengths, and building positive work cultures. As a Program Supervisor in the healthcare field, Peg transformed her staff into a highly effective and productive team. She has had success creating positive work environments while working within small to medium sized companies as well as large bureaucratic systems. Peg has a M.Ed. in Counseling Psychology & Career Development from the University of Missouri. She is a Board Certified Coach and received her coach training through the Adler School of Professional Coaching.

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**Elise Lacher** is a recovering social worker and a CPA. She works exclusively with veterinarians to help them achieve their personal, professional and business goals. Using financial statements that have been formatted to give them good information, and teaching her clients how to read the story they are telling, she helps her clients rewrite the ending. Using innovative ideas and leadership concepts that work better in the 21st century, she has helped veterinarians create an engaging, productive and profitable culture in which they and their team thrive and enjoy coming to work each day.

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Robert Trimble is a future-focused veterinarian and entrepreneur with a passion for innovating at the intersection of medicine, technology, and organizational design. He is a former co-founder of Fuzzy (a pet health technology startup based in San Francisco) and now serves as the Executive Director of the Veterinary Entrepreneurship Academy with the goal of increasing the entrepreneurial- and business savvy of today’s innovative veterinary students.

Growing up on a Midwestern dairy taught him two important life lessons:
1) Treat people like they’re your neighbors (because they are) and
2) Treat animals like your livelihood depends on their well-being (because it does).

He likes small towns, big waves, authentic conversation, and consulting with business leaders to prepare their organizations for a future of rapid change.

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Bill Kearley is the owner of Veterinary Practice Success located in Boise, Idaho. Over the past 20+ years he has served veterinarians across the country who want to raise their practice to the next level by providing business coaching and consulting services for all types of veterinary practices. His primary goal is to work with practice owners and leaders to implement the best business practices that lead to overall practice and personal success.

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