Proceedings

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A patient should be considered geriatric when he has attained 75% of the expected life span and approximately 30% of the animal population is now considered geriatric. Although “age” is not a disease, due to the reduction in functional reserve, physiologic changes, and possible concurrent diseases, geriatric patients should be considered at higher anesthetic risk. These patients are not as efficient as adult animals to respond and compensate to external changes and stress. Some physiologic differences between geriatric patients and adults are:

- Hepatic clearance of drugs is decreased due to decrease in liver mass. The overall enzymatic activity is well maintained, but the reduction in hepatic mass and possible decrease in cardiac output negatively affect drug metabolism and can prolong the effect of drugs metabolized and excreted by the liver.
- The kidneys become less efficient due to loss of cortical mass, decrease in number of functional nephrons, and decrease in renal blood flow. Their glomerular filtration rate is decreased and these patients are more susceptible to ischemic renal injury and renal failure. Geriatric animals have decrease ability to compensate for fluid, electrolyte, and acid-base disorders. Renal excretion of anesthetic drugs can be delayed resulting in prolonged recovery.
- The heart becomes more fibrotic with increased myocardial fiber atrophy, the ventricular walls thicken and the overall contractility decreases. Valvular incompetence may be present due to fibrocalcification of the valves and fibrosis of the endocardium causes a decrease in compliance. There is also an increase in systemic vascular resistance, due to decreased elasticity of peripheral vessels, which contributes to the reduction of cardiac output. Despite an increase in norepinephrine, there is a reduced chronotropic response due to a decreased affinity of the receptors. The atrial kick becomes more important in maintaining an adequate cardiac output, since geriatric patients rely more an increase in end-diastolic volume and preload to increase their stroke volume.
- There is a reduction in brain size and loss of neurons, however the brain function is not affected. The decrease in functional neurons, affinity for neurotransmitters (i.e. dopamine, serotonin, tyrosine, and norepinephrine), and neuroplasticity results in a lower anesthetic requirement.
- These patients are more prone to hypoxemia due to the reduction in total and vital lung capacity, increased rigidity of the thorax due to decrease in intercostal and diaphragmatic muscle mass, decrease in gas exchange efficiency, and increased closing volume which contributes to an increase in ventilation-perfusion mismatch.
- They have less body water content, which can contribute to an initial increase in plasma concentration of injectable drugs. In addition, geriatric patients have less serum albumin which increases the unbound (active) portion of highly protein-bound drugs. For these reasons it is best to titrate injectable drugs to effect when possible.
- Their body fat increases, which increases redistribution into the adipose tissue of lipid-soluble drugs. This delays elimination of these drugs from the body prolonging the recovery time.

**Anesthetic management**

A thorough physical examination is paramount. The results of the physical exam and the patient’s history should guide the choice of further diagnostics. Blood work should include a complete cell blood count, serum chemistry and electrolytes, and urine analysis. Serum T₄ should be checked in geriatric cats. Any preexisting disease should be treated before anesthesia whenever possible. Analgesia should always be provided if pain is expected. Geriatric patients may suffer of preexisting painful conditions, such as osteoarthritis and cancer, which can worsen after manipulation during anesthesia. Analgesic drugs can decrease this discomfort. **Opioids** are considered an excellent choice: they provide analgesia, sedation, and they are reversible. Morphine, fentanyl, hydromorphone, oxymorphone, methadone are all appropriate options. For mild to moderate pain, partial mu agonists (i.e. buprenorphine) and agonist/antagonists (i.e. butorphanol) can also be considered. Local and regional blocks with **local anesthetics** should always be performed when possible. They block the transduction and transmission of noception and decrease the amount of induction and maintenance agents. Routine use of **non-steroidal anti-inflammatory** drugs should be reserved to geriatric patients with normal hepatic and renal function. Sedation is recommended to reduce anxiety and decrease the amount of induction drugs and inhalant anesthetics. **Benzodiazepines** (diazepam and midazolam) have the potential of causing excitement in adult animals, however they are good sedative in old and debilitated patients. They are also reversible and produce minimal respiratory and cardiovascular depression, however they do not provide any analgesia. **Phenothiazines** (i.e. acepromazine) can cause hypotension, they have long duration of action, and they are not reversible. Judicious use of these drugs (low limit of clinical dose range) and careful selection of the patient are recommended when phenothiazines are administered to geriatric patients. **Alpha-2 agonists** (i.e. xylazine and dexmedetomidine) should be used with caution in old animals. They depress the cardiovascular system by increasing afterload and...
decreasing heart rate, which can be detrimental in geriatric patients, especially if they have preexisting cardiac conditions. Induction drugs such as propofol and alfaxalone can be used in old patients. Titration to effect and use of sedative/analgesic drugs is the key to minimize cardiovascular and respiratory depression. Etomidate has not been specifically evaluated in these patients, but data in adult animals show that has minimal impact on the cardiovascular system and, when used in geriatric human patients, its effects on this system are similar to propofol. Ketamine, in combination with a benzodiazepine, can also be used as induction agent, but the effect may be prolonged due to slow liver metabolism and renal excretion. General anesthesia can be maintained with inhalants. To minimize vasodilation and hypotension, the use of pre- and intra-operative sedatives and analgesics is recommended. Physiologic support and vigilant monitoring is paramount for these patients during general anesthesia. As mentioned above, they have reduced tolerance to external stress and their mechanisms of compensation are decreased. If small complications are not prevented or quickly recognized and addressed, they might become major problems that can negatively affect the outcome of the anesthetic event. Geriatric patients are predispose to develop hypothermia, which increases the risk of infection, decreases the function of the immune system, increases recovery time and can contribute to an overall poor anesthetic outcome. Shivering increases the oxygen metabolic demand and these patients are already prone to hypoxemia. Continuous monitoring is recommended during the recovery phase. Assess pain frequently during recovery and treat if required or in doubt.
Anesthesia for the Patient with Endocrine Disease
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Patients with endocrine diseases have an increased anesthetic risk. It is important to know if the disease is treated and under control before anesthetizing these patients. Ideally animals with endocrine diseases should be stabilized before the procedure whenever possible. 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 is recommended. Extra parameters should be considered for specific diseases (i.e. a thyroid panel for hypothyroidism).

**Diabetes mellitus**

Animals with diabetes mellitus should have their blood glucose (BG) regulated before anesthesia. Some veterinarians prefer to have the animal in the hospital the day before surgery for evaluation of the metabolic status and for stabilization, if necessary. On the day prior to surgery, the animal should eat a normal meal and should receive insulin as normal, with no food after midnight. It is also reasonable to have the owner bring the animal to the hospital the morning of the procedure, because often the animal does not eat when in an unfamiliar and potentially stressful environment. On the morning of the procedure check BG and if it is higher than what is normal for that specific animal or greater than 250 mg/dL, give half of the insulin dose. If it is within the patient’s normal range, do not give anything. If it is lower than 150 mg/dL, supplement the IV fluids with dextrose (1%-5%). Plan surgery for early in the morning so the animal has the rest of the day to recover and can return to his normal food/insulin schedule. BG and electrolytes should be checked frequently during and after the procedure. Placement of a jugular catheter or an arterial catheter will make blood samples easier to obtain. It is important to prevent hyperglycemia and hypoglycemia. The goal under anesthesia is to keep BG within the range the patient has been at. Monitor BG level every 45-60 minutes throughout the procedure. When the blood glucose falls below the selected range or 150 mg/dL, add dextrose to the IV fluids. Initially begin with 1%-2.5% dextrose and if BG continues to be low increase to 5% dextrose. If the blood glucose level is too high (greater than 250-300 mg/dL) give regular insulin intramuscularly or subcutaneously at 4 to 6 hour interval. The dose is approximately 20% of the total daily dose of NPH or PZI. You can also give ½ of the dose of insulin the animal is on, but might take longer to see an effect with long acting products. There are no specific contraindications for drugs, however it is better to avoid agents that will increase sympathetic activity such as dissociatives (ketamine) or that will decrease insulin secretion and increase peripheral insulin resistance such as alpha-2 agonists (dexmedetomidine). These drugs will affect BG adding another variable to the treatment decision process. Also keep in mind the stress causes an increase in tissue insulin resistance, so try to make the pre-anesthetic and recovery periods as quiet and stress-free as possible. Continue to monitor BG in the postoperative phase. The animal will usually not eat well on the night after surgery so may not need to give insulin. The day after surgery go back to the routine food/insulin schedule. If the animal does not eat, IV dextrose and reduced doses of insulin may be recommended.

**Cushing’s syndrome or hyperadrenocorticism**

This disease is characterized by an excess of circulating cortisol. There are two main causes of this disease: 1) iatrogenic due to excess use of glucocorticoids use, 2) spontaneous, either pituitary dependent (80-85% of naturally occurring hyperadrenocorticism) or due to a tumor of the adrenal glad. The animal usually presents with pendulous abdomen, due to an increase in intra-abdominal fat (in the omentum), bilateral alopecia, comedones, dermal hyperpigmentation, polyphagia, polyuria/polydipsia, muscle weakness, increased pressure on the diaphragm from the pendulous abdomen and an enlarged liver, panting, and lethargy. These animals have decreased expiratory reserve and respiratory compliance and usually they require intermittent positive pressure ventilation during anesthesia. The skin is thin with fragile veins and tends to bruise easily when IV catheters are placed. Cushingoid animals are prone to pulmonary thromboembolism caused by hypercoagulability (increase of factors II, V, VII, IX, X, XII, fibrinogen and decrease of antithrombin), increase in hematocrit resulting in vascular stasis, prolonged periods of recumbency due to muscle weakness, and fragile vessels easily damageable. This risk might increase during extensive recumbency during general anesthesia. Use drugs that are short acting and reversible. Ideally it is best if the dog can ambulate within 4 hours of surgery. They can be hypertensive due to enhanced vascular sensitivity to vasopressors, excessive secretion of renin, activation of renin-angiotensin system, reduction of vasodilator prostaglandins, and increase secretion of mineralocorticoids. Cardiac and renal diseases caused by hypertension can be present. Keep in mind that if dogs are receiving angiotensin-converting enzyme (ACE) inhibitors, they tend to be hypotensive under anesthesia and the hypotension is less responsive to inotropic agents.

Pituitary Cushing’s syndrome can be treated with seleglamine, an irreversible inhibitor of monoamine oxidase (MAO type A regular doses, and type B at higher doses). MAO is an enzyme involved in the inactivation of nonmethylated biogenic amines such as norepinephrine, serotonin, dopamine (MAO type A) and tyramine, phenylethylamine, dopamine (MAO type B). The inhibition is irreversible so there is a prolonged effect of the drug. There may be interactions with anesthetic drugs, especially opioids (meperidine
inhibits serotonin reuptake). The interaction can cause: 1) excitation due to central serotonin over activity or 2) depression due to inhibition of hepatic microsomal enzymes and decrease in opioid metabolism. In human patients a rare but life-threatening interaction between MAO inhibitors and meperidine has been documented. The combination may lead to a serotonin syndrome, which is characterized by confusion, fever, shivering, diaphoresis, ataxia, hyperreflexia, myoclonus, and diarrhea. Fentanyl and pentazocine could also interact with selegiline. In humans it is recommended to avoid Ketamine, due to its effects on sympathetic nervous system, and sympathomimetic drugs which act indirectly by releasing norepinephrine and epinephrine, such as ephedrine, because they may provoke a fatal hypertensive crisis. The direct acting sympathetic agents such as norepinephrine, epinephrine, and isoproterenol are safe but caution is needed as the effects may be enhanced by receptor hypersensitivity. These interactions have not been reported in animals and they are unlikely to occur since selegiline is a MAO type B inhibitor only in dogs (affects tyramine and phenylethylamine). Serious drug interactions in dogs receiving selegiline are still possible, so use caution in these situations.

Avoid NSAIDs, due to their side effects on the gastrointestinal track when used in combination with high cortisol concentrations. If an adrenalectomy is performed, give dexamethasone at 0.05-0.1 mg/kg IV to prevent hypoadrenocorticism and monitor sodium and potassium concentrations.

**Addison’s disease or hypoadrenocorticism**

This disease can occur following destruction of the adrenal glands resulting in decreased production of glucocorticoids and mineralocorticoids, due to decreased secretion of ACTH by a pituitary gland (uncommon). The cause of Addison’s disease can also be iatrogenic after treatment of Cushing’s syndrome or abrupt withdraw of high-dose steroid therapy. Mineralocorticoids, mainly aldosterone, are synthesized and secreted from the zona glomerulosa of the adrenal cortex. They regulate sodium, potassium, and plasma volume. A lack of aldosterone results in hypokalemia and hyponatremia. Glucocorticoids, mainly cortisol, are synthesized from the zona fasciculata of the adrenal cortex. They stimulate gluconeogenesis and glycogenesis in muscles and liver. They also stimulate erythrocytosis, enhance fat and protein metabolism, maintain normal blood pressure, and counteract the effects of stress. They also affect vascular permeability, integrity of the endothelium, and sensitivity to catecholamines.

Animals with Addison’s disease can present hypovolemia, dehydration, hypotension, and bradycardia due to the severe volume contraction, hypokalemia, hyponatremia, hypalbuminemia, mild hypercalcaemia, pre-renal azotemia, and metabolic acidosis. Avoid hyponatremia and drugs that can cause cardiovascular collapse due to peripheral vasoconstriction, decreased venous return, decreased cardiac output, and decreased contractility. These patients may have pale mucous membranes, weak pulses, and increased heart rate. If these signs are noted, treat with IV saline and/or colloids to increase intravascular volume, increase blood pressure, improve renal perfusion, and decrease potassium. If hypoglycemia is present administer dextrose. Dopamine at 2 to 5 ug/kg/min IV can be used to improve cardiac performance. If during anesthesia there is unresponsive hypotension treat with glucocorticoids, such as prednisolone sodium succinate at 1-2 mg/kg IV or dexamethasone sodium phosphate at 0.1-2 mg/kg IV. It is necessary to provide glucocorticoid supplementation to prevent circulatory collapse and adrenal crisis during surgery. Use a low dose of glucocorticoids (i.e. dexamethasone sodium phosphate at 0.1-0.2 mg/kg IV) before induction of general anesthesia in regulated patients. If possible, administer IV fluids prior to the procedure. The anesthetic regimen is not as important as the pre-operative management. Avoid etomidate because it blocks the synthesis of cortisol for 2-6 hours after administration. Monitor blood pressure frequently (invasive blood pressure monitoring is preferred). Avoid stress during induction and recovery.

**Hyperthyroidism**

Most commonly seen in middle-aged and older cats. These animals present with multisystemic clinical signs, they are easily stressed, have respiratory muscle weakness, and excessive tissue metabolism with increased oxygen needs. They are not easy to handle and may pant and hy perventilate if in a stressful environment. Animals with hyperthyroidism may present weight loss, polyphagia, vomiting, and diarrhea. The weight loss, along with a poor hair coat will lead to hypothermia during general anesthesia. They tend to be more prone to dysrhythmias, increased heart rate, murmurs, and hypertension. They can have hvpertrophic cardiomyopathy and potential increased risk for cardiac arrest under anesthesia. These patients may present with renal disease with increase in BUN and creatinine. Blood flow must be maintained to avoid post-operative renal complications. They are poor anesthetic candidates, as they tend to be old, cachetic, fragile, and have organ system dysfunction.

If possible, treat (radioiodine therapy) hyperthyroid patients prior to surgery, especially if it is an elective procedure. If total T₄ or free T₄ is above normal limits, treat with methimazole or carbimazole for 6-12 weeks prior to the procedure to minimize anesthetic complications.

Avoid anticholinergic drugs because they increase myocardial oxygen consumption and increase arrhythmogenicity. If bradycardia and hypotension are present, glycopyrrolate is preferred over atropine. Benzodiazepines and opioids are excellent choice for pre-medication. Avoid hypocarbia by pre-oxygenating, but avoid stressing the patient. Induce anesthesia using injectable drugs if possible (propofol, etomidate, or alfaxalone); anesthetic boxes and facemask technique are stressful. Avoid dissociatives (i.e. ketamine) as they stimulate the sympathetic nervous system and increase catecholamine release. Due to their respiratory muscle weakness, these animals
usually require positive pressure ventilation. Isoflurane or sevoflurane can be used for maintenance. Avoid hypotension to prevent post-anesthetic organ dysfunction. This can be challenging if the patient is on antihypertensive drugs, such as beta-receptor antagonists, anlodipine, and ACE inhibitors. Hyperthyroid patients can have an exaggerated response to catecholamines, so use reduced doses of direct-acting vasopressors such as phenylephrine. Pay close attention to heart rate, rhythm, and ECG. It is paramount to recognize and treat dysrhythmias to avoid severe cardiovascular complications.

If animals with hyperthyroidism are not properly managed under general anesthesia they may develop thyrotoxic storm in the post-operative period. Clinical signs of thyrotoxic storm include tachycardia, dysrhythmias, hypertension, fever, and shock. Monitor heart rate, blood pressure, and body temperature during recovery.

Post-operative complications of thyroidectomies include: hypocalcemia due to removal of parathyroid glads (monitor calcium levels for 4-7 days after surgery), Horner’s syndrome, vocal cord paralysis due to recurrent laryngeal nerve injury (if both nerves are involved there can be severe dyspnea and a tracheostomy may be necessary), hematoma formation, and airway obstruction.

**Hypothyroidism**

Hypothyroidism occurs primarily in dogs due to destruction of functional thyroid tissue cause by immune-mediated lymphocytic infiltration of the gland. These animals may present anemic, weak, hypothermic, bradycardic, and with weak pulses. These signs are due to decrease in thyroid hormone and basal metabolic rate. It is recommended to treat with L-thyroxine to achieve normal level of total T4 before surgery.

Animals with hypothyroidism are sensitive to sedative drugs and the lower end of normal range can be used. They are prone to cardiovascular and respiratory depression. Intermittent positive pressure ventilation may be required due to impaired ventilatory responses to hypoxemia and hypercarbia complicated by possible obesity. Anesthetic drugs that induce peripheral vasodilation can cause marked hypotension. Bradycardia and hypotension should be treated promptly with anticholinergics, IV fluids, and positive inotropes. These patients can develop hypothermia during general anesthesia due to their decreased basal metabolic rate. This can affect drug metabolism and prolong recovery time. Monitor blood glucose as hypoglycemia may be present and should be treated with dextrose IV. Delayed gastric emptying time may occur increasing the risk of regurgitation and aspiration during induction and recovery.

There are no specific contraindications for anesthetic drugs, however the use of short acting and reversible agents is recommended. Since recovery may be prolonged, titrate induction and maintenance drugs to effect.

In recovery, continue to administer oxygen and monitor body temperature. L-thyroxine treatment should be resumed post-operatively as soon as possible.
Neonatal and pediatric patients are at higher anesthetic risk and attention should be focused on the unique physiology and specific requirements of puppies and kittens. Dogs and cats are considered neonates up until their first 6 weeks of life and pediatric until their first 12 weeks. In general, pediatric patients have less organ reserve and do not respond as well as healthy adults to physiologic changes caused by general anesthesia. Some physiologic differences between neonate/pediatric patients and adults are:

- **Hepatic function may be decrease.** Hypoalbuminemia is common, which results in more free and active portion of protein-bound drugs. Young animals can be more sensitive to drugs such as ketamine, etomidate and non-steroidal anti-inflammatory drugs. They also can be more sensitive to hypoglycemia due low store of hepatic glycogen and it is important to decrease or avoid the fasting period and monitor blood glucose under general anesthesia. Their hepatic enzyme activity is decreased, resulting in a prolonged duration of drugs metabolized by these enzymes.

- **The kidneys are less efficient and neonates are more predisposed to fluid overload.** The glomerular filtration rate is decreased with slower tubular secretion, which results in prolonged effects of drugs eliminated by the kidneys.

- **The heart has less contractile tissue and their cardiac output is heart rate dependent.** It is very important to maintain a normal heart rate to avoid decrease in cardiac output. Their resting cardiac index is higher than adults and their cardiac reserve is minimal.

- **The blood-brain barrier is more permeable and allows for more diffusion of anesthetic drugs into the CNS.** Their sympathetic tone is not completely developed, which results in minimal sympathetic stimulation, increase in heart rate and contractility, and vascular tone.

- **These patients are more prone to hypoxia and hypoxemia due to decreased pulmonary functional reserve, very compliant rib cage, high oxygen consumption rate, and decreased hematocrit.** Even minor hemorrhages can greatly decrease tissue oxygen delivery.

- **They have greater body water content, which can increase the volume of distribution of some drugs.** The circulating fluid volume is centralized, which increases the delivery of anesthetics to tissues highly perfused (i.e., brain). This centralized body volume makes the neonatal patient more susceptible to hypovolemia.

- **They have low body fat, which affect redistribution of drugs in the body.** This low body fat percentage, together with an immature thermoregulatory system, makes neonates and pediatric patient more susceptible to hypothermia.

**Anesthetic management**

A thorough physical examination is paramount. Basic blood work including at least packed cell volume, total protein, blood glucose, and blood urea nitrogen should be obtained. Suckling neonates should not be held off food and older pediatric patients can be fasted for no more than 3-4 hours prior to the anesthetic event. Sedation and analgesia should be provided. In the past it was believed that neonates experienced less pain than adults due to their immature nerve system, but we now know that this is not correct. Nociception occurs in very young animals and it can lead to chronic pain condition even later in life. Pre-emptive analgesia should always be provided if nociception is expected. Sedation is recommended to calm very active animals and to decrease the amount of induction and maintenance agents. Pre-medications should be titrated to effect, since pediatric patients may require lower dosages, although in some phases of development it has been shown that human pediatric patients may require more analgesic drugs. **Opioids** are considered an excellent choice: they provide analgesia, often they sedate neonate/pediatric patients, and they are reversible. Morphine seems to cause more respiratory depression than fentanyl. Partial mu agonists (i.e., buprenorphine) and agonist/antagonists (i.e., butorphanol) cause minimal respiratory and cardiovascular depression. **Benzodiazepines** (diazepam and midazolam) are also an excellent choice for neonates. Although they can cause some excitement in adult animals, they are good sedative for the very young ones. They are also reversible and produce minimal respiratory and cardiovascular depression, but they don’t provide any analgesia. Local and regional blocks with **local anesthetics** should always be performed when possible. They block the transduction and transmission of nociception and decrease the amount of induction and maintenance agents. Routine use of **non-steroidal anti-inflammatory drugs** should be reserved to older pediatric patients, with more developed urinary system. In adult subjects, the use of **anticholinergics** (atropine and glycopyrrolate) with pre-medication drugs is not often recommended, however it should be considered in neonate and pediatric patients since they cardiac output is heart rate dependent. **Phenothiazines** (i.e., acepromazine) can cause hypotension, they have long duration of action, and they are not reversible. It is best to use these drugs in more mature individuals. **Alpha-2 agonists** (i.e., xylazine and dexmedetomidine) should be avoided, if possible. They cause cardiovascular depression by increasing afterload and decreasing heart rate, which can be detrimental in neonates. Induction drugs such as propofol and alfaxalone have been used in neonate/pediatric patients. Titration to effect and use of sedative/analgesic drugs is the key to minimize
cardiovascular and respiratory depression. Etomidate has not been specifically evaluated in these patients, but it is commonly used in human pediatric anesthesia and in veterinary medicine. Ketamine, in combination with a benzodiazepine, can also be used as induction agent, but the effect may be prolonged due to slow liver metabolism and renal excretion. General anesthesia can be maintained with inhalants. To minimize vasodilation and hypotension, the use of pre- and intra-operative sedatives and analgesics is recommended. Physiologic support and vigilant monitoring is paramount for these patients during general anesthesia. Their fluid requirement is greater than adults, since they have higher percentage of body water, immature renal system, greater body surface and higher respiratory rate, which lead to higher fluid loss. However they are more susceptible to fluid overload, due to pre-existing hypoalbuminemia and immature renal function. They have low store of hepatic glycogen and they can easily become hypoglycemic. It is important to monitor blood glucose and supplement IV fluids with dextrose if necessary. As mention above, they are susceptible to hypothermia, which increases the risk of infection, decreases the function of the immune system, increases recovery time, and contributes to an overall poor anesthetic outcome. Shivering increases the oxygen metabolic demand and these patients are already prone to hypoxemia. Continuous monitoring is recommended during the recovery phase. Assess pain frequently during recov
Danger and Safety in Anesthesia
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Dangerous situations and complications that can occur during anesthesia can be minimized with careful consideration of patient, conditions, and equipment. Planning the anesthetic event should include prevention and management of potential complications.

Drugs
Always consider drugs your patient is currently on and concurrent diseases; is there a potential for interactions with anesthetic drugs that you are about to administer? Examples: 1) Dogs receiving angiotensin-converting enzyme (ACE) inhibitors tend to be hypotensive under anesthesia and the hypotension is less responsive to inotropic agents. 2) Alpha-2 agonists will increase blood glucose and should be avoided in diabetic animals. They also increase urination so would not be the drug of choice for patients with urinary obstruction.

Can anesthetic drugs be mixed in the same syringe? Most agents used for premedication such as opioids, alpha-2 agonists, midazolam, acepromazine, anticholinergics, and dissociatives can be mixed together. This is beneficial when the animal needs to be premedicated intramuscularly (only one injection is required). Mix drugs together right before they are given. Diazepam cannot be mixed with any other drugs except for ketamine due to its preservative propylene glycol.

Always label the syringe as soon as you draw up the drug. An unlabeled syringe should never be used. Have drug labels of different colors to make it easier to identify drugs. This is helpful especially when people are busy, tired, or at the beginning of their training. Keep drugs that come in similar bottles apart on the shelf. If a different route of administration was used, consider absorption rate and how this may affect drug uptake and if another dose can be given. Will there be tissue irritation or damage following extravascular administration? Propofol perivascularly stings but will not cause tissue sloughing or affect sedation. Make sure you always write the patient’s body weight in kilograms and not pounds! If the weight is wrong you may overdose your patient.

Have a well-organized drug box and keep controlled drugs separate from non-controlled agents. Controlled substances must be double-locked. The box should be opened to access drugs only when needed and it should remain locked between uses. A limited number of people should be allowed to access the controlled drug box. Have a drug log to record patient identification along with type of drug and amount the animal received. If drugs come in more than one concentration be sure you are using the correct volume/concentration for each animal.

Accumulation: multiple injections of an agent may lead to prolonged recovery. Cats will not recover as quickly as dogs from multiple doses of propofol and can develop Heinz body anemia.

Incorrect filling of inhalant vaporizer: this can be prevented by the use of keyfill type vaporizers. There will be a lower output of anesthetic if an inhalant with a lower vapor pressure (i.e. sevoflurane) is placed into a vaporizer set for a high vapor pressure agent (i.e. isoflurane vaporizer). A high vapor pressure inhalant (i.e. isoflurane) placed into a low pressure vaporizer (i.e. sevoflurane vaporizer) will produce high and potentially lethal concentrations. Vapor pressure of isoflurane and sevoflurane are 240 mmHg and 160 mmHg, respectively. If an inhalant is placed in the wrong vaporizer by mistake, make sure you completely drain the vaporizer before using it again.

Anesthesia machines and equipment
Most anesthesia machine can accommodate multiple vaporizers. Frequently vaporizers have a lock-out mechanism so only one vaporizer can be turned on at a time, but sometimes this mechanism is absent. When using these vaporizers, one could have more than one turned on at the same time by mistake, which could lead to an inhalant overdose and subsequent detrimental cardiovascular depression.

If the anesthetic machine tips over, the inhalant can flow into the bypass channel of the vaporizer. When the anesthesia machine is placed upright and the vaporizer turned on, a high and potentially lethal concentration of inhalant can be delivered. If a machine is tipped, place it upright, but do not use this machine. Empty the vaporizer of the liquid inhalant, run a high flow of oxygen through the vaporizer with the dial at a low setting and allow the vaporizer to dry out. This can take a couple of hours.

Then use a gas analyzer to verify the output from the vaporizer is 0 when the dial is off. Now refill the vaporizer and test it using the gas analyzer dialing different concentrations. It is also recommended to send the vaporizer to the manufacturer for re-calibration.

If the pop off valve, also called adjustable pressure-limiting (APL) valve, is left closed the intra-thoracic pressure will increase. This will cause a decrease in venous return and cardiac function, which can result in the death of the animal. It will also cause barotrauma. Usually blood pressure will decrease first, followed by changes in pulse oximeter and ECG.

Gas cylinders (i.e. oxygen tanks) should be stored in a safe place, away from heat and fire. If left upright they should be placed in an appropriate rack or chained to the wall. Never leave a gas cylinder in vertical position on the floor! Temporarily you can lay it flat on the floor. If the cylinder tips over and the stem breaks off or cracks the tank becomes a missile, as the gas is under high pressure.
Misuse of endotracheal tubes can damage the mucosa of the tracheal and lead to severe complications. Overinflation of the endotracheal tube cuff can cause tracheal irritation and possible necrosis of the mucosa, which can lead to tracheal tears. This can be seen most commonly in cats following anesthesia for dental procedures. Other causes of tracheal tears in cats undergoing anesthesia are traumatic intubation, removal of the tube with the cuff still inflated, and changes in body position without disconnections from the breathing system resulting in twisting of the tube within the trachea. These animals can develop subcutaneous emphysema a few hours/days after the procedure. Only inflate the cuff so there is no air leakage when the re-breathing bag is squeezed to a peak inspiratory pressure of 15-20 cmH₂O.

If the endotracheal tube is pushed too far into the trachea, endobronchial intubation can occur. In this situation only one lung is ventilated, which can lead to hypoxemia (this is more likely in cats than dogs). The endotracheal tube should be past the larynx and not further than the thoracic inlet. Best way to avoid endobronchial intubation is to pre-measure before inducing general anesthesia. If the tip of the endotracheal tube is placed against tracheal wall ventilation can drastically decrease. The Murphy eye represents a second opening to prevent possible ventilation impairment.

Laser surgery, especially in the oral cavity, represents a fire hazard when oxygen is used. To decrease this risk use injectable anesthetics and let your patient breathe room air. If you need to provide oxygen (i.e. your patient becomes hypoxic), wrap the endotracheal tube in aluminum tape to reflect the laser beam. You could also pack some wet gauzes in the mouth around the endotracheal tube. Make sure that oxygen does not leak around the endotracheal tube cuff (i.e. damaged cuff), which will promote combustion. If you are using gauzes (4x4s) in the mouth for laser surgery or dental procedures, make sure to remove them extubation. Gauzes left in the mouth can be inhaled by the animal causing asphyxiating and death during recovery. Be vigilant and make sure the same number of 4x4s that was placed into the oral cavity is removed.

Post-anesthetic blindness can occur in cats when spring-loaded mouth gags are used (i.e. dental procedures and endoscopy). Hypotension may increase the risk. The blood supply to the feline brain is primarily via the maxillary artery. It is possible that the use of the mouth gag reduces blood flow to the brain through the maxillary artery by stretching of the vasculature and/or adjacent muscles decreasing blood flow to the eye. Some cats will regain vision but others may not. A small mouth gag can be fashioned by cutting a tuberculin syringe barrel and placing it on opposing canine teeth and thus avoid overstretching the mouth.

**Patient**

When you plan the anesthetic event, it is important to consider not only history and physical exam, but also age, size, breed, and predisposition. Neonates and geriatric patients may require less amount of drugs and there is an increased anesthetic risk associated with these patients. Larger breeds tend to require smaller amount of drugs than smaller animals due to their lower metabolic rates. Obese animals should be dosed based on their ideal body weight to avoid overdosing. Keep in mind that if these animals present a thick layer of subcutaneous fat, you might need a needle of sufficient length to reach the underneath muscle with your injections. If the drug is placed into fat there will be slow absorption and, if a 2nd injection is administered, it can lead to an overdose when both doses are absorbed. Some breeds are more sensitive to specific drugs (i.e. sighthound breeds/thiopental or MDR1 (now ABSB1) positive dogs/opioids) and dose adjustments should be considered. When choosing drugs and route of administration it is important to consider the animal’s demeanor. Have trained personnel around aggressive animals and choose drugs/route that allow for fast injection (small volume), produce deep and reliable sedation, and can be antagonized in case of complications.

**Personnel**

Always keep in mind who is responsible of administering drugs and monitoring the animal under general anesthesia and what their skill level is. Injury from animals can occur if inexperienced personnel are involved. Only a minimum number of people should help with difficult animals. This will reduce the stress to the animal and decrease the number of people that could become injured. Once sedative drugs have been administered, leave the aggressive animal in a darkened quiet room with minimal stimulation for 20 to 25 minutes for the drugs to have their best effect. I prefer not having the owner assist the restraint/injection process, unless I’m dealing with a police dog that has a handler. In healthy dogs and cats undergoing a dysphoric recovery consider a low dose dexmedetomidine, 0.5 to 2 µg/kg IV, to provide sedation and analgesia. If only sedation is required, consider acepromazine at a very low dose (0.01-0.02 mg/kg IV). This will prevent animals from injuring themselves and people around them.

Make every effort to minimize exposure to inhalant anesthetics. If possible, fill vaporizers at the end of the day. The person in charge of this procedure should rotate daily. Make sure your scavenger system for anesthetic gases works properly. If you are using an active system, make sure it creates negative pressure. If you are using F-air canisters, make sure you weigh them periodically and discard them when they gain 50 grams.

Unfortunately drug abuse is always a risk when people have access to controlled substances. Our job is stressful and illegal use of controlled substances has been reported in our profession. Keep the drug box locked and limit its access to only a small number of people. Have someone do a daily morning check on all controlled drugs and the volumes in each bottle.
Stress is common when providing anesthesia. Plan ahead for possible complications to help minimize this stress. You are providing a valuable service to the animal patient and it can be a rewarding experience.
PropFlo™ 28 is a new formulation of propofol that contains benzyl alcohol, a preservative that increases the shelf life of this compound. Regular propofol should be discarded 6 hours after opening the vial, whereas propofol 28 can be used up to 28 days. Its mechanism of action and features are the same as regular propofol: it produces general anesthesia via interaction with GABA_A receptors, does not provide analgesia, it causes respiratory (apnea) and cardiovascular depression (hypotension), which are both dose dependent. Like regular propofol, it has fast onset and short duration of action of approximately 5-10 minutes. It is labeled for IV use in the dog at a dose of 2 to 4 mg/kg IV, titrated to effect. It is formulated as an emulsion in a multi-use vial at 10 mg/mL. Shake the vial before opening and date when the vial is opened so it can be discarded after 28 days. Refrigeration is not recommended. Propofol 28 is not labeled for use in cats due to the benzyl alcohol, which has the potential to cause toxic effects in this species. Cats lack adequate glucuronic acid conjugation, which results in a decreased rate of metabolism and cumulative toxic effects such as ataxia, hyperesthesia, fasciculations, blindness, aggression, convulsions, respiratory failure, and death. Although the use of regular propofol may be preferred in cats, it has been shown that normal to high clinical doses of propofol 28 have not caused any organ toxicity in healthy cats.

Ultiva™ (remifentanil) is a synthetic opioid with direct action on mu opioid receptors. It has an ultrashort duration of action with an elimination half-life of approximately 6 minutes. Its elimination is independent of hepatic or renal function since it is metabolized by nonspecific esterases in the blood and tissue and it is excreted by the kidneys. Due to its short duration, intra-operative analgesia is achieved by a constant rate infusion. The dose in dogs is 3 µg/kg IV followed by a constant rate infusion (CRI) of 0.1-0.3 µg/kg/min.

Remifentanil CRI (0.5 µg/kg/min) has been used in combination with propofol CRI (0.2 mg/kg/min) to provide total intravenous anesthesia in dogs with a fast and smooth recovery. Bradycardia may occur and may require treatment with an anticholinergic. Remifentanil can cause respiratory depression and controlled ventilation may be required. Remifentanil does not affect myocardial contractility and does not cause histamine release, although hypotension can be noticed.

It is mainly used in animals with impaired hepatic function since its metabolism is completely extrahepatic. It decreases the amount of inhalant needed for general anesthesia, which may help preserve hepatic blood flow. Recovery is very rapid even after prolonged infusions and remifentanil does not accumulate in the body. Systemic effects, including analgesia, subside quickly, approximately 5-10 minutes after discontinuation. For this reason it is important that long-term analgesia is provided to patients that underwent painful procedures and this analgesic protocol should be administered prior to recovery from remifentanil.

Simbadol™ is a new long duration formulation of buprenorphine, has been developed for use in cats. Its concentration is 1.8 mg/mL, which is much higher compared to the concentration of regular buprenorphine (0.3 mg/mL). It is administered subcutaneously at a dose of 0.24 mg/kg once a day for up to 3 days and the first dose should be administered approximately one hour prior to surgery. This regimen can provide analgesia for up to 72 hours. Simbadol is a controlled substance and it is a schedule III product. Simbadol has an onset time of about one hour and duration of 24-28 hours. Mydriasis may be noted. Buprenorphine is metabolized and excreted by the liver. Reversal with naloxone may be difficult since buprenorphine has a high affinity for mu opioid receptors, however side effects are infrequent. It should not be dispensed to the owner to administer to their cats at home.

Alfaxan™ is a new induction formulation containing alfaxalone, the induction agent, in a non-cremophor (cycloexdran) vehicle. Although this is a new formulation, alfaxalone has been used in combination with alphadalone in veterinary and human anesthesia for many years ago. The old formulation was discontinued due to its side effects (hyperemia, histamine release, and anaphylactic shock). The new formulation Alfaxan is free of these side effects. It is a clear, aqueous solution for intravenous injection registered for the induction and maintenance of anesthesia in dogs and cats. It is a sterile solution with a pH of 6.5-7 and it comes in a 10 mL vial of 10 mg/mL. Alfaxalone is a neurosteroid injectable anesthetic, which causes CNS depression by binding to GABA_A receptors. It has rapid liver metabolism and elimination and it has a wide margin of safety. In USA it is labeled for IV use only in the dog and cat, titrated to effect for induction. The formulation does not cause pain upon subcutaneous and intramuscular injection and these routes have been used successfully in several species, including exotics, for general anesthesia and sedation. It does not contain a preservative and it should be used within 6 hours after opening. Alfaxalone is a controlled substance and it is a schedule IV product. Like propofol, alfaxalone has a rapid onset and can cause cardiovascular depression, hypventilation and apnea. Alfaxalone has a short half-life with a duration is 14 to 50 minutes and can be given as a CRI. There is good muscle relaxation and rapid recovery, but if used alone rough recoveries with myoclonus and excitement can be noticed. For this reason it is advised to use it in conjunction with analgesic and sedative drugs. Induction doses are 1 to 4 mg/kg IV given to effect. If administered IM, lower doses are used due to the volume required for the injection. In this situation, alfaxalone can be combined with a sedative/analgiesic drug to produce sedation and induction of anesthesia can be achieved with more alfaxalone IV.
NOCITA® is a bupivacaine liposome injectable suspension approved for local infiltration in dogs only. It is a new extended-release bupivacaine, which could help raise the standard of care for managing post-operative pain in dogs and cats. It has the potential to provide up to 72 hours of post-operative pain relief. It is a local anesthetic that is injected directly into the surgical site, providing local pain control. As other local anesthetics, it blocks sodium channels and stops the transduction and transmission of nociception at the injection site. It is being used for post-surgical pain in humans and it is being developed for post-surgical pain in dogs and cats, but at this time it is only approved for use in the dog. The extended-release bupivacaine technology used in this product consists of multivesicular liposomes encapsulating aqueous bupivacaine. The liposomes are microscopic structures made of nonconcentric lipid bilayers designed such that bupivacaine is gradually released from the vesicles over an extended period of time. As bupivacaine is gradually released from individual liposomes, it will distribute locally to the surrounding tissues. Nocita should not be co-administered with other local anesthetics such as lidocaine as a safe interval has not been determined. It is used as a single dose at 5.3 mg/kg (concentration is 13.3 mg/mL = 0.4 mL/kg) administered by infiltration into the tissue layers before closure of the surgical incision. You should wear gloves when handling the drug and the vial should be inverted several times (but do not shake) before withdrawing the dose. It can be administered undiluted or diluted with sterile saline or Lactated Ringer’s solution up to an equal volume (1:1 by volume). The vial should not be punctured multiple times, just draw up the dose (1 syringe per patient) and discard the remaining solution. The syringe with the Nocita must be used or discarded with 4 hours after withdrawal from the vial.

Galliprant™ (grapiprant) is a new analgesic and anti-inflammatory drug in the piprant class that functions as a selective EP4 prostaglandin receptor antagonist (PRA). Prostaglandin E2 (PGE2) is a prostanoid that serves important homeostatic functions and it is also responsible for regulating pain and inflammation. PGE2 binds to four receptors, among which EP4 is the primarily responsible for the pain and inflammation associated with osteoarthritis (OA). PGE2 is inhibited to varying degrees by steroids and cyclooxygenase inhibiting NSAIDs; however, administration of these drugs causes decreased production of PGE2 and decrease the homeostatic functions of the molecule, causing coagulation, renal, and gastrointestinal side effects. By inhibiting just the EP4 receptor, the homeostatic function of PGE2 is better maintained and a reduction in side effects is achieved. EP4 receptors are not only involved in inflammation and pain, but they may mediate central sensitization and may play a role in chronic pain. It has been shown that grapiprant can control pain associated with OA and it has an excellent safety profile. The dose for dogs is 2 mg/kg orally once a day.
Anesthesia ventilators are used during general anesthesia to provide ventilatory support during anesthesia. They are different from intensive care unit ventilators; they are simpler and lack of several functions necessary for critical patients. For this reason it is best to use these ventilators in overall healthy animals. Human anesthesia ventilators must adhere to national and international standards, however veterinary models have no regulations. It is therefore paramount that veterinarians understand how these machines work before using them on a patient.

Classification
There are different criteria to classify ventilators and sometimes the terminology used for the classification can be misleading. Usually ventilators are classified based on power source, drive mechanism, major control variable, cyclic mechanism, and bellows.

Power source
This is what is required to operate the ventilator and can be compressed gas or electricity. Most modern ventilators use electricity. There are also some ventilators that can use both sources.

Drive mechanism
This is the mechanism used by the ventilator to deliver a breath. It can be compressed gas or an electronic mechanism. Most veterinary ventilators use compressed gas as drive mechanism. These ventilators are called dual-circuit because they have a gas used to drive the ventilator (driving gas, that is between the housing and the bellows) and another gas that goes to the patients (breathing gas, oxygen mixed with inhalant anesthetics). Single-circuit ventilators only use the breathing gas and have an electronic piston instead of the bellows.

Major control variable
This is the variable that controls the delivery of the breath. Ventilators can be volume-controlled or pressure-controlled. For the first ones, the inspiratory phase stops when a preset volume is reached, for the others when a preset airway pressure is achieved. Most anesthesia ventilators are volume-controlled. To avoid barotrauma, most volume-controlled ventilators have an alarm and/or a pressure relief valve. You can set up a maximum working pressure (i.e. 20 cmH\textsubscript{2}O) above which the alarm goes off and the ventilator terminates the inspiratory phase.

Cyclic mechanism
This is the mechanism used by the ventilators to cycle from the inspiratory to the expiratory phase. Most veterinary ventilators use electronic timing mechanisms, where after the time required to deliver the gas for a breath, the ventilator cycles from the inspiratory to the expiratory phase. Some ventilators use a fluid logic unit and the cycle is based on pressure reached by the compressed gas rather than the time required to deliver a breath.

Bellows
Ventilators are classified as ascending (or standing bellows) or descending (or hanging bellows) based on the direction of the bellows during the expiratory phase. Most modern veterinary ventilators are ascending.

Types of ventilators
There are many types of ventilators for veterinary use available and new ones are available every year. The description of each single ventilator is beyond the scope of these proceedings. The description of some ventilators often used in the clinic can be found in veterinary anesthesia books.

Setup and leak tests
Setting up a mechanical ventilator is pretty easy. If they are electronic ventilators, plug the power cord into a power source. The breathing hose needs to be connected to the reservoir bag mount of the anesthesia machine. They also have a scavenger system hose that has to be connected to either an active or passive scavenger system. You have to close the APL (pop-off) valve of the anesthesia machine otherwise the system will leak. Ventilators have their own pressure relief valve, called spill valve, which is located inside the machine and it is not visible.

To test ascendi ng bellows ventilators, setup it up (see above) and place a reservoir bag at the patient end of the wye piece of the rebreathing system. Using the flush valve, inflate the bellows and the reservoir bag. Make sure the oxygen flowmeter is turned off and the anesthesia APL valve is closed. If the bellows starts going down, the system has a leak.
To leak test descending bellows ventilators, setup it up (see above) and turn on the power knob. After the bellows is fully contracted, place your hand on the wye piece of the rebreathing system and turn off the ventilator. If the bellows starts going down, the system has a leak.

**Indications**

Mechanical ventilators can be used for any patients under general anesthesia. Very small animals require smaller bellows to avoid barotrauma (see complications) and sometimes ventilating animals that weigh less than 3-4 kg can be challenging. Very debilitated patients might not tolerate mechanical ventilation due to increased positive pressure in the thoracic cavity and decrease in venous return (preload) which affects cardiac output.

Ventilators are recommended when the thoracic cavity is open and the negative pressure is lost (i.e. thoracotomies and diaphragmatic hernias). They are used when neuromuscular blocking agents, such as atracurium and cisatracurium are used during some ophthalmic procedures, such as phacoemulsification, when the central position of the eye is required or when external pacemakers are in use to avoid constant contraction of the patient’s musculature. Ventilators are used for animals with increased intracranial pressure, where precise control of ETCO₂ is paramount to avoid increase in intracranial pressure. Obese animals and patients with intra-abdominal masses (tumors or during late stage of pregnancy) usually benefit from the use of ventilators.

**Complications**

Complications can occur due to ventilator malfunction or because the operator did not setup the machine correctly. Low pressure and delay breathing time alarms will alert the anesthetist, but constant monitoring of the ventilator is very important during its use.

Ventilator failure can occur if the machine is inadvertently unplugged from the power source. If the bellows is not connected correctly to the base or the housing is not tightly secured, the gas can leak and hypoventilation can occur. If the tidal volume delivered is too large, the patient can develop signs of barotrauma. These result in rupture of alveoli that can lead to interstitial emphysema and pneumothorax. To avoid barotrauma always check the peak inspiratory pressure (PIP) on the pressure gauge of the anesthesia machine. A general rule do not go over 12-15 cmH₂O in cats and small dogs and 20 cmH₂O in larger animals. If the ventilator has a maximum working pressure alarm, set it at the pressures mentioned above. If you set the minute volume (tidal volume x respiratory rate) too high or too low, hyperventilation or hypoventilation may occur. A tear in the bellows will allow the driving gas to move inside the bellows and mix with the breathing gas, which will decrease the fraction of inspired inhalant anesthetic (your patient may get light!). All ascending bellows ventilators have a positive end-expiratory pressure (PEEP) of approximately 2 cmH₂O. Some ventilators have a PEEP adjustment knob to modify this pressure. If this is set too high, the tidal volume may decrease too much and your patient may become hypercarbic due to hypoventilation. When higher than 2 cmH₂O PEEP is required, usually 3-5 cmH₂O is used. Make sure the tidal volume is still appropriate (to avoid hypoventilation) and that the PIP is not too high (to avoid barotrauma). Intermittent positive pressure ventilation (IPPV) increases intrathoracic pressure (less negative) during the inspiratory phase. This decreases the venous return to the heart (preload) and cardiac output. Most ventilators have a preset inspiratory/expiratory (I:E) ratio of 1:2, but in some machine this can be modified. It is advised to allow for more time during the expiratory phase compared to the inspiration to promote venous return to the heart and minimize the negative cardiovascular effects of IPPV.
In an age of quick fixes and television animal trainers, it is tempting to simply punish our companion animals for performing unwanted behaviors. However, as veterinarians we should be encouraging our clients to explore underlying motivations for any behavior that is ostensibly pestering, unusual, or a sudden change from a previous behavior pattern. These issues could be an indicator of medical problem or indication of a more serious anxiety or social stress concerns. Once the human-animal bond has been damaged, a pet owner is less likely to choose optimal treatment plans for any condition. We will review the presentation and treatment of several of the most common feline behavior problems seen at a referral teaching hospital’s behavioral service.

**Inappropriate elimination** is a term often used to describe eliminating outside of the litter box. It is very important to distinguish between marking and toileting. The former is a social stress issue and the latter is primarily a problem with the litter box environment. Inappropriate elimination will not be discussed in depth in this lecture, but conditioning through external punishment and rewards is rarely helpful or needed in the treatment of house soiling. Ensuring proper urinary health and providing the appropriate litter box environment is the only necessary treatment for this issue. Urine marking/spraying can be a very frustrating problem and treatment involves decreasing social tension through behavior modification and often psychoactive medications.

**Scratching** is a common behavioral reason for euthanasia or relinquishment to shelters. Although proper pain control and surgical technique can decrease discomfort associated with a declawing, this procedure will always be considered a controversial and/or unethical mutilation. In most situations scratching can be easily directed to specific locations. The functions of scratching behavior include nail health maintenance, territorial marking (visual and scent), and possibly an attention-seeking behavior in our pet cats. Owners can do a lot to minimize unwanted scratching by keeping their cats’ nails well trimmed, and providing appropriately placed, stable scratching posts. Posts or horizontal scratching surfaces should be placed in high traffic areas in the home. Appropriate deterrents like double-sided tape or compressed air remote punishers can be effective, but only if combined with the provision of appropriate substrates.

**Aggression** towards people and other household cats is another common presenting complaint to our service. Aggression to other cats can be motivated by fear, status, territory, play, or redirected from another target. Cats may show aggression to people most commonly out of fear/pain, redirection, petting-intolerance, play, and status. Proper diagnosis of the problem should begin with a thorough physical exam. Any condition that increases irritability could potentially lower the threshold for aggression. Pain and pruritus, as well as metabolic and organ dysfunction causing vague feelings of discomfort could be a contributing factor to anxiety and aggression. Cats can become stoic or very fractious in the exam room, making pain and hyperesthesia particularly challenging to adequately assess in the clinic. Veterinarians should ask the cat owner specifically about any changes in behavior that may indicate pain. Owners rarely volunteer this information as they think these behaviors are just part of the cat’s personality quirks. Therefore, veterinarians must make a thorough behavioral history part of every appointment. In addition to changes in behavior, owners of aggressive cats should be queried on targets of all aggressive episodes, the progression of the problem, relationship to all people and animals in home, and any triggers of fear and anxiety.

Proper treatment of aggression is rarely a quick fix. Separation of fighting cats is often inconvenient, owners may be unable or unwilling to avoid triggers, and behavior modification can be too time consuming. Therefore, aggression is another common use for aversive treatment without much thought to etiology and proper consideration of the side effects posed by aversive corrections. Most aggression is motivated by fear or apprehension so treatment should involve changing this underlying emotion and not just suppressing the behavioral symptoms of this fear. Aversive punishments may cause an increase, not decrease in the aggression by ratcheting up the fear. Also, the people, animals, and locations associated with the punishment can themselves start to trigger fear in the cat.

**Nighttime waking** should always be treated with the utmost urgency. Clients’ lack of sleep quickly leads to euthanasia or rehoming of any pet causing the nighttime disturbance. A sudden onset of the behavior could be caused by underlying medical (e.g. pain or metabolic condition), but the problem may be a lax circadian rhythm, which typical in housecats. A change in feeding and activity imposed by owners can help remedy the problem. Also clients must understand basic operant conditioning principles — providing food or attention at these times positively reinforces the attention-seeking behavior. Psychoactive medications, deterrents and sound blocking tools may be helpful tools for households. Factors such as age-related cognitive decline can be addressed with additional medications.

**Excessive vocalization** like any behavior can be caused by underlying medical issue, inappropriate rewarding of the behavior, and genetic predisposition. Clients should be counseled on realistic goals for a highly vocal individual cat, although some recommendations can be made to help the client manage the problem at certain times.
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Premier/PetSafe Twist n’ Treat and Egg-cerciser http://store.petsafe.net/pet-care/toys/page/2
Pipolino http://www.pipolino.ca/eng/
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Cat Shelves www.catsplay.com, The Refined Feline, or any floating bookshelf
Video Catnip www.videocatnip.com and Cat TV www.catty.com
Fencing
Purrfect Fence www.purrfectfence.com
Cats on Deck www.catsondeck.com
www.paws.org has other products
Sticky Paws www.stickypaws.com
Spray deterrents
SSScat spray www.petsafe.org
Scarecrow www.smarthome.com
Spray Away www.havaheart.com
Scratching Post (nontraditional)
www.moderncatstudio.com (several wall mounted) wood post, e.g. www.lovethatcat.com
Fear and anxiety are the primary emotions underlying most serious animal behavioral disorders. Psychoactive medications can be a critical tool in reducing anxiety and are best used in conjunction with behavior modification and environmental management. In addition, physical conditions may contribute to behavioral signs of anxiety or alter the choice of medication. Therefore, accurate behavioral diagnosis obtained from a thorough history and observation, and proper health screening for any additional medical conditions should be conducted before initiating psychoactive therapy. Once it has been determined drug therapy is indicated, several factors can help guide a clinician in choosing the best drug or drug combinations. The intensity of the fear reaction and the owner’s ability to predict and control fear-producing stimulus exposure may influence the choice of medication used in a particular patient. Most of the psychoactive medications used in veterinary medicine alter the fear neurocircuitry by modulating one or more neurotransmitters. Certain medications, like most antidepressants (selective serotonin reuptake-inhibitors (SSRI) or tricyclic antidepressants (TCAs)), moderately reduce anxiety through a slow-acting cascade. These can be helpful to augment severe anxieties, but are not typically the first line as a monotherapy for specific events. The focus of this presentation will be medications that are best used primarily for specific, predictable fear-producing triggers for clients to use in the home setting, although suggestions for veterinary administered medications for clinic sedation will be reviewed.

Various neurotransmitters can contribute to feelings of fear and panic. Norepinephrine (NE) is a catecholamine largely responsible for behavioral arousal when the sympathetic nervous system is activated. Beta-adrenergic antagonists (e.g., propranolol and pindolol) can block the peripheral effects of NE and alpha-2 agonists (e.g., dexmedetomidine and clonidine) are centrally-acting inhibitors of norepinephrine release. Both are, therefore, used to treat the physiologic and behavioral responses associated with the autonomic “fight or flight” activation. These medications should be avoided in patients with cardiovascular disease or other severely compromised patients. The mechanism of the antipsychotics/neuroleptics is inhibition of another catecholamine neurotransmitter, dopamine. The result is a general depression of the CNS that causes deficits in cognition, awareness, and motor function. It is debatable whether these medications truly reduce anxiety. Nonetheless, acepromazine may be acceptable when inhibition of motor output is the primary goal. Hypotension, bradycardia, and extrapyramidal signs are possible adverse effects of acepromazine. Serotonin (5HT) is a monoamine neurotransmitter like the catecholamines. The most popular serotonin-altering medications in veterinary medicine are the SSRIs and TCAs, which block the serotonin reuptake transporter on the presynaptic neuron. However, trazodone, an atypical antidepressant that primarily antagonizes the 5HT2A receptor, has become an extremely popular trigger-specific medication as well as an adjunctive maintenance medication. Although rare, the most common side effects with trazodone are cause agitation and aggression. Serotonin syndrome is also possible but rare with this as a monotherapy.

Drugs that affect gamma-aminobutyric-acid (GABA) are some of the most effective and powerful anxiety-reducing medications. The benzodiazepines, such as diazepam, alprazolam, and clonazepam agonize GABA-A receptors, facilitating neuronal inhibition. In addition, to anti-anxiety effects, these medications are also anticonvulsants and may increase sedation and hunger. The GABA-analog gabapentin can also be used as an anxiolytic. Although benzodiazepines can be a very useful class of medication in decreasing anxiety, they do have some undesirable properties. Oral diazepam should be avoided in feline patients due to the potential for hepatic necrosis. All benzodiazepines can cause paradoxical excitement and disinhibition of aggression. To monitor for adverse reactions, a trial dose of ANY situational medication should be administered prior to exposure to the trigger.

Various natural supplements containing supplements such as l-theanine, melatonin, alpha-casozepine, or Magnolia and Phellodendron botanic extracts have also been shown to reduce anxiety-related behaviors in some controlled studies, possibly by modulating the GABA or glutamate systems.

### Common psychoactive drug doses

<table>
<thead>
<tr>
<th>Benzodiazepine</th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alprazolam (Xanax)</td>
<td>0.02-0.1 mg/kg q4h</td>
<td>0.0125-0.25mg/kg q8h</td>
</tr>
<tr>
<td>Clonazepam (Klonopin)</td>
<td>0.1-0.5mg/kg q8-12h</td>
<td>0.015-0.2 mg/kg q12</td>
</tr>
<tr>
<td>Clorazepate (Tranxene)</td>
<td>0.5-2.0mg/kg q4h</td>
<td>0.5-2.0 mg/kg q12h</td>
</tr>
<tr>
<td>Diazepam (Valium)</td>
<td>0.5 mg/kg q4h</td>
<td>0.1-0.4 mg/kg q12h</td>
</tr>
<tr>
<td>Flurazepam (Dalmane)</td>
<td>0.1-0.5 mg/kg q12h</td>
<td>0.1-0.4 mg/kg q12h</td>
</tr>
<tr>
<td>Lorazepam (Ativan)</td>
<td>0.02-0.5 mg/kg q8-12h</td>
<td>0.03-0.08 mg/kg q12h</td>
</tr>
<tr>
<td>Oxazepam (Serax)</td>
<td>0.04-0.5 mg/kg q6h</td>
<td>0.2-1.0mg/kg q12-24h</td>
</tr>
</tbody>
</table>

- Oral diazepam linked to idiopathic hepatotoxicity in cats
- Oxazepam, lorazepam fairly low in potentially damaging metabolites

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**From Acepromazine to Trazodone: Choosing the Right Medication for Specific Triggers**

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Knoxville, TN
• Test for desired and adverse effects with low-dose trial prior to trigger exposure

<table>
<thead>
<tr>
<th>SSRI</th>
<th>Dog (all PO QD)</th>
<th>Cat (all PO QD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citalopram</td>
<td>0.5-1.0 mg/kg</td>
<td>0.25-0.5 mg/kg</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>1.0-2.0 mg/kg</td>
<td>0.5-1.5 mg/kg</td>
</tr>
<tr>
<td>Fluvoxamine</td>
<td>1.0-2.0 mg/kg</td>
<td>0.25-0.5 mg/kg</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>1.0-1.5 mg/kg</td>
<td>0.5-1.5 mg/kg</td>
</tr>
<tr>
<td>Sertraline</td>
<td>0.5-4.0 mg/kg</td>
<td>0.5-1.5 mg/kg</td>
</tr>
</tbody>
</table>

• Potential side effects including vomiting, diarrhea, constipation (cats), urinary retention, seizure, increased agitation/aggression/anxiety, decreased appetite, sedation

• Typically start at 50% dose for 7-14 days

<table>
<thead>
<tr>
<th>Tricyclic Antidepressant</th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amitriptyline</td>
<td>1-6 mg/kg q 12h</td>
<td>0.5-2.0 mg/kg q 12-24h</td>
</tr>
<tr>
<td>Clomipramine (Clomicalm*)</td>
<td>1.0-3.0 mg/kg q 12h</td>
<td>0.25-1.3 mg/kg q 24h</td>
</tr>
<tr>
<td>Doxepin</td>
<td>3.0-5.0 mg/kg q 8-12h</td>
<td>0.5-1.0 mg/kg q 12h</td>
</tr>
</tbody>
</table>

• FDA approved for Separation Anxiety in dogs
• Typically start at 50% dose for 7-14 days
• See SSRI for side effects, increased anticholenergic effects with TCA
• Trazodone (SARI antidepressant): dogs 2-7 mg/kg PO PRN 60-90 min before stressor exposure; also can be given q 8-12 hour maintenance; cats 25-100 mg/cat PO PRN 60-90 min before stressor exposure
• Buspirone (azapirone antidepressant): 0.5 – 2 mg/kg q 8-12 hr
• Clonidine (alpha-2 agonist): 0.007-0.049 mg/kg PRN 30 min before stressor
• Gabapentin (alpha 2 ligand, GABA analog): dog 3-10mg/kg BID-TID; cat 2-5 mg/kg BID
• Sileo® (orotrasmucosal dexmedetomidine gel): 125 mcg/m2 (following package dosing instructions)

References
Charts adapted from Crowell-Davis & Murray (2006) Veterinary Psychopharmacology
Kids and Pets: Creating a Harmonious Home
Julia Albright, DVM, DACVB
University of Tennessee
Knoxville, TN

Raising children in a home with pets can help teach kids responsibility and compassion, in addition to providing a lot of fun for the family. However, millions of kids are injured each year by a family pet and millions of dogs are relinquished to shelters or euthanized because of problems caused by careless interactions between children and pets. As veterinarians, we can be an initial and quality source of information to prevent many of these problems.

Not all kids are the same and not all dogs (or cats, guinea pigs, horses…) are the same. Thoughtful pairing of personalities can circumvent frustration. Unfortunately, most of our clients do not talk to us about obtaining a new pet prior to bringing the animal into the home. Even more common and tragic is waiting until the behavior problems are quite severe to broach the issues with the family vet. Providing material throughout the hospital or proactively asking questions may prompt clients to judiciously consider their decision to obtain a certain breed, or even bring a pet into a home with certain toddlers at all. There are several good books and pamphlets that could be left. Questions that you can ask to help guide a family are 1) is that breed of dog or cat appropriate for your family? 2) Should you get a puppy or an adult dog? 3) Should you obtain a dog from a shelter or a breeder?

There are no hard and fast rules to any of these questions. We need to throw out the extreme views that “there are no bad dogs, only bad owners,” or, conversely, any individual of any breed is predetermined to behave in a certain way. The truth is in the middle – genetics do play a role in behavior, but the environment experiences (especially the developmental socialization period of about 4-14 weeks of age) can have a strong influence on behavior. Choosing a dog that is anxious and not socialized with children during this sensitive period could be problematic. Successful dog-child relationships exist because an individual animal most likely has a genetic predisposition for tolerance, but also was exposed to young children and handling as a puppy. Children will inevitably hug, pull, grab, chase, stare at, and irritate the family pets. A good breeder or puppy class instructor will gently expose a puppy to these human actions while also pairing with good rewards. Some dogs that have not been around many people or children during their socialization period will tolerate children and threatening actions on the part of the human, but the risk for fear and defensive aggression is much higher in these animals. We must counsel our clients of the risk of obtaining animals from pet stores and large-operation breeders whose facility they cannot visit. As we know, these animals are prone to a slew of anxiety and aggression problems, but the general public still does not understand what constitutes a “puppy mill” and how poorly these animals adapt to life as a family pet.

Veterinarians should also make it standard practice to ask behavior-related questions whenever a client with both kids and pets has an appointment. We see many major behavior problems that could have been curbed months or years before the consult if someone had helped the client recognize their current methods of managing the dog should be changed. For example, a dog has always been a “velcro dog” or constantly following a certain adult around the house is probably not going to tolerate suddenly being locked out of the bedroom when the new baby arrives. A dog that is growls and snarls at strangers who try to pet it is likely to do the same (at least initially) to a toddler that wants to hug the dog. The veterinarian should help the client address any stress or anxiety disorder because dogs that show separation distress or other anxiety disorders are at a higher risk of aggression towards children in the home.

Future parents can take steps to prepare resident dogs or cats for the arrival of a new child. As mentioned above, ask clients about sound sensitivities, fear of certain people, and separation anxieties. Any changes should be addressed before the arrival of the new baby if possible. Adults should slowly ask the pet to spend more time behind a barrier and out of the bedroom at night. Changes for cats can be very stressful too. Environmental alterations like moving down to dark basement in a remote part of the house or not being allowed on furniture may not be readily accepted by the pet, so we may need time to acclimate the pet or even formulate alternative plans.

Data indicate aggression towards children is most likely defensive or anxiety-related, not dominance driven as many TV shows and traditional trainers would have you believe. A harmonious pet–children home should center on helping the pets feel more relaxed around the children, teaching the children proper ways to interact with the pets, sharpening foundation behavioral cues, and having realistic expectations. Pets should be given a “safe haven” (e.g., dog bed in adult bedroom, or crate) to which they can escape when feeling annoyed or overwhelmed by family life. Reinforcing the action of leaving the current area for this spot can greatly decrease defensive aggression, particularly if spot is strictly off limits to children. Reward-based training is critical in home with young children. Children mimic how parents interact with the pets, not how parents tell children to interact. A child that sees a dog leash-corrected and physically punished by adults in the home will also start to treat the dog in that manner. This may have no noticeable consequences when administered by the adult, but is very likely to result in aversion and even defensive biting of the child. There are rare cases, particularly involving infants and toddlers, in which aggression is motivated by predatory, not defensive aggression. These
are very dangerous situations and removing that animal from the home must be seriously considered due to the high risk of serious injury.

Appropriate play can be a fantastic way for kids to interact and reinforce appropriate behaviors (in the kids and pets). Children should NEVER use their body parts in play. Allowing a dog to mouth, jump, or engaging in wrestling will result in injury. Games like fetch and even gentle tug-of-war with a long rope toy is acceptable, and can be a great time to teach “drop it/give it,” “leave it,” and reinforce that the breaking of human rules such as no-teeth-on-skin or over-exuberant play will result in the end of the game.

References
Stop the 77 (stopthe77.com)
Family Paws Parent Education (www.familypaws.com)
As our pet population enjoys a longer-life span, age-related neurodegenerative diseases also become more prevalent. Cognitive dysfunction syndrome (CDS) is the most well-recognized form of dementia in pets and according to one internet survey, 5% of dogs aged 10-12 years and 41% of dogs 14 years or older show signs of CDS. An estimated 28% of cats 11-15 years and 50% over 15 years of age are affected. A large international study estimated this syndrome at 14.2% in older dogs. This same study found that only 1.9% of the dogs with signs consistent with CDS were diagnosed as such by a veterinarian. Recommendations published by the American Animal Hospital Association Senior Care Guidelines Task Force (www.aaha.org) emphasize the use of structured questionnaires to assess both the current and changing health and behavior of each pet.

Pathophysiology of cognitive dysfunction
Cats and dogs suffer non-specific brain aging changes such as neuronal loss, parenchymal reduction, and increased ventricular size. Widespread neuronal loss, especially pronounced in the frontal lobe and hippocampus, and decreased hippocampal neurogenesis are correlated with more severe cognitive impairments than aged-matched human and canine patients with less pronounced cognitive decline. The major hallmark of CDS pathology is accumulation of β amyloid (Aβ) plaques, similar to human Alzheimer's disease (AD) patients. The mechanism for Aβ deposition is presumed to be oxidative damage. Production of reactive oxygen species by the mitochondria accelerates with aging. ROS levels eventually outstrip the body’s natural antioxidant defensive mechanisms and significant oxidative damage develops. The CNS is particularly sensitive to oxidative damage due to the high lipid content and low levels of endogenous free radicals. Furthermore, Aβ aggregates in the cerebrovascular system result in increased cerebral microhemorrhages and this is also thought to contribute to cognitive decline. Some research suggests neuronal dysfunction and neurotransmitter level alterations in the cholinergic, serotonergic, and dopaminergic systems associated with AD and CDS are a result of Aβ damage and this, in turn, may lead to much of the symptomology associated with CDS.

Clinical and behavioral signs
Purpose-bred laboratory beagles have been extremely useful in revealing a correlation between cognitive functioning and brain pathology. Aged dogs show declines in spatial learning, executive functioning, and memory compared to younger animals in controlled laboratory psychological testing. Memory deficiencies preceded noticeable behavioral changes and brain pathologies. The link between cognitive decline, age, and neurodegenerative changes has not been studied as thoroughly in cats to date, but some memory and learning deficits similar to canine subjects were demonstrated in most studies.

This information can be clinically relevant by observation in the exam room and also obtaining a history from pet owners about exploratory behavior, elimination behavior, alterations in daily schedule or sleep patterns, and social interactions in the home environment. The acronym DISHAA describes the behavioral signs.

D – Disorientation
I – Interactions (altered social interactions)
S – Sleep-wake cycle alterations
H – Housesoiling
A – Activity level alterations
A – Anxiety level

Behaviors such as night-time waking, housesoiling (in cats particularly), separation anxiety, environmental fears, and aggression often prompt older cat and dog owners to seek veterinary help. However, the disease is probably quite severe by this stage. Since CDS is a progressive disease, therapies are most effective with early detection. A more detailed survey that can be answered quickly by clients during a clinic visit are available through several sources.

These longitudinal owner-queried surveys are an efficient, reliable, and inexpensive general method of aging assessment and should begin at approximately 6 years of age in our patients.

Medical differentials for behavioral changes
Cognitive Dysfunction is primarily a diagnosis of exclusion. A minimum database of physical exam, complete blood count, serum chemistry profile, and urinalysis should be obtained on any veterinary patient suspected of having CDS. Pain, discomfort, metabolic diseases and neurologic conditions that affect mentation are commonly linked to behavior changes in senior pets. Imaging (radiographs or ultrasound) for metabolic disease and organopathy evaluation should be considered. Magnetic resonance imaging (MRI), computed axial transmission (CAT) and positron emission tomography (PET) imaging can be useful in identifying other neurologic diseases that could show similar signs to CDS and the severity of age-associated parenchymal loss.
Treatment

Treatment of metabolic diseases is essential, but CDS can be concurrent with any other disease. Underlying metabolic diseases and any source of pain should be addressed. However, medications (e.g. steroids, phenylpropanolamine) used in the treatment of other conditions should be evaluated for behavioral side effects. Once a CDS diagnosis is strongly suspected, a multifactorial treatment plan encompassing medical/nutritional therapies, behavior modification, and environmental management should be employed. Current therapies do not reverse the underlying pathology, but research particularly in purpose-bred laboratory beagles, has shown improvement in cognitive functioning with several treatment modalities. This can translate into noticeable quality of life improvements in our patients even if the disease process is not reversed.

Environmental strategies

Maintaining basic social interactions through structured reward-based training and encouraging play can improve cognition. Solitary mental stimulation through food-dispensing toys and scent games may also be beneficial to aging dogs and cats. Sleep-wake cycles often undergo alterations resulting in more night waking. Providing more mental and physical stimulation during the day can promote better rest at night. Behavior modification techniques to foster physical and emotional relaxation are extremely useful to help owners cope with dogs that become anxious and restless.

Pharmacological, nutraceutical and adjuncive therapies

The biogenic amine systems (e.g., dopamine, norepinephrine, and serotonin) are compromised in CDS patients. Memory, social interactions, and other cognitive signs can be improved with the use of antidepressants such as selegiline (MOAB inhibitor), which may have the added neuroprotective benefit of slowing ROS production. Anticholinergic drugs should be avoided in senior patients due to the likelihood of age-related compromised in the cholinergic system. Cholinergic enhancers such as donepezil and galantamine are approved in the US for AD, but not commonly used in CDS veterinary patients. The first and still most popular therapeutic modality for CDS is dietary or supplements. The principle disease pathology is presumably a result of oxidative damage, and consequently many of the pharmacological agents are related to antioxidant effect. Vitamins B, E, and C, β-carotene, β-lipoic acid, flavonoids and carotenoids are contained and diets such as Canine b/d (Hills Pet Nutrition). Senelife (CEVA Animal Health) is comprised of antioxidant (ginko biloba, vitamins B6 and E, and resveratrol) and phosphatidylerine, a factor thought to improve neuronal membrane fluidity and functioning. Studies report cognitive enhancement in laboratory and client-owned dogs on Senelife and Canine b/d. There are no published studies in cats although Senelife is labeled for this species. Melatonin and omega-3 fatty acids may also have antioxidant effect. Omega-3 fatty acids anti-inflammatory and cell membrane health effects may be beneficial in CDS treatment as well. Nestle Purina has tested a blend of antioxidants, fish oil, and the arginine in improving feline cognitive testing. The company has added this blend to their Prime Plus line of Pro Plan Diets. Another Purina diet (Pro Plan Bright Minds and Veterinary Diets Neurocare) is formulated with medium chain triglycercides to increase ketone bodies as alternative source of energy as brain glucose metabolism wanes in senior patients. Additional dietary supplements mechanisms that have been tested and marketed include S-adenosyl-l-methionine (SAMe) (Novifit, Virbac; Zentonil, Vetoquinol), which is essential to major metabolic pathways and results in enhancing transmission of neurotransmitters and boosting endogenous antioxidants. Yet another compound - jellyfish protein, apoqequorin (Neutricks) - is a calcium buffer that may protect the cell against excitotoxic and ischemic events related to calcium and ion dysregulation in the aging brain. Behavior problems resulting from CDS can also be treated symptomatically along with most of the aforementioned supplementation or diets. As in pets without age-related cognitive deficits, on-going situational or generalized anxieties may be treated daily with most antidepressants such as fluoxetine, paroxetine, or the mild anxiolytic busiprone. Tricyclic antidepressants (e.g. clomipramine) are typically avoided due to the anticholinergic profile of this class. Use of antidepressants, opioids, and a few other medications (amitraz, trimethoprim sulfa) are contraindicated with selegline due to serotonin syndrome or neurologic side effect risk. Fast-acting anxioytics and sedating medications like the trazodone or gabapentin can be dosed situationally for predictable triggers or nighttime waking. Benzodiazepines are an option, but are known to increase cognitive decline in geriatric people. General calming nutraceuticals like l-Theanine (Anxitane, Virbac), α-casepepine (Zylkene, Vetoquinol), or non-systemic pheromone products (Adaptil and Feliway, CEVA Animal Health) can also be safe and useful layers to an anti-anxiety treatment plan in an aging pet.

References

Top Small Animal Behavior Tips and Myths Debunked
Julia Albright, DVM, DACVB
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Knoxville, TN

1. Dogs are not little wolves
Many myths and misconceptions abound regarding canine origins, social structure and communication. New studies of free-ranging dogs, canine cognition and comparative genomics provide insight into the real world of dogs. Dogs are descended from the gray wolf but are not little wolves as many pop culture “dog experts” strongly assert. The domestication process has drastically altered canine genetics resulting in a creature unique from any other canid species in terms of appearance, physiology, and behavior. Dominance hierarchies in dogs (and many domestic species) are a hotly debated topic. Behaviorists and ecologists do agree on the existence; however, observations of free-ranging and feral dogs reveal a fairly loose social structure. Both dogs and wolves appear to maintain hierarchies largely through submissive signaling, not physical confrontation. Therefore, when humans try to assert dominance through actions like “alpha-rolling” or physically forcing the dog down, we are not mimicking natural relationships, but likely scaring the dog, conditioning an aversion, and increasing the risk of a bite. Recommending clients try these tactics is dangerous. A structured human-animal relationship can successfully be formed through non-confrontational methods.

2. Dogs are not little people
The human-animal bond is equally at risk when we assume animals have human thought processes. A perfect example is the statement “she knew she shouldn’t have done it,” implying the dog is feeling guilty or remorseful after destruction, aggression or eliminating in the house. Animals do not have complex language that allows us to discuss the past and future with them. Learning and cognition research confirms that a consequence must occur within about 1 second if the consequence is to be paired with the animal’s action. Therefore, when someone arrives home to a guilty looking dog, and the dog did indeed eliminate in the house, it is understandable (but erroneous) to jump to the conclusion that the dog understands he did something wrong. In fact, evidence strongly suggests the dog is taking his cues entirely from his human’s current and previous reactions. In other words, last time there were feces on the carpet, the human became very angry. Now that there are feces on the carpet and the human has returned, there may be yelling and physical discomfort. The danger is then punishing the dog at this delayed stage. This doesn’t correct the undesirable action but only increases anxiety by making correction and the person unpredictable in the dog’s mind. If the undesirable behavior isn’t witness, it cannot be successfully rewarded or punished.

3. Look below the surface
Recognizing low-level stress signals is essential in keeping animals calm and averting aggression in many situations. Most of us humans do not recognize a dog or cat is distressed until we see overt body language of crouching, extreme tail tuck, refusal to move, eliminations, or growls and snarls/hissing. Therefore, many aggressive instances appear to be “out of nowhere.” In reality, the animal was likely displaying many forms of (to us) subtle behaviors indicating apprehension or fear. Lip licking, yawning, eye aversion, and slow or stiff body postures in dogs are signals we can learn to recognize, and then subsequently change the situation to keep the peace. For example, vet staff may try a different type of approach if the dog stiffens and licks her lips. Clients can be advised to remove the dog from room if she yawns and averts her eyes when kids are rough housing close to the dog resting on her dog bed. A cat bite could likely be avoided if the cat owner ceased petting or approaching a cat when he gave a tail lash, stiffened, or slightly moved away.

Furthermore, some common behaviors are assumed to have a specific motivation when the real reasoning is more complex. Mounting in dogs, for example, has long been attributed as a purely sexual or dominance-asserting action. However, closer observation of the context typically reveals that the animal was feeling stressed or frustrated in that moment. We common see this when one dog behaved inappropriately during play or a visitor is ignoring the dog. Self-directed scratching, excessive licking, or other repetitive behaviors can also fall into this category. Most people using some form of aversive correction to stop these behaviors but this may be self-defeating in the short or long run by increasing the stress in these situations. Redirection to a more appropriate behavior can both stop unwanted behavior and reduce stress.

4. It’s all in the approach
Many instances of human-directed aggression, especially in dogs, is a direct result of the way the human approaches the dog. For decades we have been taught to stick a hand out to let the dog smell and become accustomed to us. However, reaching out and over any dog is considered threatening to some degree. Simply being approached can make most dogs somewhat nervous. We should advocate in the vet clinic and in public education that allowing a dog to approach a person, and being aware of unthreatening human body postures—turning to side, kneeling down, avoiding any outward motions towards a nervous dog, and using food to lure—can be the difference between an aggressive and friendly encounter in any setting.
5. Training does not solve most behavior problems
Animal “training” is the process of changing the behaviors we can observe. Various techniques can be employed to motivate a change in activity and almost all of these techniques involve either learning how to gain a reward or avoid something unpleasant. Decades of extensive research demonstrates the efficacy of both categories. However, a poor emotional state or association with a person, place, or other environmental trigger motivates many behavior problems. Relying on typical “training” completely ignores this emotional basis, and, in fact, many aversive training techniques, which appear to successfully stop the unwanted behavior, do not solve the problem because the underlying association has not been improved.

The good news is by using simple associative learning (elicited so well by Pavlov’s dog), we can help clients improve many behavior problems without in-depth dog training and the high risk of side effects (e.g., increased fear, increased redirected aggression) associated with aversive tactics. For example, many dogs are reactive to other dogs while on leash walks and 50% cats show severe aggression when first introduced. By adopting some common sense safety protocols (e.g., head halters, baby gates, adequate distance) and providing food just BEFORE and DURING the interaction without any stern correction, the pet will start to associate the trigger with the reward. In essence this tactic addressing that core emotional motivation and not just the surface-level behavior.7

6. It’s YOUR license and reputation too
Critically assess any behavior professional to whom you refer your clients and beware of the unregulated nature of the dog training industry. There is no licensing, and “certification” terminology is often provided by for-profit entities whose programs do not undergo the scrutiny to the extent of veterinary certification program. Unfortunately terms like “behaviorist” and “specialist” are usually self-labeled. Attending a seminar does not make an expert. Likewise high-levels of success in dog sports, police, protection, search and rescue do not automatically indicate this individual has the knowledge base to identify emotion-based behavior problems. Although most trainers are ethical and many effective, you cannot know that from a brief conversation, marketing material, or even personal testimonials. Ask about continuing education attendance, your role in your patient’s care, and client safety for trainers that employee aversive tools (shock collars, chains, prong collars). Do not take specific medication recommendations from a trainer. All veterinary clinics should strongly consider providing additional behavioral education to a veterinary staff member, particularly a technician who can become specialized through Academy of Veterinary Technicians, as the behavior resource for clients. This allows information and revenue to stay within the practice.

7. “Socialization” is not an appropriate behavior modification technique
The socialization development period, which is between approximately 4-14 weeks in dogs and 2-7 weeks in cats, is the critical stage during which the neural system is primed to receive input regarding future social and environmental stimuli. Socialization is critical, evidenced by extreme fear and fear-related aggression in many poorly socialized animals. Unfortunately some people falsely assume that socialization of adult animals can solve existing behavior problems and put pets in dangerous situations for the sake of “socialization.” Dogs showing aggression to other dogs should not be indiscriminately exposed to unsuspecting dogs and people in dog parks, day care, or shopping areas. Not only is this not safe, but the tactic ignores the possibility of sensitization, or worsening of the negative emotion. Not all animals will become accustomed to any stimuli with repeated exposures. Solid foundation behaviors, safety measures and practice interpreting body language should be implemented before any type of public exposure. For example, the pet should master a redirection cue for a reward (watch me, leave it) in increasingly distracting situations prior to a walk through the pet store.

8. Early socialization and vaccines
Puppy and even kitten socialization is extremely important for a behaviorally healthy animal.8 Behavior problems are a factor in almost every case of rehoming or relinquishment to shelters. Encouraging clients to limit a young pet to environmental stimuli until all core vaccines are completed could be a mistake because 16 weeks is past the critical socialization period. Recent studies have shown puppies from diverse areas that received one or two rounds of vaccines and attended puppy socialization classes were no more likely to contract infectious diseases than those that did not attend a class. Of course ensuring other animals that been in the area are properly vaccinated, and that the facility is using proper biosafety standards is important. For this reason, reputable private facilities (including vet clinics!) are the best choice and public dog spaces should be avoided.

9. Avoidance IS a good behavior modification technique
Along the same lines as indiscriminate exposure to triggers possibly causing more problems, avoidance of triggers is an important step for veterinarians to recommend. Safety of our clients, patients, and the public is the top priority. Recommending dogs be exposed to triggers can be a serious public health concern and a legal liability. Assurance from the veterinarian that avoidance is not worsening the problem can provide a great comfort to the family and possibly be the difference between life and death for that pet. On an emotional and biological level, avoidance prevents the problem from worsening by keeping the animal in a calmer state and not strengthening the negative association. Exposure to the trigger can then occur on a gradual level.
10. Psychoactive medications as “the last resort?”

The goal of most psychoactive medication usage is to provide antianxiety effects through various mechanisms. The prevailing public sentiment is psychoactive medications should only be used as a “last resort.” Although the use of these drugs should not be taken lightly, early intervention of all behavioral therapies, including medications, can limit the damage and improve success. Most of us would not hesitate to institute pain medications or antibiotics early in the treatment of injury or disease, yet many of the same practitioners wouldn’t consider psychoactive medications until the problem is a very severe stage. Mental health should be considered part of overall health. You can decrease your clients’ fears that commitment to start medications is somehow a life-long commitment to keep the patient on medication for the rest of that animal’s life.

References
It is good medicine to choose antimicrobial regimens using the principles of judicious use. According to the FDA-CVM, judicious use of antimicrobial drug use is “an approach to maximize therapeutic efficacy and minimize selection of resistant microorganisms.” And while some of these principles require the results of a culture and susceptibility, many can be implemented when using empirical therapy.

**AVMA principles of judicious antimicrobial use**

Disease prevention strategies, such as appropriate husbandry and hygiene, routine health monitoring, and vaccination, should be included as part of a comprehensive animal/herd health plan.

Once disease has occurred, other management and intervention strategies may be considered prior to antimicrobial treatment.

Judicious use of antimicrobials should include appropriate veterinary oversight.

Extralabel use of antimicrobials must meet all the requirements of the veterinarian-client-patient relationship as defined in the AMDUCA amendments to the Federal Food, Drug, and Cosmetic Act and its regulations.

Extralabel use in food animals necessitates an extralabel withdrawal interval to be assigned by the attending veterinarian, on the basis of information on the species, dose, route, and frequency of treatment, in conjunction with available scientific pharmacokinetic data.

Antimicrobials requiring a prescription must be used only by, or under the order of, a licensed veterinarian. This should include a veterinarian-client-patient relationship.

A Veterinary Feed Directive must be issued only by a licensed veterinarian in the course of the veterinarian’s professional practice. This should include a veterinarian-client-patient relationship.

Accurate records of treatment and outcome should be maintained.

Antimicrobials should be used in animals only after careful review.

- Use narrow-spectrum antimicrobials whenever appropriate.
- Use microbial culture and antimicrobial susceptibility results to aid in the selection of antimicrobials when clinically relevant.
- Regimens for antimicrobial treatment, control, or prevention of disease should be based upon current scientific and clinical principles, such as microbiological and pharmacological tenets.
- Antimicrobial use should be confined to appropriate clinical indications. Inappropriate uses such as for uncomplicated viral infections should be avoided.
- To minimize selective pressure, therapeutic exposure to antimicrobials should be minimized by treating only for as long as needed for the desired clinical response.
- Limit therapeutic antimicrobial treatment to ill or at-risk animals, treating the fewest animals indicated.

Minimize environmental contamination with antimicrobials whenever possible.

**Tips for maximizing therapy**

- Whenever possible use a gram stain to further refine your antimicrobial drug choice.
- Think beyond the pathogen to find ways to alter the microenvironment, increase healing, reduce pain, and speed healing to decrease time to normal function and time on antimicrobials.
- Train staff in techniques of client adherence models so that your treatment plan is implemented correctly the first time.
- Don’t be afraid to write a prescription to get access to the therapeutic you need to treat your patient most effectively.
In order to get the most out of a culture and susceptibility a clinician should consider pathogen, patient, drug, and public factors when making a rational therapeutic decision.

**Pathogen factors**
The most important pathogen factor is correct identification of the bacterial pathogen. Culture and sensitivity is considered the gold standard for identification of pathogens. However, results from testing can take 3 to 5 days minimum to be reported. Often treatment must be initiated prior to having results. In that case, a clinician can use empirical knowledge to help select the appropriate antimicrobial. This includes knowledge of the anatomical location of the infection, possible sources of infection, common pathogens found in that disease process, and normal flora. Additionally, gram stains provide a basic identification method that helps narrow the scope of antimicrobial agents to be used.

Culture and sensitivity provides information on the minimum inhibitory concentration (MIC). By definition, an MIC is the minimum concentration of a drug needed to stop visible cell growth. It is specific for strain cultured from the patient relative to specific drugs. MIC’s are determined using serial dilution methods in test tubes. Alternatively, a zone of inhibition can be measured using disk diffusion methods (aka Kirby-Bauer technique) and then the zone is transformed into an equivalent MIC. It is imperative that you remember that MIC’s are in vitro designations and do not account for the patient response and so can underpredict the efficacy of some drugs. It is also important to remember that MIC information alone is not enough information to make a good antimicrobial drug choice. In order to assess the efficacy of a drug to a pathogen, you also need to know the breakpoint for that drug-pathogen complex. This number is considered the cut off MIC that is used to determine sensitivity of a bug to a drug. This measure takes into account the overall population of strains of bugs within a given species. It also is based on plasma/urine concentrations for a “reasonable” dose given. The actual breakpoint is set by committee in the Clinical Laboratory Standards Institute (CLSI). Practically, a pathogen with an MIC below breakpoint is considered to be sensitive to the drug. Knowledge of MIC and breakpoint allow us to compare the relative susceptibility of the pathogen to that reported in the literature, between drugs in our clinical patient, and development of resistance in our patients and hospitals.

**Drug factors**
Antimicrobials can be classified in a myriad of ways. Each has its benefit and utility. When choosing an appropriate antimicrobial agent it is important that you have a good understanding of a drugs mechanism of action. This can be broadly classified into drugs that inhibit cell wall growth, protein synthesis, DNA/RNA, and then others. These categories can be helpful in determining appropriate empirical therapy. Additionally, knowing the drug’s spectrum of action provides a more utilitarian approach for deciding most empirical therapy. Finally, drugs can be classified as bactericidal or bacteriostatic depending on whether normal concentrations kill or merely inhibit cell growth. This provides a useful classification system for choosing between drugs with a similar mechanism of action based on your patient’s needs. For example, a bactericidal drug should be used in immunocompromised patients since there would be a limited immune response to kill the bacteria which is essential to killing for a bacteriostatic drug. A single drug could be classified differently depending on the pathogen strain.

Drug factors are intrinsically linked to pathogen factors. We assume that there is a link between plasma concentrations and the pathogens response. This pharmacodynamic-pharmacokinetic link is classified as either time or concentration dependent killing. Time dependent killing requires that plasma concentrations remain above MIC for as long as possible. This is essential for bacteriostatic drugs and is also a common need for bactericidal drugs as well. Conversely, some bactericidal drugs exhibit concentration dependent killing. This requires peak plasma concentrations to be 4-10 times greater than MIC. These drugs often have a prominent post antibiotic effect such that killing continues long after drug concentrations fall below MIC levels. This is used to our advantage with drugs such as Aminoglycoside agents where killing is determined by peak concentrations and toxicity is determined by trough concentrations. Dosing regimens are designed to maximize killing (high peak concentrations) but take advantage of short half lives to reduce toxicity (trough levels of 0). These links are also dependent upon patient factors that alter the pharmacokinetics of the drugs.

**Patient factors**
When choosing an antimicrobial agent, it is important to consider patient factors. The health status of our patients can alter immune status, blood flow to/from sites of infection, and alter pharmacokinetic factors. Additionally, it is important to remember where the infection is located as there are anatomical barriers to sites such as the brain and eye that can make it difficult to reach therapeutic
concentrations. And you must take into account toxicity due to drugs that could be enhanced in certain disease conditions such as renal impairment.

Also within our patients it is important to consider the microenvironment of the site of infection. Alterations of pH, oxygen tension, and the presence of debris can significantly alter the efficacy of certain drugs. Additionally, the size of the bacteria inoculum is important. It is easier to have efficacy with a drug if the inoculum is relatively lower.

Public factors
Finally, it is important to remember our legal responsibilities are important factors in rational drug use. The FDA, USDA, and EPA are all valuable sources of information and are the place to report adverse drug effects. Under the Food, Drug, and Cosmetic Act, it is illegal to dispense out of date medication. You also must have a VPCR to prescribe a prescription drug. The Animal Drug Use Clarification Act recognizes the importance of extralabel drug use in veterinary medicine. However, you may not go off label if there is another drug approved for use in that species for that disease. Not having that drug in your pharmacy is not adequate justification for extralabel use. AMDUCA also requires specific medical records and labeling on dispensed products. The best way to increase compliance and to increase public support is to have drugs dispensed in appropriate containers with labels.

Maximizing treatment
Maximizing therapy is done by using the right drug, at the right dose, at the right time. In general, avoid use of drugs with MIC’s close to breakpoint values. If possible, choose an antimicrobial agent that is farthest away from the breakpoint value as it will have greater relative efficacy. And remember that MIC testing is in vitro and that breakpoints are almost always for plasma and not for urine concentrations.

No matter what therapeutic choice you make, it is important to have a clear understanding of the time frame needed to see a response. In most cases a clinical change should occur between 48 and 72 hours of initiating treatment. Alternatively, therapeutic drug monitoring can be implemented to check for therapeutic levels of drugs and alter dosing regiments appropriately.
Choosing Antimicrobials: Why Isn’t this Drug Working
Jennifer Buur, DVM, DACVCP
Western University of Health
Pomona, CA

Maximizing antimicrobial treatment
If you use the proper drug at the proper dose for the proper time you will achieve excellent patient care. However, in veterinary medicine the proper dose and proper duration are often unknown. In human medicine it has been shown that short duration therapy of 2 to 3 days is often enough to achieve clinical cure in uncomplicated disease such as UTI. However, no studies have been done in veterinary medicine. If you choose short term therapy, the optimal drug would be bactericidal with a rapid onset and few adverse effects. You will also be most effective is the pathogen inoculums are small such as immediately after a dog bite or post operatively after ovariohysterectomy in a pyometra.

Evaluation of our patients is also critical in maximizing treatment. Clinical change should occur after 48-72 hours. You can measure drug levels to confirm time or concentration above MIC or repeat cultures. Current standard of practice is a minimum of 7-10 day duration of treatment or 3 days past clinical cure. If no response has occurred in your patient, you should implement some form of therapeutic drug monitoring.

Principles of therapeutic drug monitoring (TDM)
Direct TDM – Measurement of drug concentrations at site of action or in easily accessible compartment with a known correlation to site of action (such as serum or plasma).

Indirect TDM – Use of measurements besides drug concentrations to determine appropriate clinical dosing regimens. These could include physiologic responses such as heart rate, enzymatic targets such as T4 levels, or response to treatment such as wound healing.

Alterations of pharmacokinetics can lead to adverse drug reaction including therapeutic failure and drug toxicity. Therapeutic failure can be due to alterations in PK from the disease, poor drug choice, and poor client compliance. It is important to take into account when assessing a clinical patient if the disease or concurrent medications will significantly alter the pharmacokinetics of the drug of interest. The gold standard for determining if a dosing regimen is correct is therapeutic drug monitoring. This information can lead us to change the drug, the formulation or route of administration, the dose, or the interval of drug given.

Samples for TDM should ideally be taken when drug concentrations are at steady state. This is generally occurs once 5 half lives have passed. Ideally both peak (2 to 4 hours post administration) and trough (prior to next dose) samples should be taken. With these samples, individual pharmacokinetic parameters can be estimated for the patient and dosing regimens altered. This is very important if the half life of the drug is less than the dosing interval. For drugs with long half lives, a single sample can be taken. Peak samples look for toxicity and trough samples look for therapeutic failure.
Creating safe and effective dosing regimens for patients with chronic disease can be challenging depending on the therapeutic and chronic disease. However with a few guided questions, you can make more informed drug choices and determine if when and how a dosing regimen needs to be altered.

**Practical pharmacokinetics**

Pharmacokinetics is defined as the study of the movement of drugs in the body, including the processes of absorption, distribution, biotransformation, and excretion. As clinicians, our understanding of pharmacokinetics and the underlying physiological mechanisms that govern pharmacokinetics is essential in determining and altering drug dosing regimens to provide appropriate care for our patients.

**Absorption**

Absorption is the process whereby xenobiotics are moved from the site of administration into systemic blood circulation. In most cases, this requires drugs to transfer across membranes via passive transfer. The rate and amount of drug that is able to cross a membrane is dependent upon many variables. These include physiochemical properties of the drug (size, shape, pKa, lipid solubility), physiological characteristics of the membrane (surface area, thickness), and environmental factors (pH, solubility in the matrix, binding to proteins or other adsorptive materials, concentration gradient). In general, in order for a drug to be absorbed from any site of administration, it must be in solution, non-ionized, not bound by proteins or particulate matter, and lipid soluble.

Oral absorption varies between drugs and species due to the wide range of anatomy, gastric transit times, differing pH’s of gastric millue, presence/absence of feed particles, chelation, and presence of normal microbial flora. Additionally, there are specific transport proteins including the p-glycoprotein pumps that actively remove substances from the blood back into the gastric lumen. Disease states can also alter any of the normal physiology and thus alter both rate of absorption and bioavailability. For example, inflammatory bowel disease can increase the thickness of the bowel wall and limit both rate and extent of absorption. Shock decreases blood flow to the GI track and limit absorption by altering ΔC. Besides disease states, concurrent drug therapy can also alter physiology. Ketoconazole inhibits p-glycoprotein pumps and can increase extent of absorption. Changes in pH are also common with concurrent drug therapy.

**Distribution**

Distribution can be thought of as the absorption of drug from the systemic blood into tissues. Blood flow:mass ratios of organs, protein binding, tissue binding, and anatomic barriers are important considerations. Organs with a high blood flow:mass ratio tend to have higher concentrations of drug within the tissues than those organs with lower ratios. Binding of drugs, whether to plasma proteins or to tissue proteins, limits distribution since only unbound drug is available to distribution, metabolism, or elimination. Drug bound to tissue proteins form depots that will prolong both tissue and plasma concentrations. This is important if the patient is a food producing animal as prolonged tissue residues can constitute a public health concern. Additionally, some organs have increased barriers to distribution which can be increased layers of membrane (i.e. glial cells within the blood brain barrier) or efflux pumps such as p-glycoprotein pumps. These include the brain, testes, prostate, synovium, placenta, and eye. These compartments may also have different pH’s which can further alter distribution.

Volume of distribution (Vd) is the pharmacokinetic parameter used to describe the extent of tissue distribution. This measure is an apparent measure and has no biological or physiological basis. It is merely the ratio of dose to plasma concentration. A large Vd implies that not a lot of drug remains within the blood. A low Vd implies that most of the drug remains within the blood. This term is useful in calculating dosing regimens.

Disease states can alter distribution in much the same way as it changes absorption properties. Additionally, it can alter the amount of albumin (most common plasma binding protein). It can also alter water balance and alter extracellular fluid balance. This can lead to depot formation (if the drug partitions into excess ECF) or increase plasma concentrations if ECF is severely decreased. Disease also alters blood flow. That changes the blood flow:mass ratios and thus can alter distribution. Drug interactions can also influence distribution by altering pH, blood flow, and protein binding capacity.

**Metabolism**

Metabolism is the alteration of a xenobiotic within the biological system. Metabolism occurs in many tissues. Primary organs for metabolism include the liver and kidney. However, lungs and skin also can contribute to the metabolism of specific drugs.
Metabolism is often described as either Phase I or Phase II. Phase I generally involves cytochrome P450 enzymes (along with others) that add functional groups onto molecules. These functional groups are then used in Phase II metabolism for the addition of endogenous compounds. The purpose of metabolism is to increase polarity and water solubility thus limiting further distribution and increasing elimination via glomerular filtration. There are large differences between species in their abilities to do certain phase I and phase II reactions. This is most noticeable in Phase II reactions. For example, cats do not glucuronidate well while dogs do not acetylate well. These limitations can make extrapolation of dose between species difficult. Age can also alter a patient’s ability to metabolize compounds. Older animals generally have less metabolic capacity. Also neonates do not have the developed enzymes required for these reactions. In both cases, normal dosing regimens may cause increased plasma concentration levels. Genetic predisposition can also alter the relative concentrations of these enzymes within a species or within a breed.

Disease can alter blood flow to the major metabolic organs. This can decrease the metabolism rate and thus increase plasma concentrations. Additionally, concurrent drug administration can both induce and inhibit major metabolic enzymes. For example, Phenobarbital administration induces P450 enzymes that can lead to an increase in its own metabolism as well as that of other drugs. This can lead to therapeutic failure due to low plasma concentrations.

Elimination
The final aspect of pharmacokinetics is elimination. Elimination is simply the drug leaving the body. This could be due to metabolism, renal, hepatic, pulmonary, sweat mechanisms. Clearance (CL) is the term that encompasses both metabolism and elimination. It is defined as the amount of blood cleared of drug per unit time. Clearance is an additive process where total body clearance (CLb) is the sum of all clearance mechanisms within the body. As such, it can be mathematically expressed as CLb = CLh + CLr + CLo where b, h, r, and o are body, hepatic, renal, and other respectively. Additionally, within each organ you can describe CL as the sum of the mechanisms. In the liver, CLh is the sum of intrinsic clearance due to metabolism and elimination via the biliary pathway. Renal clearance is the sum of glomerular filtration and tubular secretion minus the reabsorption.

\[
CLh = Clint + CLbil
\]
\[
CLr = Filtration + secretion – reabsorption
\]

Disease states can alter blood flow to the elimination organs and thus reduce or enhance elimination. Additionally, disease can alter organ function and thus inhibit active elimination processes. Drug therapy can also alter pH of elimination matrices such as urine and thus alter reabsorption. This is used therapeutically to increase urine antimicrobial concentrations.

Therapeutic drug monitoring (TDM)
Alterations of pharmacokinetics can lead to adverse drug reaction including therapeutic failure and drug toxicity. Therapeutic failure can be due to alterations in PK from the disease, poor drug choice, and poor client compliance. It is important to take into account when assessing a clinical patient if the disease or concurrent medications will significantly alter the pharmacokinetics of the drug of interest. The gold standard for determining if a dosing regimen is correct is therapeutic drug monitoring. This information can lead us to change the drug, the formulation or route of administration, the dose, or the interval of drug given.

Samples for TDM should ideally be taken when drug concentrations are at steady state. This is generally occurs once 5 half lives have passed. Ideally both peak (2 to 4 hours post administration) and trough (prior to next dose) samples should be taken. With these samples, individual pharmacokinetic parameters can be estimated for the patient and dosing regimens altered. This is very important if the half life of the drug is less than the dosing interval. For drugs with long half lives, a single sample can be taken. Peak samples look for toxicity and trough samples look for therapeutic failure.

Practically, dosing regimens can be altered by altering the amount or dose of drug or by altering the interval in which a drug is given using the formula below.

\[
\text{New Dose} = \frac{\text{Old Dose} \times \text{Target Cp}}{\text{Observed Cp}}
\]

\[
\text{New Interval} = \frac{\text{Old Interval} \times \text{Observed Cp}}{\text{Target Cp}}
\]
Flow charts and other forms of decision trees are often used to help clinicians make therapeutic decisions in the context of complicated diseases such as mast cell neoplasia or chronic kidney disease. But these types of tools can be used to promote a more thorough and rational choice of therapeutics in any circumstance.

**Tool #1**
The steps of Evidenced Based Drug Use are:
- What’s the diagnosis?
- What are the treatment options?
- What drug?
- Which formulation?
- What dosing regimen? How long?
- How/when will I know it is effective?
- How/when will I know if there is a problem?

**Tool #2**
Maximizing patient care does not end when you choose a treatment option. Optimizing care requires the veterinary team to follow up with patients and monitor response to treatment. The most important questions to ask prior to implementing any treatment plan are:

1. How and when will I know if the treatment plan is working?
2. How and when will I know if there is an adverse effect?

These questions are the basis for good therapeutic drug monitoring (TDM) programs. TDM can be implemented in a variety of ways depending on the needs of the patient. Interpretation of TDM results can lead to earlier modification of treatment plans and more effective therapy. Thus, TDM is essential for maximizing patient care.

Types of Therapeutic Drug Monitoring (TDM)
Therapeutic drug monitoring plans can range from monitoring response to treatment to the direct measurement of drug concentrations in the tissues of interest. In general, TDM is divided into direct and indirect measurements.

Direct TDM is the classic procedure of measuring the molecule of interest in the tissue of interest. Because taking tissue biopsies is invasive, a surrogate tissue (blood, plasma, or serum) is generally collected instead. When choosing this type of TDM it is important to remember:

1. Contact the laboratory that will be performing the analysis to make sure you are collecting the correct volume needed and that it is collected and stored in the appropriate containers. Some drugs of interest, like phenobarbital, adhere to serum separator barriers or other chemicals and thus inappropriate collection and storage can impact drug analysis and interpretation.
2. Ship samples using laboratory recommendations to prevent degradation of the molecule of interest.
3. Provide the clinical pharmacologist with as much information as possible about treatment history, concern, and timing of samples so that interpretation can be as accurate and complete as possible.
4. The laboratory should ideally measure the active form of the molecule and any active metabolites in order to provide an accurate picture.
5. The laboratory should use validated methods for the molecule of interest in the species of interest and should routinely perform quality assurance protocols.
6. Interpretation of these numbers requires a known pharmacokinetic-pharmacodynamic relationship and therapeutic window for the disease of interest.

Indirect TDM is more common. This form of TDM looks at surrogate markers rather than the specific molecule of interest. This can be measurement of other biological molecules (blood glucose levels after insulin administration), physiologic changes (heart rate after administration of a beta1 antagonist), or behavior changes (sleeping comfortably after administration of analgesic agent). Response to treatment is a longer term form of indirect TDM. Indirect TDM can provide both quantitative (glucose levels) and qualitative (sleeping) results. Making changes to the treatment plan based on qualitative results can be easy if the response is quick and easy to detect (inhalant anesthetic gasses) or trickier if response can be delayed or hard to detect (behavior modification drugs). However indirect TDM, however tricky, is an important tool to maximize patient care.

Sampling Schedules
To maximize TDM results, it is important to know what samples to take and when to take them. Peak samples occur 2-4 hours after oral administration, immediately after IV administration, and after 1-2 hours for SQ or IM administration. Trough samples are taken right before administration of a dose. While taking both peak and trough samples is the gold standard and can be used to determine pharmacokinetic properties of that molecule in a specific patient, TDM is most often accomplished with single samples. Which sample or samples to draw depends on the goals of TDM.

Initial treatments, with or without loading doses, can be monitored with single samples. Patients should be at “steady state” conditions (3-5 drug half-lives). If the drug has a long half-life (phenobarbital), a single sample taken at any time will be sufficient since plasma concentrations do not change much during a dosing interval. Drugs with short half-lives require either a peak (if concerned with toxicity) or trough (if concerned with efficacy) sample. Loading doses should be monitored at peak after loading dose administration.
and after the first half-life of the maintenance dose. If the concentrations are the same, a final check after “steady state” has been reached will confirm the dosing regimen. If the concentrations do not match, adjustment of maintenance dose should be initiated.

Maintenance dosing regimens are monitored by single samples taken at peak (if concerned about toxicity) or trough (if concerned about efficacy) at 6-12 month intervals (well controlled patients) or 3 month intervals (poorly controlled patients) for most drugs.

Investigation of adverse effects is more complicated. Single samples can confirm toxicity (peak) or lack of efficacy (trough). But alterations in pharmacokinetics due to disease, drug-drug interactions, or high interindividual variability generally require sampling at both peak and trough concentrations. If drugs have an extremely short half-life, trough concentrations may be too low to quantify. Thus sampling should be done at peak and 2-4 half-lives later.

**Interpreting Results**

Single samples can be interpreted only if a therapeutic window is known for that drug with that disease in that species. Many veterinary therapeutic windows are extrapolated from human data and may or may not reflect the physiological differences between the species. Like any laboratory value, therapeutic ranges are population-based statistics. It is important to remember that you are treating the patient and not the laboratory result. If adjustment to dosing regimens are required, another TDM plan is required to monitor the response to this change.

Pharmacokinetic parameters can be calculated based on multiple sampling protocols. These values are then used to determine patient specific dosing regimens. This is useful when a patient experiences a change in chronic therapy (induction of metabolism enzymes leading to lack of effect), if the drug has a narrow therapeutic window such that individual variability can lead to adverse effects (aminoglycosides), or if the patient is critical and therapeutic levels must be reached as soon as possible. In most instances a clinical pharmacologist is extremely helpful in calculating the parameters and helping clinicians to implement the results.

Alternatively, dosing regimens can be proportionally adjusted based on the target concentration. Both dose and interval can be adjusted based on easy to use calculations.

Practically, dosing regimens can be altered by altering the amount or dose of drug or by altering the interval in which a drug is given using the formula below. Target and observed refer to plasma concentrations of either direct TDM molecules or surrogate molecules from indirect TDM.

\[
\text{New Dose} = \text{Old Dose} \times \frac{\text{Target}}{\text{Observed}}
\]

\[
\text{New Interval} = \text{Old Interval} \times \frac{\text{Observed}}{\text{Target}}
\]
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 CPR 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 CPR. 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 CPR include intravenous fluids, narcotic reversal agents, vasopressors, vaso-glycotics, anti-arrhythmics, 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 CPR 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 CPR 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 CPR, 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 CPR 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 CPR 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 CPR 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 by passing 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, CPR 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 biphase 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 biphase defibrillator, the pediatric settings should be used (2.4 J/kg). 7

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. 7

Open chest CPR

There are a number of absolute indications for open chest CPR. 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 CPR. Some advocate open chest CPR immediately in large breed dogs because of the limited success of restoring adequate circulation with external compressions while others prefer to perform external CPR for 5 minutes and then open the chest if there is little or no evidence of effective circulation. Open chest CPR 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

### Table 1. Drugs commonly used in CPR

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38
Vasopressin
(20 u/ml)

| Vasopressin (0.4 mg/ml) | 0.8 u/kg | 0.2 | 0.4 | 0.6 | 0.8 | 1 | 1.2 | 1.4 | 1.6 | 1.8 | 2 |

External Defibrillation
3-5 J/kg

| 50 | 100 | 150 | 200 | 200 | 300 | 300 | 300 | 360 |

Note: Atropine, epinephrine, lidocaine, amiodarone, and naloxone may all be approximated using the rule of thumb 1 ml/20 lb.

References


2005 American Heart Association Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2005;112(suppl.)

Emergency Management of Pericardial Effusion

Ari Jutkowitz, VMD, DACVECC
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Pericardial effusion is defined as the accumulation of fluid within the pericardial space. As the pressure within the pericardial space increases, right sided cardiac filling is impaired, resulting in decreased stroke volume with subsequent decreases in cardiac output and ultimately decreased oxygen delivery to the tissues (shock). These manifestations of pericardial effusion are referred to as cardiac tamponade. Successful emergency management of dogs with life threatening pericardial effusion depends on early triage, a thorough physical examination, point of care diagnostic imaging techniques, and subsequent pericardiocentesis or placement of an indwelling pericardial drain.

Key etiologic and pathophysiologic points

Pericardial fluid accumulation and cardiac tamponade in the dog most often occurs secondary to a neoplastic process. Hemangiosarcoma (HSA) is most commonly identified in the region of the right atrium or right atrial appendage while chemodectoma (common in brachycephalic breeds) is most often identified at the heart base. Mesothelioma and any metastatic tumor are additional neoplastic causes. Although location and breed are frequently suggestive of tumor type, definitive diagnosis is dependent on a biopsy specimen.

Idiopathic pericardial effusion tends to be an inflammatory process and is frequently recognized in similar breeds to those that frequently develop HSA. Significant efforts in recent years have been directed towards developing diagnostic tests to help differentiate malignant from benign pericardial effusion (idiopathic). Pericardial fluid pH was initially thought to aid in making this differentiation, however, pericardial fluid pH has now been clearly shown to be of little diagnostic value. Recent evidence suggests that blood concentrations of cardiac troponin I (cTnI) are significantly higher in dogs with masses consistent with HSA than in dogs without evidence of an underlying cause (idiopathic).

Vitamin K antagonists (anticoagulant rodenticides and coumadin) can also result in pericardial effusion. Therefore, it is the authors’ practice to always perform an ACT or other point-of-care coagulation assessment at the cage side prior to pericardiocentesis. If significant coagulopathy is present and patient condition permits, correction of coagulopathy with blood products (fresh frozen plasma or fresh whole blood) is indicated prior to pericardiocentesis. Subsequent institution of Vitamin K therapy for 4 weeks is indicated.

Left atrial tear is an uncommon consequence of chronic mitral regurgitation and left atrial dilatation, however, it has been recognized as a cause of acute pericardial effusion in the dog. An infectious cause of pericardial effusion is fungal disease (coccidiomycosis). Bacterial pericarditis and pericardial effusion secondary to trauma also occur, but are uncommon.

Numerous additional conditions such as congestive heart failure, uremia, decreased oncotic pressure, and a host of systemic inflammatory processes frequently result in small volume pericardial effusion accumulations without evidence of cardiac tamponade.

Key clinical diagnostic points

Triage and physical examination in pericardial effusion

The most common presenting complaints from the owners of dogs with pericardial effusion and cardiac tamponade are lethargy, anorexia, collapse or syncope, abdominal distention, and dyspnea. Major body systems assessment of the dog with pericardial effusion will likely reveal compromise to one or all of the major body systems. Assessment of the cardiovascular system may frequently reveal the following:

- Pale mucous membranes: due to vasoconstriction and poor peripheral perfusion
- Slow CRT: due to decreases in cardiac output
- Increased heart rate: due to compensatory activation of the sympathetic nervous system
- Poor pulse quality: due to decreased stroke volumes and low blood pressure

Assessment of the respiratory system will frequently reveal increased respiratory rate and effort.

Assessment of the central nervous system will frequently reveal a decreased level of consciousness secondary to decreased oxygen delivery to the brain. Any one or combination of these findings should necessitate movement to the treatment area for further assessment including full physical examination, measurement of blood pressure, oxygen saturation, cardiac rhythm (ECG), and placement of an intravenous catheter from which a small blood sample for PCV / TS / Blood Glucose +/- venous blood gas and electrolytes can be rapidly acquired. If possible, blood for CBC, serum biochemical profile, and coagulation profile or ACT should also be collected. Concurrently, a second team member will be able to collect a full medical history.

Physical examination should still be centered on the major body systems, but subtle findings supportive of pericardial effusion may be noted including:

- Jugular venous distention: due to right sided congestive heart failure.
- Muffled heart sounds normal lung sounds: unlike pleural effusion which will frequently cause decreased heart and lung sounds, pericardial effusion will frequently only cause decreased heart sounds.
- Abdominal distention: ascites and hepatic engorgement may result from longstanding (days) pericardial effusion due to right sided congestive heart failure. Abdominocentesis will frequently reveal a relatively clear fluid will low cellularity and a protein concentration greater than 2.5g/dL but less than 3.5g/dL most consistent with a modified transudate.
- Pulsus paradoxus: An inspiratory fall of arterial systolic blood pressure of more than 10mmHg resulting in variation in pulse intensity with respiratory cycle due to increased venous return during inspiration, increased right sided filling, shifting of the interventricular septum to the left with decreased left sided diastolic filling and subsequent decreased left sided stroke volume.²
- Other physical examination findings specific to the underlying cause of the effusion such as fever in septic or fungal pericarditis.

Pericardial effusion causing cardiac tamponade should be HIGHLY suspected based on signalment, history, and physical examination findings, supported by diagnostic testing such as abdominocentesis and electrocardiography (+/− radiography) and confirmed through point of care diagnostic imaging techniques.

**Diagnostic techniques**

**Abdominocentesis**

See above.

**Electrocardiography**

Assessment of ECG in patients with pericardial effusion may reveal sinus tachycardia +/- ventricular arrhythmias. Ventricular arrhythmias may result from decreased myocardial oxygen delivery or aberrant conduction associated with the underlying cause of the effusion. QRS complexes <1mV in amplitude and the presence of electrical alternans (regular or irregular variation in QRS complex amplitude associated with the heart moving within the pericardium to and from the positive pole of lead II) are supportive of pericardial effusion.⁴

**Echocardiogram**

Echocardiogram is the diagnostic test of choice for confirmation of the presence of pericardial effusion in the dog. Many dogs with pericardial effusion have SEVERE cardiovascular compromise and can be on the verge of death. The stresses associated with radiographic imaging may put cause these patients to decompensate. Consequently, in the ideal world, radiographic imaging should be avoided initially. The authors have found that the presence of a small, portable ultrasound machine with a mid-range frequency transducer placed at the primary treatment station in the emergency room / treatment area to be of great utility for identifying conditions like pericardial effusion, pleural effusion, and to assess patients with acute abdomen for the presence of abdominal fluid. Echocardiographically, pericardial effusion appears as a hypoechoic space located between the hyperechoic pericardium and the right ventricular wall when viewed through the right cardiac notch. The presence of pericardial effusion provides excellent contrast to aid in the diagnosis of cardiac masses, however, pericardiocentesis should NOT be delayed in a patient with signs of shock simply to aid the diagnosis.

**Thoracic radiography**

As previously mentioned, thoracic radiography can be an extremely stressful procedure for dogs with cardiac tamponade. However, not all practices are equipped with ultrasound capabilities. If thoracic radiography is performed in dogs with suspected pericardial effusion, ventrodorsal positioning should be avoided. A dorsoventral projection can be acquired with minimal stress. Lateral thoracic radiographs may also be performed. Supportive radiographic findings include an enlarged, globoid cardiac silhouette. Acute effusions may not cause severe enlargement of the cardiac silhouette because the pericardium has not had time to stretch. Concurrent pleural effusion may be present. The other primary differential for a globoid heart is dilated cardiomyopathy (DCM) or other underlying cardiac disease. Key findings to try to differentiate DCM from pericardial effusion include:

- **Heart sounds**: Heart sounds in dogs with DCM are frequently normal in contrast to the decreased heart sounds seen in pericardial effusion. A systolic murmur may be noted in dogs with DCM and is uncommon in dogs with pericardial effusion.
- **ECG**: Atrial fibrillation is common in dogs with DCM. Atrial fibrillation is uncommon in dogs with pericardial effusion. Electrical alternans may be seen in dogs with pericardial effusion.⁴
- **Cardiac Silhouette**: The silhouette of the heart on thoracic radiographs of dogs with pericardial effusion tends to be extremely round with sharp borders. The silhouette of the heart in dogs with cardiomyopathy can be round, but often, there are still some dimples or “waist” associated with the divisions between the chambers and the borders of the cardiac silhouette tend not to be as sharp because of motion artifact.
- **Pulmonary infiltrate**: Pulmonary edema is common in DCM and uncommon in pericardial effusion.
- **Pulsus paradoxus**: Common in pericardial effusion, uncommon in DCM.
Key therapeutic points
Pericardiocentesis
Pericardiocentesis can be a stressful procedure. Use of cardiovascularly sparing sedatives (narcotics and benzodiazepines) may alleviate patient stress and facilitate safe pericardiocentesis. Numerous techniques have been described for pericardiocentesis in the dog including, but not limited to the use of a large-gauge over-the-needle catheter, through the needle catheter, and catheters placed using the Seldinger technique. Numerous commercial pericardiocentesis trays / kits are also available. The authors prefer to use a 14-16g, 5.5” over-the-needle catheter (Abbocath T, Hospira Inc. Lake Forest, IL) with two additional small side-holes or a commercial multi-lumen intravenous catheter placed using the Seldinger technique (Arrow Triple Lumen Central Venous Catheter, Arrow International, Reading, PA). The former is much less expensive while the latter may be left in place for ongoing drainage.

Pericardiocentesis is the keys to the rapid identification of pericardial effusion in the dog. Rapid identification of problems and triage and careful attention to physical examination findings supported by ancillary diagnostic tests and point of care diagnostic imaging are the keys to the rapid identification of pericardial effusion in the dog. Rapid identification of problems and institution of treatment will maximize the likelihood of a positive outcome.

Monitoring
Patient response to decompression of significant pericardial effusion is often very rapid and very gratifying as vital signs and physical examination findings improve dramatically. Monitoring for recurrence of fluid accumulation by frequent reassessment of major body systems, physical examination and echocardiography is useful. Placement of a central venous catheter and monitoring of central venous pressure can also be a useful technique in that re-accumulation of pericardial fluid will result in a rise in central venous pressure.

Key prognostic points
Prognosis for dogs with pericardial effusion will depend on the underlying cause of the disease. Surgical removal of a mass on the right atrial appendage will at least temporarily alleviate signs of recurrent pericardial effusion. Surgical removal of right atrial / appendage HSA followed by chemotherapy will prolong life in dogs with pericardial effusion. Pericardectomy will temporarily palliate clinical signs of pericardial effusion for most neoplastic processes, and will most often be curative for idiopathic pericardial effusion. Thoracoscopic pericardectomy or creation of a pericardial window may have similar effects. Treatment with fresh frozen plasma, vitamin K1, and pericardiocentesis will be curative for dogs with anticoagulant rodenticide intoxication. Culture and sensitivity based antimicrobial therapy +/- surgical debridement is indicated for the management of infectious pericarditis. Dogs with left atrial tear secondary to chronic mitral valve regurgitation and left atrial dilation carry a guarded prognosis. Surgical repair of such a lesion has been described.

Summary
Triage and careful attention to physical examination findings supported by ancillary diagnostic tests and point of care diagnostic imaging are the keys to the rapid identification of pericardial effusion in the dog. Rapid identification of problems and institution of treatment will maximize the likelihood of a positive outcome.

References/Suggested reading
GDV and the Other Organ Torsions
Ari Jutkowitz, VMD, DACVECC
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East Lansing, MI

Background information, risk factors, and pathophysiology
GDV refers to the progressive dilatation and rotation of the stomach resulting in a variety of physiologic alterations 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). Much research interest has focused on identification of risk factors for GDV such that prophylactic measures may be instituted to try to prevent 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 relatives with a history of GDV.
- Faster speed of eating
- Raised feeding bowl
- Stress

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 / major body systems assessment 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. Septic can also contribute to some of the clinical signs seen in long-standing GDV. It is important to note that just as there is hypovolemia induced decreased oxygen delivery to the systemic tissues, so too is there decreased oxygen delivery to the heart itself via the coronary circulation. Decreased coronary flow during the shock state may be one of the instigators of the ventricular arrhythmias frequently identified 12-26hrs after onset of GDV.

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 (decreased oxygen delivery to the tissues due to inadequate circulating volume). 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 be avoided due to the decreased venous return from the caudal vena cava seen in dogs with GDV. 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 major body systems 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 orogastric 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 orogastric intubation (often requiring sedation) and the risk of esophageal or gastric injury. During lavage, aspiration pneumonia is a significant risk. 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. A mouth gag is required (2-3inch PVC tubing works well) to prevent trauma to your orogastric tube. Once the stomach is entered and the gas decompressed, lavage of the stomach with warm water is indicated. The author prefers to endotracheally intubate the patient if gastric lavage is to be 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)/ Lidocaine1 (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 hemorrhage, resection of areas of necrosis or suspected necrosis, splenectomy if indicated, and finally gastroscopy. It is crucial to perform a complete abdominal exploratory in dogs with GDV and the abdominal incision should extend from xyphoid 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 (the outermost layer being inverting). If a surgical stapling device is used for gastric resection, the staple line should be oversewn with an inverting pattern. 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). The authors prefer the incisional gastropexy in which an incision in the seromuscular layer of the stomach is sewn to an incision in the right body wall 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. Synthetic colloids are indicated at 20ml/Kg/24hrs in cases in which a hypovolemic state is present and clinical signs such as hypotension or peripheral edema are developing. 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 160bpm, 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 hrs 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 abdominocenteses and cytologic evaluation.
The final common postoperative complication of GDV is decreased gastric motility. The authors find this 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 (often gleaned from the venous blood gas). In one large scale study, 98% of dogs without gastric necrosis survived and only 66% of those with gastric necrosis survived. These were the highest survival statistics reported to date. 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. 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**

Immune-Mediated Anemia: Current Perspectives and Emerging Therapies

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Immune-mediated hemolytic anemia (IMHA) is one of the most common hematologic diseases seen in dogs with a reported mortality rate that ranges between 29% and 77% in the veterinary literature.\(^1\)\(^2\) Hemolysis results from the binding of immunoglobulins to red blood cell surface antigens, causing those cells to be lysed by complement intravascularly or removed from circulation by mononuclear phagocytes. IMHA may be a particularly frustrating disease for both owners and clinicians because of its waxing and waning clinical course, the potential for sudden complications, and the expense associated with treatment.

Immunemediated red blood cell destruction may be classified in a number of ways. Primary or idiopathic autoimmune hemolytic anemia (AIHA) refers to immune-mediated hemolysis in the absence of an identifiable trigger factor, whereas secondary IMHA results from an underlying process such as neoplasia, infectious disease, or drug reaction. IMHA may also be categorized based on whether it results in intravascular or extravascular hemolysis. Intravascular hemolysis results from the lysis of red blood cells by complement within the vasculature, and may be identified by the presence of free hemoglobin within the plasma and urine. Extravascular hemolysis results when there are insufficient antibodies present to cause complement fixation, and antibody-labeled red blood cells are removed by the reticuloendothelial system within the spleen and liver. Extravascular hemolysis tends to be a more gradual process and may be identified by the presence of bilirubin, rather than hemoglobin within the plasma and urine. IMHA may also be classified based on the presence or absence of autoagglutination. Autoagglutination is the spontaneous clumping of red blood cells and results from the cross-linking of erythrocytes by large numbers of antibodies. We have noted autoagglutination in approximately 70% of dogs treated for IMHA.\(^3\)\(^4\)

IMHA is typically a disease of middle-aged to older pets. As with other types of immune mediated disease, a female gender predisposition has been reported. At Michigan State University in the past eight years, approximately 2/3 of IMHA cases were seen in female dogs.\(^3\)\(^4\) Although any breed may develop IMHA, a number of breed predispositions have also been reported and include Cocker Spaniels, Poodles, Shih Tzus, Lhasas, Old English Sheepdogs, Border Collies, and Springer Spaniels. A seasonal predilection has also been suspected, as some studies have observed a larger number of cases presenting in spring and summer months. This may be a result of increased exposure to outdoor allergens or antigenic stimulation, or may simply reflect the overall increase in patient admissions seen during these months.

Clinical signs of IMHA may be acute or chronic, depending upon the rate of hemolysis. With chronic disease, symptoms such as lethargy, weakness, inappetence, vomiting, diarrhea, and pigmenturia are most commonly reported, whereas with more rapid hemolysis, acute collapse may be the first symptom noted. It is not uncommon for dogs to be brought in for “possible urinary tract infection” because the owners have noted discoloration of the urine with hemoglobin or bilirubin. Symptoms related to anemia, including tachycardia, tachypnea, and systolic ejection murmurs may also be noted on physical exam. Hepatosplenomegaly is not unusual as these organs are common sites for extramedullary hematopoiesis as well as clearance of antibody labeled erythrocytes. Fevers are frequently seen as a result of release of endogenous pyrogens like IL-1 and IL-8. Reactive lymphadenopathies may also be seen.

Initial in-house diagnostics should include PCV/TS, blood smear, and slide agglutination test, as these are inexpensive, easy to perform, and will frequently provide a great deal of information about the cause of the anemia. The importance of interpreting the PCV in conjunction with the total solids (TS) cannot be overemphasized. If the PCV and TS are both low, blood loss (rather than hemolysis) should be suspected. In contrast, a low PCV with a normal TS 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 hemolysis. Blood smears may also be useful in differentiating hemolysis from decreased production anemia, as the presence of significant polychromasia and anisocytosis indicates the presence of a regenerative response. Blood smears should also be evaluated for blood parasites and telltale alterations in red blood cell morphology. 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. Ghost cells, which appear as “empty” cell membranes may be seen with intravascular hemolysis. 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 several drops of saline. Autoagglutination may be evidenced by the observation of obvious flecks within the drop of blood. 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.

A number of other diagnostics should be considered in the evaluation of animals suspected to have IMHA. CBC, chemistry, and urinalysis should be run as part of a minimum database. The presence of hemoglobinemia/hemoglobinuria or
bilerubinemia/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 from tissue damage secondary to hypoxia and thrombosis. White blood cell counts in excess of 45,000/µl have been associated with a more guarded prognosis. 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. Reticulocyte count should always be performed to assess regenerative response. Immune-mediated hemolytic anemias are typically strongly regenerative, though it may take three days for regenerative response to be noted. Non-regenerative anemias should prompt suspicion of red cell aplasia, precursor-directed immune-mediated anemia (PIMA), or other form of decreased production anemia. 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 antisemur 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.

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. Sulfad drugs, penicillins, and cephalosporins may also cause IMHA by acting as hapten, substances that become adsorbed to erythrocyte membranes. If these hapten are targeted by the immune system, the entire red blood cell may be destroyed. Neoplasias such as hemangiosarcoma, lymphoma, myeloproliferative diseases, and hemophagocytic histiocytosis 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 tick-borne illnesses such as Ehrlichiosis and Babesiosis.

Treatment of IMHA consists of improving tissue oxygen delivery, suppressing the immune response, preventing some of the major complications of IMHA (such as thromboembolic disease), and hopefully preventing future recurrence. In the emergent patient, tissue oxygenation may be improved greatly by the administration of intravenous fluids. Although some clinicians worry about “diluting” an already anemic patient with IV fluids, in actuality, fluids will improve tissue oxygen delivery in the hypovolemic patient by maximizing cardiac output. However, the majority of dogs with IMHA will also require blood transfusion or oxyglobin during the course of their hospitalization, as immunosuppressive therapies are not rapidly effective in stopping the hemolytic process. The decision to transfuse is based on a number of factors, including hematocrit values, clinical signs, and the chronicity of the anemia. Clinical signs of anemia such as reluctance to eat, tachycardia unresponsive to fluids, tachypnea, dyspnea, lethargy, and altered mentation should prompt consideration for transfusion.

A number of drugs may be considered for the purpose of immunosuppression. Prednisone is the mainstay of therapy in dogs with IMHA, and at this time no other drug has been proven to work better than prednisone alone. Clinical experience suggests that 2 mg/kg/day in dogs provides adequate immunosuppression in the dog. Higher doses are not necessarily more immunosuppressive but may be associated with an increased risk of gastrointestinal complications. Prednisolone, rather than prednisone, should be used in cats as the bioavailability of prednisone is limited in this species. Additionally, cats may require higher doses than dogs, and the author typically uses 4 mg/kg/day in cats. A growing number of retrospective studies have suggested that azathioprine may improve long-term survival, and that cyclophosphamide may be associated with a poorer outcome. Caution should be used in interpreting these studies as inherent bias may be present due to their retrospective or small scale nature. In our clinic, we frequently use azathioprine (2 mg/kg q24h for 7 days then q48h) or myophenolate (10 mg/kg PO q12h) as adjunct therapies and to facilitate prednisone weaning later in the course of treatment. Intravenous immunoglobulin (IVIG) is also occasionally used in patients who are slow to respond to conventional therapy. IVIG is essentially purified IgG antibodies collected from the pooled plasma of over 2000 human donors. It is believed to act primarily by blocking macrophage Fc-receptors, thereby decreasing phagocytosis of red blood cells. Downregulation of antibody production, enhanced catabolism of antibodies, and suppression of cytokine release are other possible mechanisms of action. Although one small prospective study did not demonstrate more rapid response times in IMHA patients receiving IVIG at presentation, clinical experience and a number of retrospective studies have demonstrated its utility as a rescue therapy in individual patients. IVIG is typically dosed at 0.5-1 mg/kg given over 6 hours. Side effects include vomiting, fever, potential for anaphylaxis, and possible increased risk of thrombosis.

Thromboembolic disease (TE) is a frequent complication of IMHA. In studies of dogs with IMHA that underwent necropsy, TE was identified in 60-80% of cases. Sites most commonly affected were the pulmonary and splenic vasculature. Although exact mechanisms for the prothrombotic state have not been elucidated, increased concentrations of procoagulant factors, decreased concentrations of anticoagulant and fibrinolytic factors, vasculitis, enhanced platelet reactivity, the presence of antiphospholipid antibodies, liberation of RBC stroma, blood transfusion, and administration of steroids have all been hypothesized to play a role in the development of TE. Changes in primary hemostasis are also thought to play a role in the development of a pro-thrombotic state. Weiss & Brazzell demonstrated increased platelet P-selectin expression in dogs with IMHA, supporting the hypothesis that platelets circulate in an activated state. Documentation of the pro-thrombotic state remains challenging in clinical cases and has traditionally
been based upon detection of increased fibrinolysis (increased fibrin degradation products (FDPs) and D-dimers) and decreased endogenous anticoagulants (antithrombin) rather than rate of clot formation. Recently, our group has documented hypercoagulability as assessed by thromboelastography in this patient population. 26/26 dogs with idiopathic IMHA enrolled in this study all had an MA (maximal amplitude; a reflection of clot strength) that was significantly greater than normal.\(^5\)

Though antemortem identification of thromboembolic events can be challenging, data from 110 dogs treated for IMHA at the Michigan State University Veterinary Teaching Hospital between 2004 and 2007 showed that 34% were suspected to have developed TE during their hospital stay. Of the dogs with suspected TE, 51% had pulmonary thromboembolism (PTE) alone, 8% had portal venous thrombosis (PVT) alone, and 41% had both PTE and PVT.\(^2\,^3\) The development of TE appears to significantly contribute to the morbidity and mortality of IMHA. In our data, survival to discharge in dogs with TE was significantly lower than in dogs without TE (49% vs 81%) and median duration of hospitalization was longer (7 days vs 4 days). Of note however, 7 of 7 dogs with PVT identified on ultrasound whose owners opted for aggressive therapy all survived, suggesting that early identification and management of this problem may improve outcome.\(^4\) We are currently evaluating CT angiography as a technique for definitive identification and monitoring of pulmonary and portal clots.\(^d\)

Because hemostatic abnormalities are common in dogs with IMHA, obtaining baseline coagulation testing at the time of admission is strongly recommended. In our critical care unit, dogs with IMHA are then treated with heparin sodium at a loading dose of 150 units/kg IV followed by a continuous infusion of 30-60 units/kg/hour. The heparin dose is adjusted daily to prolong the activated partial thromboplastin time (aPTT) to 1.5-2 times the baseline value. Twenty-six dogs prospectively enrolled in a coagulation study and heparinized based upon this protocol all survived to discharge and serial evaluation of thromboelastography showed normalization of parameters related to clot formation by 30 days, once hemolysis was no longer taking place.\(^3\) Low dose aspirin (0.5 mg/kg PO BID)\(^2\) may also be started during hospitalization, particularly in cases where there is failure to achieve a target aPTT. Plavix (2 mg/kg q24h) or aspirin (0.5 mg/kg q12h) are frequently started at the time of discharge to prevent rebound hypercoagulation associated with heparin withdrawal.

Gastrointestinal protectants, such as pepcid (0.5 mg/kg q24h) or sucralfate, are used by many clinicians in hopes of preventing GI ulceration. At this time there is no evidence to suggest that these medications are effective in preventing ulcers, and in our hospital, they are typically administered only once ulceration is suspected to have occurred. Gastric ulceration should be suspected if melena, vomiting, or reluctance to eat develop, or if serum total protein begins to fall in conjunction with the hematocrit. It is important to recognize the development of GI blood loss, because the resulting drop in hematocrit can otherwise be easily confused with treatment failure.

Dogs with idiopathic IMHA are at risk for recurrence of disease, and care should be taken not to wean the immunosuppressive drugs too quickly. Prednisone is typically maintained within the immunosuppressive range for at least one month following hospital discharge, and then may be decreased by approximately 20-25% each month, provided that the hematocrit remains stable. If azathioprine or other adjunctive agent is being administered in conjunction with the prednisone, it may be discontinued one month after discontinuing prednisone. In total, the weaning process should span at least 4-6 months. Labwork should be rechecked one week after each decrease in drug dosage to make sure that the change is tolerated. If relapse occurs during the weaning process, immunosuppressive dose prednisone should be reinstituted, then gradually weaned back to the lowest effective dose. Following weaning, it is frequently recommended that vaccines be avoided, though the association between vaccines and IMHA development is still unproven. Splenectomy may be considered for dogs with recurrent or refractory disease.

Abstracts and unpublished data

References
Management of the Difficult Urethral Obstruction
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Overview and pathophysiology
Feline urethral obstruction is one of the most common emergency presentations in the cat, accounting for approximately 9% of feline emergency admissions.\(^1\) While there are many factors that may play into the development of lower urinary tract diseases in the cat, matrix-cystaline plugs and urolithiasis are the most common causes of obstruction.\(^2\) Cats with urethral obstruction may have signs localized to the lower urinary tract including dysuria, stranguria, pollakiuria, hematuria, vocalizing, and pain, or they may show signs of systemic illness such as vomiting, lethargy, or collapse. Cats with obstructive urinary tract diseases may or may not have demonstrated preceding signs of lower tract disease.

Following the development of urethral obstruction, clinical signs of uremia typically develop within 24 hours.\(^3\) Dehydration occurs due to decreased water intake and ongoing fluid losses secondary to vomiting. Acid-base (metabolic acidosis) and electrolyte disturbances (hyperkalemia and hyperphosphatemia) develop due to impaired excretion. Accumulation of metabolic wastes leads to post renal azotemia. Bladder capacity is reached, leading to rising intravesicular pressure and subsequently falling glomerular filtration rate (GFR). Prolonged obstruction may result in intrinsic renal failure. Damage to the urothelium and detrusor muscle may also develop during this time. If left untreated, death secondary to cardiopulmonary failure or hyperkalemia may occur within 3-6 days. Damage to bladder mucosa or urethra may shorten survival times.\(^3\)

Diagnosis of urethral obstruction
Diagnosis of urethral obstruction is generally made on the basis of history and physical exam findings. Abdominal palpation typically reveals a turgid, painful bladder, though in rare cases, the bladder may be moderate in size if the cat is presented to the veterinarian shortly after clinical signs develop. Blood and/or crystalline debris may be visualized at the urethral orifice. The presence of bradycardia frequently indicates hyperkalemia, and severe systemic signs in conjunction with free abdominal fluid should prompt consideration of bladder leakage or rupture. In contrast, cats that present with stranguria but appear systemically healthy and have palpably small bladders typically have non-obstructive lower urinary tract disease.

At the time of presentation, a peripheral IV catheter is placed and blood is collected for complete blood count, serum biochemistry panel, and venous blood gas/electrolyte panel. The blood gas/electrolyte panel is particularly helpful as it provides rapid information on parameters such as potassium concentration (as well as acid-base status and renal values) that may affect initial interventions. Electrocardiography can also be helpful in the initial evaluation of the patient with urethral obstruction. Early ECG changes suggestive of hyperkalemia include bradycardia, dampened P-waves, tented T-waves, and prolongation of the P-R interval. As hyperkalemia worsens, loss of P-waves (atrial standstill) and widening of the QRS complex may develop. Electrocardiographic changes typically do not develop until potassium levels are greater than 7 mEq/L, but there is a great deal of individual variation in terms of patient response to hyperkalemia. Metabolic acidosis, hypokalemia, and hypocalcemia may contribute to the likelihood of hyperkalemic cardiotoxicity.

Once the animal has been medically stabilized and deobstructed, urine is submitted for urinalysis and culture. Because crystalline and cellular composition of the urine may change over time, evaluation of a fresh, undiluted sample is preferred. Diagnostic imaging should be performed to rule out cystic or urethral calculi. If a urolith or crystalline-matrix plug is retrieved at the time of deobstruction, composition should be determined as this may impact future therapies.

If free abdominal fluid is identified, fluid chemistry may be helpful in determining whether urinary tract rupture has occurred. An abdominal fluid:serum creatinine ratio of 2:1, or abdominal fluid:serum potassium ratio of 1.9:1 (cat) or 1.4:1 (dog) is predictive of uroperitoneum.\(^3\) Cytology of the fluid sample should also be performed to rule out urosepsis. Contrast cystourethrography is used to determine location and severity of the rupture.

Treatment of urethral obstruction
Fluid therapy
Initial management of urethral obstruction in the cat should focus on correction of hypovolemia, hyperkalemia, and other acid-base and electrolyte disturbances. In most cases, appropriate fluid therapy followed by restoration of urine flow will effectively correct these abnormalities. A peripheral IV catheter should be placed and fluid therapy instituted immediately using 0.9% sodium chloride or balanced electrolyte solution such as lactated Ringer’s solution (LRS). A shock rate of fluids (66 ml/kg/hour in the cat) is calculated and then administered to effect in increments of approximately ¼ of the calculated dose, reassessing major body systems after each bolus. For example, the calculated shock rate in a 5 kg cat is approximately 330 ml, and should be administered in individual boluses of 50-100 ml every 10-15 minutes until cardiovascular status is restored. The goal of fluid therapy should be normalization of vital signs such as heart rate, level of consciousness, pulse quality, blood pressure, and capillary refill time. The specific type of intravenous
flут selected is of lesser importance than the administration of appropriate volume. Although 0.9% sodium chloride has traditionally been selected due to its lack of potassium, studies in both experimental and clinical cases have shown that potassium containing solutions (LRS, Normosol-R) do not adversely affect the rate of resolution of hyperkalemia in cats with urethral obstruction when compared with 0.9% saline.\textsuperscript{5,6} Additionally, the buffered solutions are more efficient at restoring electrolyte and acid-base balance in severely affected animals.

**Hyperkalemia**

Relative or absolute bradycardia should be immediately investigated by monitoring electrocardiography and serum electrolyte concentrations. Severe electrocardiographic changes such as atrial standstill, widened QRS complexes, or sine wave formation provide strong indication for the administration of calcium gluconate. Calcium gluconate (10%) is given slowly at a dose of 0.5–1.5 ml/kg IV while carefully watching the patient’s ECG for arrhythmias. Although calcium gluconate does not lower the serum potassium level, it has the immediate effect of buffering the myocardiurn from the toxic effects of hyperkalemia by restoring the normal difference between resting and threshold membrane potentials. Other intermediate to long-term interventions for hyperkalemia include the administration of regular insulin/dextrose and sodium bicarbonate, though these therapies are rarely warranted in animals with urethral obstruction as fluid therapy followed by timely restoration of urine flow are generally effective at reversing the hyperkalemia.

However, if needed, 50% dextrose may be diluted 1:1 with saline and given at a dose of 1 gm/kg body weight to promote endogenous insulin release with subsequent potassium uptake by the cells through stimulation of sodium-potassium pumps. If regular insulin is used, it should be given at a rate of 1 unit insulin per 3 gm dextrose, though this is generally unnecessary and creates the need for careful blood glucose monitoring thereafter to avoid hypoglycemia. Sodium bicarbonate may also be given at a dose of 1 mEq/kg intravenously to facilitate intracellular potassium shifting in exchange for hydrogen ions.

**Techniques for urethral deobstruction**

During the initial exam, the urethra may be gently massaged, followed by careful palpation of the bladder to potentially dislodge superficial plugs. Extreme care should be taken to avoid accidental bladder rupture. While this technique is rarely effective, it is a simple extension of the initial physical exam and therefore may be worth trying in less severely affected cats prior to catheter deobstruction.

Although severely depressed patients may be deobstructed without the need for chemical restraint, sedation/analgesia is employed in the majority of “blocked” cats to improve patient comfort, facilitate deobstruction, and avoid urethral or bladder trauma secondary to patient struggling. Ketamine (100 mg/ml) may be combined with diazepam (5 mg/ml) in equal parts by volume and given at a dose of 1 ml/10 kg of the 50:50 mix. However, this combination should be avoided in cats with known or suspected hypertrophic cardiomyopathy, or when an undiagnosed murmur or gallop rhythm is present. In these cases, hydromorphone (0.05 mg/kg) in combination with diazepam (0.2 mg/kg) may provide a safer option.

Following sedation, the cat is positioned in dorsal recumbency with the legs pulled forward over the head. In this position, the prepuce may be retracted and the penis extruded by simply pushing the prepuce downward towards the anus. A further advantage to this technique is that it allows the urethra to be maximally straightened to facilitate deobstruction. The author’s preferred technique for deobstruction uses an olive tip catheter (FUS needle 21 g x 1”, Jorgensen Laboratories, Loveland, CO). This is a metal, bulb-tipped catheter that can be used to flush the urethra and either break down matrix-cry stalline plugs or hydropulse them atraumatically into the bladder. Initially, the olive tip catheter is lubricated and inserted gently into the urethra to the site of the obstruction, approximately 1–2 cm. A 3 cc syringe is then used to lavage and break down the plug. Bits of the plug will often be seen emerging from the urethral orifice during the lavage. When the catheter is withdrawn, a strong stream of urine will frequently force the remainder of the plug from the urethra. Gentle bladder palpation may be used at this point to assist in the expulsion of the plug. To avoid urethral trauma, the catheter should not be forced past the obstruction. Instead, the lavage solution should be allowed to do the work. Additionally, acidic solutions should not be used for lavage as these have not been shown to be effective at plug dissolution and may further traumatize the urethral mucosa. If lavage alone is not successful at dislodging the urethral plug, the tip of the urethra can be pinched around the bulb tip of the catheter and hydropulsion used to push the plug back into the bladder.

Many clinicians use polypropylene “tomcat” catheters for the purposes of unblocking cats. These have the potential to cause additional trauma to the urethra when the rigid catheter is forced past the site of obstruction. If used, a number of steps may help to minimize iatrogenic urethral damage and maximize chances of success. (1) Completely straighten the urethra by pushing the prepuce dorsally towards to anus until the penis is parallel to the spine. (2) Use copious amounts of lubrication. (3) Hydropulse with sterile saline prior to advancing the catheter to assist in dislodging the plug. (4) Use a very light touch when advancing the catheter. Hold the catheter between index finger and thumb and twirl gently while advancing. Think about “picking a lock” when attempting to advance the catheter. Use finesse instead of force. (5) Once the catheter is well seated in the urethra, the penis may be allowed to retract into the prepuce. The prepuce may then be pulled caudally (toward tail tip) to further straighten the urethra while the catheter is advanced.

Some experienced clinicians advocate the use of cystocentesis prior to deobstruction to decompress the bladder and to potentially facilitate hydropulsion of urethral plugs. The author prefers to reserve this technique for use only as a last resort due to the number of
cats presenting to the emergency service with uroperitoneum and apical bladder tears following cystostenosis of overdistended bladders. However, it should be noted that our institution may see a biased population of more severely affected animals.

Cats that are critically ill, and those demonstrating large amounts of “sandy” crystalline debris in the urine, blood clots, uroliths, plugs hydropulsed into the bladder, bladder atony, or urethral narrowing are particularly at risk for reobstruction post-unblocking. For this reason, a soft, indwelling, 3.5-5 French red rubber catheter is placed following deobstruction to facilitate urine drainage overnight and to assist in quantitation of urine output. Indwelling catheters should be placed using liberal clipping and scrubbing of the perineum and aseptic technique to minimize risk of catheter-induced urinary tract infection. The tip of the catheter should sit just past the bladder neck to reduce risk of kinking or knotting. The catheter should then be connected to a sterile, closed collection system. To decrease the likelihood of premature catheter removal, careful attention should be given to suture placement. A piece of butterfly tape is placed around the catheter and appositional sutures are placed *at the margin* of the butterfly tape to prevent kinking of the catheter. The catheter body is then taped to the tail. An Elizabethan collar should be placed prior to anesthetic recovery.

**Hospital management**

**Fluid therapy**

Following initial stabilization and correction of hypovolemia, fluid rates should be adjusted to account for remaining fluid deficits, daily maintenance requirements, and ongoing losses. Deficits can be estimated as follows based upon clinical signs of dehydration: mild (5-6%), moderate (7-8%), and severe (8-10%). Multiplying the estimated percent dehydration by body weight gives the fluid deficit, which may then be replaced over the next 24 hours. For example, a 5 kg cat estimated to be 8% dehydrated would have an estimated deficit of 400 ml. To this value must be added maintenance needs (approximately 60 ml/kg/day) and ongoing losses. Ongoing losses following “unblocking” result from post-obstructive diuresis and can be estimated most easily by quantitating urine output. Normal urine output is approximately 1-2 ml/kg/hour (5-10 ml/hour in the average 5 kg cat). Urine output in excess of this amount typically results from post-obstructive diuresis. During the first 24 hours of therapy, a fluid rate should be selected that accounts for these ongoing losses. In other words, the intravenous fluids administered should slightly exceed measured urinary losses.

Urine output is quantified every four hours. Inadequate urine production (<1 ml/kg/hr) indicates inadequate fluid administration or urinary catheter occlusion with debris. After troubleshooting the catheter, a fluid bolus followed by an increase in fluid rate is indicated if urine output remains low.

Fluid therapy is typically tapered over the next 24-36 hours. Daily monitoring of electrolytes and renal values should be performed to ensure that azotemia resolves and electrolytes normalize. Potassium supplementation may be required during post-obstructive diuresis should hypokalemia develop.

**Urinary catheter care**

Indwelling urinary catheters and tubing should be cleaned externally once daily with a dilute chlorhexidine solution. Gloves should be worn and aseptic technique used when handling the catheters to avoid nosocomial infection. Bladder palpation should be performed every 4-6 hours to ensure that the bladder remains decompressed. When moving the patient, the urine collection system tubing should be clamped and the bag held below the level of the patient to prevent retrograde flow of urine into the bladder.

To minimize likelihood of catheter-induced urethral irritation or urinary tract infection, catheters should be removed as soon as possible. For most cats, the catheter is removed within 48 hours, but the presence of excessive crystalline debris or blood clots in the urine may necessitate longer indwelling catheter duration to avoid reobstruction. Use of antibiotics during hospitalization is not recommended as this is unlikely to prevent catheter-related infection, but may contribute to antibiotic resistance of organisms protected by the catheter biofilm. Culture should be performed prior to catheter removal, with antibiotic therapy initiated as indicated based upon results of culture and sensitivity.

Following catheter removal, patients should be monitored for an additional 12-24 hours to ensure that the urethra remains patent. Cats will typically urinate small volumes frequently following catheter removal due to irritation resulting from obstruction and catheterization. Although they may appear to strain in the litterbox, the bladder should remain small on palpation. A progressively distending bladder post-catheter removal typically indicates reobstruction (firm bladder, difficult to express) or bladder atony (large, flaccid, expressible). Cats with suspected urethral spasm post catheter removal may benefit from a smooth muscle relaxant following catheter removal (prazocin 0.5 mg/cat q24h).

**Pain management**

Urinary obstruction and initial management are frequently associated with significant discomfort. In our practice, buprenorphine (0.01 mg/kg IV q6h) is commonly used to provide analgesia for the first 24-48 hours.

**Long term management**

Strategies for long-term prevention of recurrence focus primarily on environmental modification and dietary changes. Occasionally, pharmacologic intervention may be warranted. An ample number of litterboxes should be provided, particularly in multi-cat households, and litterboxes should be cleaned regularly to encourage more frequent use. Canned or moistened food may decrease frequency of lower urinary tract episodes by promoting a more dilute urine and increasing frequency of urination. Fresh water should
be available at all times. In cases where obstruction was caused by struvite-matrix plugs, an acidifying diet may be of benefit. Antibiotics, anti-inflammatories, and antispasmodics have not been associated with reduction in frequency of episodes and their routine use is not recommended.

**Perineal urethrostomy**

Perineal urethrostomy may be considered in cases where frequency of urethral obstruction is unacceptable despite appropriate medical management or when irreversible changes in the urethra (stricture, scarring, urolithiasis) cause recurrent or persistent obstruction. Perineal urethrostomy has been associated with significant short and long term complications including recurrent urinary tract infection and stricture, and as such should not be considered a first line recommendation for cats with urethral obstruction.

**References**

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. 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 multigravid bitches may occur several days before parturition. A sudden drop in body temperature (>2°F) is generally noted within 24 hours of parturition in dogs and cats as a result of decreases in progesterone levels, but this finding is not always reliable. In one recent 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.

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. 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:

- A definite cause is apparent (i.e. 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 that 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 (e.g. 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.\textsuperscript{4,8}

**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 intraterine 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.\textsuperscript{10} 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.\textsuperscript{11}

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.\textsuperscript{4,12} The oxytocin dose may be repeated in 30 minutes if expulsion 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 noted\textsuperscript{4,12} 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.\textsuperscript{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:\textsuperscript{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. 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. Alternatively, 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 mask inductions 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, and methoxyflurane.13-15

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.3,7-9 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.3,7 For these reasons, the decision to proceed to caesarian 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.13,14

Periparturient emergencies
Mastitis
Mastitis is a postpartum complication seen in both dogs and cats that results from bacterial infection of the mammary glands. Bacteria most commonly enter through the nipple as a result of nursing, trauma, or poor hygiene, but may also be spread hematogenously. In mild cases, discomfort, swelling, and inflammation may be seen, while in severe cases, signs of systemic illness such as fever, anorexia, and lethargy frequently develop. Dogs often refuse to allow their young to nurse and may be reluctant to lie down. Severe mastitis often progresses to abscission and necrosis.

Diagnosis of mastitis is generally based on history and clinical signs (fever and swollen, painful glands in the postpartum animal), but baseline CBC and chemistry as well as milk cytology and culture are useful for assessing severity of illness and appropriateness of antibiotic selection. Milk expressed from the gland may be purulent and cytology typically shows large numbers of white blood cells and intracellular bacteria. The most common bacteria isolated on culture include E. coli, Staphylococci, and Streptococci.

Treatment is initiated immediately with broad spectrum antibiotics. Amoxicillin-clavulanic acid or cephalaxin are good first choices and are safe for nursing neonates. Other measures that may be useful in the management of mastitis include warm compresses, hydrotherapy, and frequent milk stripping. If a fluctuant abscess pocket is identified on palpation, early lancing and flushing may limit the degree of skin necrosis that follows. Large, ruptured mammary abscesses may be successfully managed as open wounds with warm compresses, hydrotherapy, and systemic antibiotics, but in these cases mastectomy may provide a more rapid and cosmetic resolution of the problem.

Endometritis
Endometritis is a bacterial infection of the uterus that is generally seen within the first three days (up to one week) after whelping, though it may develop during pregnancy as well. Potential causes include retained fetuses or placentas, abortions, uterine trauma secondary to dystocia or obstetrical manipulation, and ascending infection from the vaginal canal. Typical signs include fever, lethargy, anorexia, vomiting, diarrhea, poor lactation, neglect of offspring, and foul-smelling vaginal discharge. Just as in the non-pregnant dog, any purulent vaginal discharge noted during or after pregnancy is abnormal and should prompt investigation.

Labwork abnormalities consistent with sepsis may be seen, including leukocytosis with a left shift or leukopenia, thrombocytopenia, elevated liver values, and hypoalbuminemia. Coagulation testing should be performed to rule out disseminated
intravascular coagulation. Radiographs or ultrasound are indicated to evaluate for fetal death, retained placentas, or evidence of uterine enlargement. Cytology of vaginal discharge typically shows degenerate neutrophils and macrophages with intracellular bacteria. The most common organisms associated with uterine infections include *Staphylococci*, *Streptococci*, *E. coli*, *Salmonella*, *Campylobacter*, and *Chlamydia*.

An animal suspected of having septic metritis should be treated aggressively with IV fluids. Broad spectrum antibiotic combinations such as ampicillin-enrofloxacin, ampicillin-aminoglycoside, or cefazolin-aminoglycoside-metronidazole, should be administered. Following stabilization, ovariohysterectomy is the treatment of choice for metritis. If the animal is not showing signs of sepsis and the owner wishes to use her for breeding purposes in the future, evacuation of the uterine contents using PGF₂α (Lutalyse) may also be attempted in conjunction with broad spectrum antibiotics. PGF₂α is typically administered at doses of 0.1-0.25 mg/kg SQ once daily for 5 days. If initial dosing does not result in adequate expulsion of uterine contents, the author generally increases treatment frequency to twice daily. Potential complications of PGF₂α include vomiting, abdominal discomfort, uterine rupture, and septic peritonitis. Because PGF₂α treatment may require several days to achieve a good effect, animals that are severely ill should always be treated with ovariohysterectomy. Ovariohysterectomy is also the best choice when the animal is not intended for future breeding or if the health of the dam is a higher priority than possible future breedings.

**Eclampsia**

Eclampsia or puerperal tetany is a life threatening condition that results from the development of hypocalcemia in the periparturient period. It is one of the more common complaints noted following parturition, accounting for roughly 1/4 of periparturient emergencies. Eclampsia is results from the loss of calcium through lactation and fetal skeletal mineralization, in excess of that entering the extracellular fluid through gastrointestinal absorption and bone resorption. Other factors such as inadequate diet or parathyroid atrophy resulting from oversupplementation of calcium may also contribute, though diet in affected animals has not been reported to be significantly different from non-affected animals. Increasing litter size to maternal body weight ratio has also been identified as a significant factor in the development of periparturient hypocalcemia.

Eclampsia is most commonly seen in small dogs, first-time whelpings, and dogs with large litter sizes. It typically develops 2-4 weeks after parturition but is occasionally seen in late gestation. Clinical signs in dogs most commonly include stiff gait, trembling, twitching, seizures, tachycardia, panting, and hyperthermia, but some dogs may present with atypical signs such as whining, vomiting, diarrhea, and behavior changes. If untreated, death may result from respiratory impairment, or from hyperthermia and cerebral edema. Cats may present with clinical signs similar to dogs, but unlike dogs, are more prone to hypothermia, and may present with hypertension, hyperglycemia, or flaccid paralysis in place of clonic-tonic muscle spasms.

Diagnosis of eclampsia is made on the basis of history and physical exam findings in conjunction with low total or ionized calcium levels. Ionized calcium represents the physiologically active portion of calcium within the body, and is involved in muscular contraction, as well as neurologic and cardiovascular function. Ionized calcium levels are therefore believed to be a more sensitive indicator of extracellular calcium levels than total calcium, and typically fall below 0.8 mmol/L in dogs with eclampsia (reference range: 1.2-1.4 mmol/L). However, total calcium levels have been found to be decreased in all dogs with eclampsia, suggesting that total calcium levels may provide sufficient information in this disease if ionized calcium measurement is not available.

Animals presenting with eclampsia should have an IV catheter placed and intravenous fluids administered to address fever, dehydration, and tachycardia. Calcium gluconate (10%) should immediately be administered intravenously slowly to effect. Most animals will have tremors controlled at doses ranging from 0.5 to 1.5 mL/kg. An ECG should be monitored during calcium administration and the infusion stopped if bradycardia or arrhythmias develop. Ionized calcium levels should be rechecked post administration to make sure that ionized calcium levels remain within the normal range. Temperature should be carefully monitored in animals presenting with tremors, and active cooling measures (cool fluids, alcohol applied to footpads) should be instituted for patients with severe hyperthermia. Body temperature generally falls quickly once tremors are controlled, so active cooling measures should be discontinued once the temperatures falls below 103°F. Oral calcium carbonate (Tums) supplementation should be continued at a dose of 100 mg/kg/day throughout lactation. Up to 20% of dogs may have recurrence of eclampsia despite supplementation if puppies are allowed to nurse, so bottle feeding and early weaning of the puppies is recommended.

Supplementation of calcium prior to whelping is not recommended, as this may downregulate parathyroid hormone secretion, decreasing intestinal calcium absorption and increasing the risk of eclampsia during lactation. Instead, calcium administration (100 mg/kg/day divided) should be instituted following whelping in dogs at risk and dogs with a previous history of eclampsia.

**References**

Prior to anesthesia, the overall pet must be reviewed for any preexisting systemic or local complications. Review of the medical record and blood work prior to a pre-anesthetic examination will allow for a smooth anesthetic procedure and recovery.

Make dental radiography your friend by taking images prior to undertaking any oral surgery. The clinician must know how much ventral cortex is available prior to undertaking a surgical extraction of a mandibular canine tooth or 1st molar.

The following cases will be reviewed with pearls to help the practitioner avoid harmful pitfalls:

1. Mandibular 1st molar extractions
2. Mandibular canine extractions
3. Oronasal fistula repair
4. Removal of retained tooth roots
5. When to remove a tooth vs. crown amputate (dogs and cats)
6. How to biopsy an oral mass without compromising margins or architecture
Almost every ancient civilization has worshipped a god of the sun as healing powers attributed to the sun are believed to be broad-reaching. Even today, sun exposure is widely felt to induce a sense of well-being. Solar exposure is essential in humans for the synthesis of Vitamin D3 and the setting of internal clocks. However, sunlight can also cause deleterious changes like acute and chronic inflammatory skin reactions, skin cancer, photo-aging and solar exposure can also elicit adverse reactions to certain drugs. Other sources of ultra-violet light (UVL) and visible radiation besides the sun include fluorescent lights, incandescent bulbs, photocopy machines, phototherapy lamps, tanning salons and dental or nail salon procedures to harden acrylics.

The electromagnetic (EM) spectrum spans from high energy x-rays down to low energy microwaves and radio-waves. UV radiation and visible light are included in the EM spectrum, too. The shortest wavelengths carry the highest energy radiation and vice versa (longest wavelength, lowest energy radiation). Fortunately, wavelengths shorter than 290nm are absorbed by the ozone layer and do not reach the earth’s surface at sea level (slightly shorter wavelengths have been detected at higher altitudes). 200-290nm = UVC (germicidal radiation) and is strongly absorbed by DNA. This wavelength can be lethal to viable cells of the epidermis and bacteria. UVC lamps used for air and water purification emit wavelengths of 254nm. Avoiding exposure (eyes, skin) to UVC wavelengths is paramount as UV keratitis and mutation can occur. 290-320nm = UVB (mid-UV or “sunburn spectrum”) comprises the biologically most active wavelengths reaching the earth’s surface. Ordinary window glass transmits only longer wavelengths and most sunscreens efficiently reflect or absorb these wavelengths. Sunscreen SPF is based on testing against this waveband; “broad-spectrum” products also protect against UVA (SPF > 45). 320-400nm = UVA (long-wave UV) is also called “black light” as these wavelengths are not visible to the human eye. However certain substances can emit visible fluorescence. UVA wavelengths comprise ~95% of UV radiation reaching the earth’s surface. The UVA spectrum is further split into UVAI (340-400nm) and UVAII (320-340nm) with UVAII being more damaging.

The basics: so you walk out into the sunlight… what happens? And why do we care?

UV and visible light photons reach the skin surface … radiation penetrates to appropriate level of the skin where it is absorbed by chromophores … photochemical reactions convert chromophores into new molecules (= photoproducts) … photoproduct molecules stimulate cellular signal transduction pathways … leading to biochemical changes and cellular effects (acute and chronic skin response).

In greater detail: UV and visible radiation strike the skin with three potential outcomes: part can be remitted (reflected and scattered), part can be absorbed by chromophores and part can be transmitted inward to different layers of the skin until the energy of the incident beam has been dissipated. Absorption and scattering are the two major processes limiting penetration of UV and visible radiation into the skin. Wavelengths less than 320nm are readily absorbed by proteins, DNA and other components of epidermal cells. The process of scattering and absorption account for the low penetration of these wavelengths into the skin. 5-10% of incident light is reflected by the stratum corneum. The phenomenon of “specular reflectance” accounts for the surface appearance of the skin in that it will appear glossy if skin surface is smooth, wet or oily and rough/dull if there is an irregular surface due to scattered light. In Caucasians with fair skin, ~50% of incident radiation is light that has penetrated the epidermis and scattered within the dermis and back through the epidermis and skin surface. Melanin, normally present only in the epidermis, acts as a neutral density filter to diminish dermal remittance. The greater overall melanin content in darker skin absorbs more visible light and causes the skin to appear darker because less light is reflected back to the observer. Hemoglobin within the dermis absorbs shorter (blue) visible wavelengths and is responsible for the reddish hue we perceive to blood based on total remittance of light. Abnormal location and/or quantity of these or other pigments accounts for the appearance of the skin in pathologic conditions like melasma (extra pigment in the epidermis and/or dermis) and vitiligo (absence of epidermal melanin).

Absorption is the process of a photon being taken up by a chromophore. This molecule now contains extra energy and may be altered to form a new molecule (= photoproduc) which initiate a variety of responses in skin cells. Absorption is the first step in a photobiologic response. Skin is comprised of water, organic molecules (proteins, lipids, nucleic acids) and inorganic ions (sodium, chloride, calcium). Organic molecules absorb photons in UVA and UVB spectrums and only absorbed radiation can initiate a biologic response (First Law of Photochemistry, 1818).

After absorbing the energy of a photon, the chromophore is in an excited state. This “excited state” lasts for only a few nanoseconds as the excited chromophore can return to its ground state by emitting light (fluorescence) or by releasing energy as heat OR can undergo a chemical reaction to form a photoproduc. A fourth option is to convert to a triplet excited state with intersystem crossing, but this is far too nerdy and not applicable for our lecture. An excited state chromophore reacts with nearby molecules and
the photoproducts initiate transduction processes that lead to the observed responses in the skin (= photobiologic responses). The photoproducts (or transformed chromophores) are important for many UVB-induced responses in the skin. Cyclobutyl pyrimidine dimer (CPD) is a 4-membered ring structure that is created when thymine or cytosine absorbs UVB light and forms a covalent bond to an adjacent thymine or cytosine. This CPD leads to mutations at sites on DNA strands as a result of sun exposure. 7-dehydrocholesterol (pro-vitamin D3) is a chromophore in skin that absorbs UVB light and represents one of the rare positive UVB-induced photochemical reactions in the skin! The pro-vitamin D3 as an excited state molecule is converted into pre-vitamin D3, isomerizes to Vitamin D3 (cholecalciferol), enters circulation and ultimately is hydroxylated in the liver and kidney, forming the active hormone 1,25-dihydroxy vitamin D (calcitriol).

Photosensitization is the process when certain drugs and dyes (photosensitizers) absorb UV and/or visible radiation and cause delayed erythema or inflammation. In most cases of photosensitization, oxygen is required. Photosensitivity responses are mediated by reactive oxygen species (ROS - singlet oxygen, hydrogen peroxide, superoxide anion, hydroxyl radical, nitric oxide) and oxidized unsaturated lipids, certain amino acids in proteins (histidine, methionine, tryptophan, cysteine) and nucleic acids. Products formed during photosensitization initiate signal transduction processes and lead to the production of inflammatory mediators (like prostaglandin E2 and cytokines TNF-α, IL-1). The photosensitizer absorbs the UV photon and the change in electron energy creates a “triplet excited state.” The photosensitizer can then react with ground state oxygen to transfer energy and return the photosensitizer to its ground state while also generating a highly reactive singlet oxygen. This singlet oxygen is unique among reactive oxygen species because it is an excited state molecule. Single oxygen has a short lifetime in cells (< 4 ms) because it reacts with many cellular molecules (ex. wheal and flare response in erythropoietic protoporphyria and photoxicity mechanisms of phototoxic drugs). Endogenous UVA-absorbing chromophores are believed to generate singlet oxygen in keratinocytes and fibroblasts. Other ROS are produced in the skin after photon absorption by photosensitizers and endogenous chromophores. For example, hydroxide peroxide and superoxide anion are produced after direct photon damage to mitochondria and also after redox active enzymes in keratinocytes and fibroblasts are stimulated in cells exposed to UVB or UVA radiation. It is believed that UVA- and UVB-induced responses in normal skin and photosensitivity conditions are generally dependent on the production of ROS. Antioxidants act by chemically reacting with and removing ROS and other free radicals before they can damage cellular molecules.

Skin cancer is caused largely by direct damage to DNA, leading to specific gene mutations. However, for clinically apparent skin cancer to develop, a second UVB-induced mechanism is required (UVB-induced immunosuppression). Experiments by Kripke in the 1970’s demonstrated that UV-induced tumors, which had been transplanted onto syngeneic recipient mice, were rejected if the recipient mouse had not been exposed to UVB light. This implies that UV-induced tumors have a highly antigenic phenotype and that a functioning immune system can reject tumor formation.

DNA is a chromophore for UVB radiation and can also be a direct target. Pyrimidine dimer formation is the direct cause of UV-induced immunosuppression. UVB irradiation leads to CPDs in antigen presenting cells, causing impairment of their antigen presenting capacity. This damage persists for several days and the damaged cells migrate from site of original damage within the skin to the lymph node(s). Topical application of photolyase-containing liposomes to UVB-exposed sites prevents UVB-induced immunosuppression as these products contain a DNA repair enzyme. The generation of ROS is likely involved in photoimmunosuppression as studies have shown that topical application of an antioxidant before and after UVB irradiation to mouse skin protected against UVB-induced immunosuppression.

Antioxidants have the ability to reduce oxidative stress, inflammatory responses and skin cancer. The cell membrane of an immunocompetent skin cell can also be another target of photoimmunosuppression. The cell membrane transfers signals from outside into the cell via membrane receptors. UVB exposure leads to clustering and internalization of cell surface receptors for epidermal growth factor, TNF-α and IL-1 in the absence of their ligands. Clustering results in the strong activation of stress-induced c-Jun NH2-terminal kinases, members of the mitogen-activated protein kinase family. Abnormal receptor clustering may subvert signaling pathways normally used by growth factors and cytokines.

UVB radiation of keratinocytes alters expression of surface molecules and induces synthesis and secretion of immunomodulatory soluble factors (IL-1, IL-6, IL-8, TNF-α, PGE2). TNF-α and IL-10 are two potent immunosuppressive cytokines. TNF-α affects dendritic cells and draining lymph node(s), leading to local immunosuppression. IL-10 is a T-helper cell type 2 cytokine that disrupts the production of TH1 cytokines, especially IFN-γ UVB irradiation can shift the immune response from Th1 to Th2 response, explaining why Th1-mediated cellular immune response is often impaired by UV radiation.

Langerhans cells (LC) make up ~2-5% of epidermal cells and are dendritic cells which originate in the bone marrow. LC are professional skin-specific antigen presenting cells that ingest antigen locally in the skin and lack a co-stimulatory capacity. After antigen uptake, LC migrate to the draining lymph node and mature to potent stimulators for antigen-specific T cells. These LC express high levels of MHC molecules and co-stimulatory molecules. UV radiation leads to the disappearance of LC from irradiated sites and impaired antigen-presenting capacity. UVB-irradiated LCs preferentially activate CD4+ Th2 cells.

Acute effects of UV radiation: UV radiation (UVR) absorption by DNA leads to the formation of dipyrimidine lesions, both the previously mentioned CPD’s and pyrimidine (6-4) pyrimidone photoproducts. Unless DNA is repaired via nuclear excision,
dipyrimidine lesions give rise to highly characteristic UVB signature mutations: C → T or CC → TT transitions. Solar UVB causes much more damage in the basal layer than UVA. In human dermatology, terms like “action spectrum” and “standard erythema dose” are applied for erythema and epidermal DNA photodamage by skin phototypes.

Sunburn: Sunburn (erythema) shows the classic signs of inflammation: redness, warmth, pain and swelling. Intensity of sunburn is determined by UVR exposure dose, irrespective of the time over which this is delivered. Within an hour of UVR exposure, mast cell pre-formed products are detected within the skin and may play a role in the initial development of erythema (esp. histamine, serotonin and TNF-α). The inflammatory infiltrate of neutrophils and T lymphocytes are evident in the upper epidermis 3 hours after UVB exposure and increases rapidly until 48 hours. Between 48-72 hours, neutrophils and macrophages are also observed. These and resident keratinocytes contribute to the inflammatory process. The pharmacology of sunburn is incompletely understood, but there is a documented role of prostaglandins (PGE2 especially) and nitric oxide. The resolution of erythema is likely dependent on the synthesis of Th-2 anti-inflammatory cytokines IL-10, IL-4 and transforming growth factor β1.

Macrophages are also important in this process. Afferent sensory nerves innervating the epidermis and dermis release a variety of neuropeptides after UVR exposure. Substance P and calcitonin gene-related peptide may act as mediators for pain and itch and also inflammation and immunomodulation.

Two of the more common responses of the skin to sunlight are tanning and hyperplasia of the dermis, epidermis and stratum corneum. Skin types that tan well have eumelanin (the darker, insoluble form of melanin) versus pheomelanin (lighter, alkali soluble, sulphur-containing melanin). Melanogenesis or “delayed tanning” is primarily a UVB response and is caused by the increased activity and numbers of melanocytes within the skin. This process also includes increased melanocyte tyrosinase activity, elongation and branching of melanocyte dendrites and an increase in the number and size of melanosomes.

Antioxidant defenses: Various enzymatic and non-enzymatic antioxidants protect against oxidative damage in UVR-exposed skin. These antioxidants are dramatically reduced after UVR exposure. Examples include superoxide dismutase, catalase and thioredoxin reductase. Natural antioxidants like Vitamins A, C and E and glutathione act directly to prevent oxygen radical damage in the skin.

Chronic effects of UVR radiation: The three more common tumor types in humans are basal cell carcinoma, squamous cell carcinoma (SCC) and cutaneous (malignant) melanoma. UVB is a major causal factor for all types of skin cancer. The wavelength dependence for human SCC is very similar to that for human erythema (sunburn). There is a direct link between non-melanoma skin cancer and dipyrimidine DNA photodamage. UVB signature mutations in p53 are very prevalent in SCC and actinic keratosis (AK).

p53 protein is expressed at very low levels in normal human epidermis, but is readily induced by a single dose of solar simulating radiation. p53 responds to DNA damage either by inducing transient cell cycle arrest at G1 checkpoint to facilitate DNA repair OR by triggering apoptotic death. UVB induces human epidermal apoptosis in the form of sunburn cells (apparent within 30 minutes of exposure, maximal at 24 hours). Approximately 2 erythemal doses are required to induce sunburn cells.

So maybe we should just all stay inside … Now on to the dog and cat version…

Diagnostic methods: The importance of a systematic approach to dermatological diseases cannot be over-emphasized, especially in regards to solar dermatitis. Everything starts with the patient’s signalment, including the color of the animal. Lightly pigmented, thinly haired skin is at an increased risk of developing solar dermatitis, actinic keratosis, solar elastosis and squamous cell carcinoma (SCC). White-eared cats have an increased incidence of solar dermatitis and SCC on their pinnae. Beagles, boxers, Dalmatians, Staffordshire bull terriers and Great Danes develop solar-induced lesions on white-skinned, poorly haired regions of their abdomen and thighs.

Solar dermatitis: By definition, an actinic reaction on white skin, light skin or damaged skin (depigmented or scarred areas) not sufficiently covered by hair. The condition develops when skin is exposed to direct or reflected sunshine. The rapidity of onset and severity of reaction is determined by the animal affected, the duration of sun exposure and the intensity of sunlight. The sun’s rays are most intense in the summer months between 9 and 3pm (most severe between 11-2pm) and for every 1000 feet of elevation change, the sun’s intensity increases 4%. Three disease states we will cover include canine nasal solar dermatitis, feline solar dermatitis and canine solar dermatitis of the trunk and extremities.

Canine nasal solar dermatitis: This disease is caused by an actinic reaction in poorly pigmented nasal skin. Dogs may have been born without pigment or the nasal tissue may have undergone spontaneous non-inflammatory depigmentation. Australian shepherds appear to be at an increased risk for this disease. However, any dog with an active or resolved traumatic or inflammatory condition that causes hair loss, depigmentation or scarring of the nasal area is susceptible.

Clinical features of this disease include lesions principally at the junction of haired and hairless skin, but again any area on the nasal planum or face can be affected if it is sparsely haired and lightly pigmented. The area devoid of pigment becomes erythematous and scaly and with continued sun exposure, peri-lesional hair loss is appreciated which leads to the involvement of newly exposed skin. Exudation and crusting develop; ulceration may be seen (especially if rubbing occurs). If intense photoprotection is started early, the affected area can heal completely. However, in most cases, measures are adopted too late and lesions heal by scarring. Typically, the scar includes a larger area than original area of involvement and the scarred area is more susceptible to subsequent solar
and traumatic damage. Progression and enlargement of the lesions can occur each year, especially during more prolonged exposure periods to intense sunlight (summer months; reflection from snow in winter months). More chronic cases present with deep ulcers, loss of tissue at the nares and nasal tip (with exposed nasal tissue bleeding easily) and vertical fissures at the nasal tip. These fissures are often permanent once noted and are primarily located dorsal to and involving the nares. SCC rarely develop, but have been documented. Diagnosis of canine nasal solar dermatitis can be straightforward or complicated, depending on the accuracy of the patient’s history and chronicity. Key points include restriction of lesions to sun-exposed, non-pigmented, sparsely haired skin, onset of signs after solar exposure, absence of skin lesions in the affected area before the current condition began and complete or near-complete resolution of lesions with removal from sunlight. Post-exposure hyperpigmentation is noted in some cases. Early cases present with red, scaly changes to the nasal tissue and normal architecture with adjacent areas of black skin normal in appearance. More chronic cases can be problematic and difficult to diagnose due to the scarring process that occurs. Abnormal skin from previously unrecognized or unreported episodes may complicate the situation and a chronic case will not heal completely with strict photo-isolation. A dog may have chronic solar dermatitis or another nasal skin disease with secondary photodermatitis!

Differential diagnoses for facial dermatosis include: discoid or systemic lupus erythematosus, dermatomyositis, epidermolysis bullosa, pemphigus erythematosus or foliaceus, drug reaction or an infectious folliculitis (bacteria, dermatophyte, demodex, yeast, leishmania). Except for discoid lupus erythematosus (which starts on and remains restricted to perinasal area), these diseases tend to start in haired skin on the bridge of the nose and work towards the nasal planum. These diseases also tend to involve the pinnae and mucocutaneous junctions and there is no predilection for non-pigmented skin. If the nasal planum is extensively ulcerated, fissured and friable, differentials should include vasculitis, neoplasia (SCC, fibrosarcoma, lymphoma) or granulomatous diseases (sterile pyogranulomatous syndrome).

Diagnosis is made via biopsy where histopathology changes include fewer melanocytes and less melanin pigment, epidermal hyperplasia with intraepidermal edema +/- vaculated (sunburn cells) and apoptotic keratinocytes. Perivascular accumulations of inflammatory cells are noted in the upper dermis with vascular dilatation in the lower dermis. Solar elastosis (basophilic degeneration of elastin) is usually not seen and often requires a special stain (PAS) for best visualization. Classic histopathologic finding is bandlike superficial dermal fibrosis. If ulceration is present, there may be a disappearance of epidermis or even dermis and underlying cartilages, depending on severity and depth. Rarely with advanced cases can you see an increase in cells within the basal layer (large polyhedral tumor cells invade the dermis and subcutaneous tissue). SCC eventually develops with cords of neoplastic cells invading the tissue at the level of nasal cartilage.

Clinical management of canine nasal solar dermatitis is variable, but prevention of new lesions is paramount! Photoprotection allows lesions to heal spontaneously in early cases. Corticosteroids to decrease inflammation can be used in more advanced cases, with topical products being the most beneficial, but difficult to apply and unreliable in some patients. Avoidance of direct or reflected sunlight is essential, especially in more chronic cases where solar sensitivity is extreme. Affected dogs should be kept indoors or in the shade during the hours of 9-3pm. However even an indoor dog can sunbathe! The glass of a closed window filters wavelengths <320nm, but an open window or door defeats the purpose. Dogs must also avoid reflections from white concrete sidewalks or the flooring of kennels or outdoor runs. Strict photo-isolation is usually impossible. Sunblocks or sunscreen can allow some sun exposure; addition of artificial pigmentation has also been experimented with. Some benefit may be seen with b-carotene administration (topically applied Vitamin C), but treatment with systemic agents is usually unrewarding. When solar dermatitis has progressed to actinic keratosis or SCC or massive tissue destruction, a poor prognosis is given to the dog as treatments (including retinoic acid, hyperthermia, cryosurgery, surgical excision, photochemotherapy, chemotherapy, radiotherapy, etc) are of little benefit and data on efficacy is limited.

Feline solar dermatitis: Definition- chronic actinic dermatitis of white ears and occasionally the eyelids, nose and lips of cats caused by repeated sun exposure. Theses lesions can progress into an actinic keratosis or true SCC. Feline solar dermatitis occurs in white cats or colored cats with white-haired areas on the face and ears; blue-eyed white cats are the most susceptible. Like the canine counterpart, actinic damage to the ear tip occurs from repeated exposure to UVB light. Early lesions are often ignored or unrecognized. Damage makes the area more susceptible to further actinic insults. This disease occurs mostly in warm, sunny climates like California, Florida, Hawaii, Australia and South Africa.

Clinical features include erythema and fine scaling of the pinnae margins (often earliest sign of the disease) with inflammation causing additional hair loss. There is almost no perceivable discomfort to the cat at this stage of the disease. In susceptible cats, first lesions can occur as early as 3 months of age! More advanced lesions consist of severe pinnaal erythema, peeling of the skin and formation of marginal crusts. At this point, many cats demonstrate pain with scratching of the ears causing further damage. Margins of the pinnae may be curled, too. The margins of the lower eyelids, nose and lips may also be affected, especially in the more susceptible blue-eyed, white haired cats. AK or invasive SCC can develop in some cases (ears, nose, etc) and carcinomatous changes may occur (usually after 6 years, but sometimes as early as 3 years). SCC typically present as an ulcerating, hemorrhagic and locally invasive lesion that is partially crusted and can progress to destroy the pinna. Tentative diagnosis can be made from clinical appearance, color of the cat and history (same quandary as dogs as far as primary or secondary solar dermatitis?).
Differential diagnoses for pinnal lesions include dermatophytosis, early notoedic mange, fight wounds, vasculitis, and possibly frostbite or cryoglobulinemia.

Diagnosis: Biopsy is the preferred method of disease diagnosis and can also detect dysplastic or neoplastic changes. Early stages show superficial perivascular dermatitis with spongiotic hyperplastic changes. Vacolated (sunburn) or apoptotic keratinocytes may be seen, as in the dog. Solar elastosis may be noted in the superficial dermal connective tissue. Formation of a SCC is marked by an ulcerated epidermal surface and invasion of the dermis by nests of polyhedral epithelial tumor cells (variable nuclei size, frequent mitotic figures). Advanced cases show masses of tumor tissue extending to the level of the cartilage.

Clinical management is similar for dogs with solar dermatitis (sun avoidance, sunscreen application). After early irreversible lesions develop, consideration should be given to amputation of the affected ear tips as this process can round off the ears, removes the thinly haired tips and allows the hair to cover and protect the pinna. Cats with AKs that are not candidates for surgery may benefit from treatment with retinoic acids, superficial irradiation (plesiotherapy - handheld strontium probe) or topical imiquimod application. If all therapies fail or the cat has advanced disease at presentation, radical amputation of the pinna may be necessary. Imiquimod (Aldara) is a topically applied immune response modifier whose exact mechanism is unknown, but the product activates the immune system through toll-like receptors. The end result is destruction of transformed cells at site of application. This medication can be applied 2-3 times weekly until active disease is gone (typically 6 weeks or longer). Treated tissues will become inflamed and may be so severe that treatment is discontinued until inflammation (and possibly pain!) is resolved. Imiquimod has been used in veterinary medicine for the treatment of horses with flat warts (aural plaques) or sarcoïds and cats with virally-induced SCC in situ or solar dermatitis.

Canine solar dermatitis of the trunk and extremities: A combination of factors are necessary for sun damage to occur: skin must be unpigmented or poorly pigmented; only a sparse hair coat covers the skin, allowing UV rays of sun to reach the epidermis; area must be exposed to the sun (dogs that like to sunbathe, dogs confined to areas where no sun shelter is available). The nose and ears are the most exposed and most susceptible to actinic damage, but other regions of the body can be affected. Predisposed breeds include the Dalmatian, American Staffordshire terrier, German shorthaired pointers, white boxers, Whippets, Beagles and white bull terriers. Dogs that sunbathe in right or left lateral recumbency will present with their flanks and ventrolateral abdomen most affected. Lesions can also be seen on the nose, ears, tail tip or distal limbs. Dogs that sunbathe on their backs or that are caged on wire above white concrete/reflective surface can present with the entire ventrum involved. The duration of sun exposure influences the damage done to the skin. A single prolonged exposure can result in full-thickness necrosis. At first, regular sunbathing occurs and affected areas are erythematous and scaly. 1-3 days later, the affected skin becomes tender and can become necrotic. With repeated, but episodic exposure, initial sunburning is followed by actinic folliculitis, actinic follicular cyst formation or dermal fibrosis. White areas of the skin are thinned whereas the black pigmented skin is normal. Biopsy reveals variable degrees of superficial perivascular dermatitis and folliculitis with prominent superficial dermal fibrosis +/- solar elastosis. With more chronic exposure, sunburned areas become thicker and dogs can develop erosions, ulcerations, crusting and comedones. Occasionally necrosis is noted along with fistulization and scarring. Biopsy may reveal follicular cysts, pyogranulomatous inflammation and premalignant actinic keratosis. Finally, a SCC can develop, especially if the dog continues to be exposed to direct sunlight. SCC should be removed surgically and the procedure repeated if necessary (often it is, multiple times!). There is always the danger of metastasis to regional lymph nodes, but most SCC that are identified and treated early are more locally invasive. Skin with solar damage is also more likely to develop a hemangiomia or hemangiosarcoma. The most successful and primary therapy for canine solar dermatitis of the trunk and extremities is photoprotection. At risk or affected dogs should be kept out of the sun and topical sunscreens or t-shirts utilized, if practical. Δ-carotene with anti-inflammatory doses of prednisone or prednisolone have been reported as effective in early cases. Retinoids may also be of some benefit; Imiquimod should be effective with early lesions, but often the large surface area that needs treatment makes this an impractical option. A dense hair coat protects most small animals from excessive exposure to sunlight. Some dogs have pigmented skin which also protects from UV radiation damage. However, when non-pigmented, sparse or non-haired skin is exposed to sunlight, solar damage may occur. Some animals respond with hyperpigmentation, others may incur sunburn or solar dermatitis. More chronically exposed cases may present with solar-induced neoplastic lesions (SCC, hemangiomia or hemangiosarcoma).

Sunblock: Opaque agents that reflect and scatter incident light constitute sunblock. White or colored zinc oxide preparations are the most common human products that may be of benefit to dogs. Many sources caution the use of these products as the dog may lick the product off and lead to zinc intoxication, but this is unlikely. Most people prefer clear sunscreen products, but these absorb incident light (typically only UVB light, but some products contain ingredients to screen UVA as well). Waterproof products with an SPF of 30 or greater should be used as these absorb >96% of incident UVB which is sufficient to protect most animals. Sunscreen products should be applied and gently rubbed into the skin 15-30 minutes before sun exposure and reapplied at least twice daily on a regular basis. Topical sunscreens may act physically or chemically. Chemical sunscreens, like aminobenzoic acid or benzophenone derivatives, act to absorb UV rays and are available as clear, cosmetically acceptable lotions or gels. Physical sunscreens that contain zinc oxide and titanium dioxide act to reflect and scatter light by forming an opaque barrier on the surface of the skin. These products are available in a variety of colors, are water resistant and not easily removed by the pet, but can be messy, especially in long-haired
dogs. Sunscreens are rated for efficiency by sun protective factor (SPF). SPF 2-4 is a mild blocker; 8-10 ranked as more moderate protection; 15 or higher giving more complete blockage (SPF >30 recommended). The frequency and thickness of application, temperature, humidity, potency of light exposure, a patient’s sensitivity and many other factors affect the results of sunscreen application. One condition that makes sunscreen use in our canine and feline patients less successful is that sunscreen should be applied 3-4 times per day for greatest effectiveness. Photo-decomposition is a problem with many sunscreens, but products containing titanium dioxide or zinc oxide tend to resist this phenomenon. These products are preferred if repeat application is not possible, but in the human field, these products also have a higher incidence of irritation and contact reactions. A common misconception is that a dog licking the area where sunscreen was applied will remove the sunscreen. This is true for physical blockers (zinc oxide for example), but not so much for chemical blockers as they are absorbed into the skin and pool within the stratum corneum as a reservoir of protection. Oral sunscreens contain chemicals like b-carotene and chloroquine which act to quench free radicals and stabilize cell membranes. These products have not been proven to prevent sunburn in humans, but have been useful in cases of light-induced dermatitis. A b-carotene derivative canthaxanthin has been used in cats and dogs to reduce phototoxicity, but the safety of this product has been challenged (side effects included orange-brown skin, brick-red stools, crystalline gold deposits on the retinae and orange-colored plasma). Sun suits are an alternative to more regular sunscreen application and are available as lycra body suits that are lightweight, breathable and offer more full coverage, but the head, paws and tail are still exposed. Artificial pigmentation (applying black ink to the surface of the pet’s skin) can be beneficial, but does not negate the need for other measures. Black skin can still absorb some sunlight and be burned. Felt tipped markers, permanent laundry or stamp pad ink or even tattooing can be used to darken a patient’s skin, but these processes have limited use currently due to irritation caused by solvents in markers, poor results, expense of tattoo equipment and need for multiple treatments under general anesthesia. Topical retinoids, particularly the third generation polyaromatic retinoids (Tazarotene, Adapalene, Bexarotene), can be used topically on affected skin to help reduce the cystic and comedone-like changes to the skin. Our Australian contingency commonly uses Solaraze (diclofenac sodium), a non-steroidal anti-inflammatory medication, on localized actinic lesions, but also the oral retinoid acitretin (1mg/kg q24 hours for ~6 weeks). In the United States, veterinarians can no longer prescribe oral retinoids as easily and caution must be taken for their use in pets (recommended to monitor bloodwork and tear production regularly).

Actinic keratosis: AKA actinic carcinoma in situ or solar keratosis. AKs are epithelial plaques that form due to solar exposure. Keratosis is a non-specific term referring to horny or warty growth or callosity. AKs are the only commonly occurring keratosis in dogs and cats. The incidence of AKs varies substantially with geographic location, climate and behavior with predisposing factors being lower latitude, higher altitude, arid or semi-arid environment and prolonged sun exposure. AKs may present as single or multiple, plaque-like or papillated lesions, usually 1cm in diameter or smaller. There is frequently prominent scale, crust or scabs noted. AKs occur most commonly on the pinnae, nose and eyelids of white-faced cats or the ventral abdomen, ventral flanks and medial thighs of short-coated, white-haired or piebald breeds of dogs (Dalmatians, Pit Bull terriers, Beagles, basset hounds). Histopathology reveals irregular hyperplasia and moderate to severe laminated keratosis (atypia to dyskeratosis).

Hemangioma (angiomas): Uncommon in dogs; rare in cats. Hemangiomas are benign neoplasms arising from endothelial cells of blood vessels. Studies strongly suggest that chronic solar damage may be the cause of hemangiomas in ventral glabrous skin of lightly pigmented, sparsely haired dogs, but there is no evidence to support UV induction of these lesions in cats. Hemangiomas are more common in dogs than hemangiosarcomas and frequently affect dogs with an average age of 10 years and no sex predilection. Hemangiomas are frequently located on the lightly pigmented and sparsely haired ventral abdominal and thoracic skin. Breeds predisposed to their development include the boxer, golden retriever, German shepherd dog, English springer spaniel, Airedale terrier, whippet, Dalmatian, beagle, American Staffordshire terrier, basset hound, saluki and English pointer. These lesions are usually well circumscribed, firm to fluctuant, rounded, bluish to reddish black, 0.5-4cm in diameter and dermal to subcutaneous in location. Lesions in cats are similarly described, but hemangiomas are less common than hemangiosarcomas and again occur in patients greater than 10 years of age (most often male cats). Lesions are typically solitary and found on the ears, face, neck and limbs. Spontaneous or trauma-induced bleeding may occur in either species. Hemangiomas are diagnosed via biopsy and the finding of proliferation of blood-filled vascular spaces lined by single layers of well-differentiated endothelial cells. These lesions can be sub-classified as either cavernous or capillary, depending on the size of the vascular spaces and amount of intervening fibrous tissue. Solar-induced lesions are often less well circumscribed and both solar dermatitis and elastosis are also present. Hemangiomas stain positive with vimentin, Factor VIII-related antigen (vWF), Type IV collagen and laminin, markers all found within vascular proliferations. vWF and CD31 (platelet endothelial cell adhesion molecule) mark normal vascular endothelium and all hemangiomas. Electron microscopy reveals Weibel-Palade bodies, a specific cytoplasmic marker for endothelial cells. Patients may present with hematologic abnormalities (anemia, thrombocytopenia, hypofibrinogenemia or other findings associated with disseminated intravascular coagulation) or have unremarkable bloodwork findings. Management includes surgical excision, cryosurgery, electrosurgery and observation without treatment.

Hemangiosarcoma (angiosarcomas, malignant hemangioendotheliomas): This is an uncommon malignant neoplasm of dogs and cats that arise from endothelial cells of blood vessels. It has been suggested that chronic solar damage may be the cause as again
ventral glabrous skin of lightly pigmented, sparsely coated dogs and pinnae of white-eared cats are frequently affected. It is important to distinguish between superficial dermal hemangiosarcomas (chronic UV exposure) and subcutaneous hemangiosarcoma as the dermal variation carries a much better prognosis for the pet. Affected dogs average 10 years of age with no sex predilection. German shepherd dogs, golden retrievers, Bernese mountain dogs and boxers more commonly develop hemangiosarcomas, but the breeds listed for hemangiomas are also at an increased risk for solar-induced superficial dermal hemangiosarcoma. These lesions are often rapidly growing and located on the trunk or extremities. Solar-induced dermal hemangiosarcomas often present as multiple lesions on the ventral thorax and abdomen that are well to poorly circumscribed, red to dark blue plaques or nodules and usually less than 2cm in diameter. Subcutaneous hemangiosarcomas (not solar induced) can be solitary or multiple and are poorly circumscribed, dark red or blue-black, bruise-like spongy masses that can measure up to 10cm in diameter. Dermal or subcutaneous hemangiosarcomas may also present with alopecia, thickened skin, hemorrhage and ulceration. An epithelioid variant was recently described in dogs and has similar behavior to the subcutaneous form of hemangiosarcoma. In similar fashion, affected cats are 10 years of age or older and white cats may be prone to superficial cutaneous hemangiosarcoma due to UV exposure. Lesions are usually solitary and rapidly growing and occur most frequently on the head and pinnae, limbs and inguinal or axillary regions. Dermal and subcutaneous forms are present, just as described in the dog. Biopsy reveals an invasive proliferation of atypical endothelial cells with areas of vascular space formation. Solar-induced lesions may have associated solar dermatitis and elastosis. Hemangiosarcomas are also special stain positive (vimentin, S-100 protein, Factor VIII-related antigen [vWF], Type IV-collagen, laminin). In one study, vWF marked 73% and CD31 marked 100% of canine hemangiosarcomas investigated. Anemia, purpura, thrombocytopenia, hypofibrinogenemia and findings associated with disseminated intravascular coagulation have been reported. An epithelioid variant of hemangiosarcoma has been reported with a distinct cellular morphology (epithelioid appearance of neoplastic endothelial cells and occasional cytoplasmic vacuolization). It is important to remember that visceral hemangiosarcomas can metastasize to the skin and if this is suspected, staging of the patient is recommended. Diagnostics to stage a patient with suspected visceral disease include CBC, chemistry, U/A; thoracic radiographs with abdominal ultrasound; echocardiogram. Therapy of choice for hemangiosarcoma is radical surgical excision, however after any form of therapy, prognosis is guarded. Local recurrence and metastasis of subcutaneous hemangiosarcoma is common. These tumors are highly invasive and malignant in dogs with average survival times reported at <6 months after the time of diagnosis. Palliative radiation and/or doxorubicin and vincristine have been used as systemic chemotherapeutic agents. Hemangiosarcomas frequently recur after surgical excision in cats, but the metastatic potential for feline cutaneous hemangiosarcoma appears variable (different rates reported in different studies). Amputation is usually curative when on a distal limb or digit. Canine cutaneous hemangiosarcomas are staged based on depth of histologic location: stage I (dermal), II (subcutis) and III (down to muscle). Stage I lesions are often small, raised red-purple nodules (likely solar induced), commonly found on ventral abdomen, prepuce and pelvic limbs. These are relatively non-invasive tumors and have a relatively benign behavior (median post-surgical survival time of 780 days). Stage II and III lesions are larger, poorly circumscribed, soft to fluctuant, often bruise-like with no anatomic site predilection. These lesions carry a shorter median post-surgical survival time of 172 days (stage II) and 307 days (stage III). With concurrent vincristine, doxorubicin and cyclophosphamide (VAC) therapy, 6 dogs with subcutaneous stage II hemangiosarcoma had 425 days MST.

Squamous cell carcinoma (epidermoid carcinoma): Common malignant neoplasm of dogs (1 tumor); also affect large animals. SCC arise from squamous epithelial cells (keratinocytes) and are most frequently found in sun-damaged skin (preceded by actinic or solar keratosis). SCC rarely arise from burn scars or chronic infections, but have been reported in cases of more chronic discoid lupus erythematosus and multiple canine infundibular cysts. 50% of canine SCCs tested were positive for papillomavirus structural antigens (suggesting an etiologic role for viruses?). Similar studies were negative in cats (but 45% of Bowen’s disease [multicentric squamous cell carcinoma in situ] cases were positive). Dogs average 9 years of age at the time of diagnosis with no sex predilection, but Scottish terriers, Pekingese, Boxers, Poodles and Norwegian elkhounds are breeds more commonly affected. SCC at the claw-bed has been noted in larger breed black-coated dogs (Labradors, Standard poodles, giant Schnauzers, Dachshunds, Bouvier de Flandres). Canine solar-induced SCC are more often found in short-coated breeds with white or piebald ventral coat and skin color (Dalmatian, American Staffordshire terrier, Bull terrier, Beagle, etc) and affected dogs often spend many hours each day in the sun. Canine nasal SCC is rarely a sequela to depigmentation (discoid lupus erythematosus, pemphigus erythematosus, vitiligo). Lesions are found on the trunk, limbs, digits, scrotum, lips, anus and nose of dogs and can be proliferative or ulcerative. Proliferative SCCs are papillary masses with a cauliflower-like appearance and frequently have an ulcerated surface that bleeds easily. The ulcerative form presents as a shallow, crusted ulcer that can deepen and become crateriform. Lesions are usually solitary, but multiple lesions can occur on the trunk of sunbathers or at numerous clawbeds. Subungual SCC often affects a single digit and these lesions are swollen and painful. SCC is the most common neoplasm of the digit of the dog. Cats average 9 years of age with no breed or sex predilection. White cats (both short or long-haired) are affected by SCCs 13 times more frequently than other cats as they have an increased susceptibility to actinic damage. External nares (80-90%), pinnae (50%), eyelids (20%) and lips are affected; SCC of the nasal planum is rare. Feline lesions, like the dog, are either proliferative or ulcerative and multiple lesions
occur in 45% of cats. Generally, SCC in cats are locally invasive and slow to metastasize, but SCC arising from the digits are more aggressive (may be misdiagnosed as paronychia or pyoderma).

**Differential diagnoses include (solitary tumors) any number of neoplastic or granulomatous diseases; clawbed origin can resemble an infectious paronychia.**

Histopathology shows irregular masses or cords of keratinocytes with a downward proliferation and invasion of the dermis. Keratin formation, horn pearls, mitoses and atypia are noted +/- solar elastosis. These tumors are typically well differentiated within the skin. SCC stain cytokeratin positive. SCC are locally invasive and slow to metastasize, but metastasis to local lymph nodes and lungs have been reported (often in advanced or poorly differentiated neoplasms). Clawbed disease is more aggressive with ~70% of tumors invading the bony tissue of P3. Clawbed SCCs metastasize more frequently (up to 22% to regional lymph node). The prognosis in cats correlates with the degree of histopathologic differentiation. Clinical management includes surgical excision, cryosurgery, electrosurgery, hyperthermia and radiotherapy. Amputation can be performed either of the pinnae (pinnectomy) or nasal planum (nosectomy). Chemotherapy protocols include bleomycin, vincristine, hydroxyurea, cisplatin, 5-fluorouracil, benzaldehyde, thioprine and oral retinoids, but all are typically ineffective. Avoidance of sunlight is the most important and successful form of “therapy” with sun suits being more effective/realistic than sunscreen or tattoos, etc. in already affected animals.

Miscellaneous effects of solar exposure: Exposure to UV light is an important factor in precipitating and potentiating a number of skin lesions. UV light may also exacerbate generalized systemic disease activity. Patients with discoid or systemic lupus erythematosus, pemphigus erythematosus or pemphigoid disease should avoid sun exposure just as our predisposed solar breeds.

**References**

It’s All About the Crust:
A Case-Based Approach to the Crusty Patient
(Parts 1 and 2)
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Meghan Trainor likes to sing that it’s “all about that bass,” but, if you are a dermatologist, it’s all about the crust, about the crust. Crust is defined as dried exudate, “a hard outer layer or covering; cutaneous crusts are often formed by dried serum or pus on the surface of a ruptured blister or pustule.” Scale, on the other hand, is composed of exfoliated keratinocytes, “a thin flake or compacted plate-like body, as of cornified epithelial cells.”

Sometimes one crusty dog looks an awful lot like another crusty dog… diagnostic tests to consider when facing the crusty patient include impression smears for cytology of the skin and/or ears, skin scrapes (superficial or deep), dermatophyte culture, aerobic culture of the skin/ear, and multiple punch biopsies for histopathology. For each case presented, we will discuss what diagnostics were performed and why (no diagnostics, empirical therapy; cytology of the skin/ear; skin scrape - superficial vs. deep; dermatophyte culture; aerobic culture of the skin/ear; multiple punch biopsies for histopathology).

Smears of pustules and exudative lesions can help establish the presence of organisms (bacteria, yeast, both; fungal spores). However, a negative impression smear could have been a sampling error (repeat to check yourself!) or could represent a non-infectious process. Impression smears also allow us to assess the inflammatory infiltrate (what cells types are present? intracellular vs. extracellular organisms?). Acantholytic cells can be detected on impression smears from pustular lesions (or recently ruptured lesions and ulcerated skin); neoplastic or atypical cells may also be identified. Diseases where impression smears are beneficial are more superficial bacterial skin infections, systemic and opportunistic fungal diseases, otitis externa, pemphigus foliaceus and mycosis fungoides.

Equipment needed for impression smears are glass microscope slides, +/- wooden cotton applicators, a heat source for sample fixation, Diff-quick staining materials and a microscope with a scanning lens (4x) and oil immersion lens (100x). Personal preference will dictate if you perform direct impression smears or tape-prep, one slide versus multiple slides, etc.

Dermatophyte culture is the most accurate method of diagnosing dermatophytosis in all species. Hair and scale specimens are collected and grown on dermatophyte test media. In general, fungal cultures are 90% effective as compared to a skin biopsy (historically 60-70% effective), but skin biopsy results are available in a more timely fashion (a few days versus up to 21-30 days for DTM). There are two techniques for collecting samples for a dermatophyte culture. Hair plucks are utilized when there are visible lesions (hypotrichosis, barbered to more complete alopecia, scaling debris, etc.). The Mackenzie toothbrush technique comes in handy when sampling animals with more mild and/or generalized symptoms versus a more active, focal lesion. Asymptomatic cats in a household with a known infected cat can be screened with this technique, brushing the entire cat with a sterile toothbrush. The culture plate is then inoculated with the collected material.

There are several options for dermatophyte test media; I most commonly use the Sab-duet media culture. The Sab-duet media culture has two halves of media to help grow and isolate fungus. Dermatophyte test media (DTM) contains antibiotics and antifungal agents to present contamination with saprophytes (cycloheximide, gentamicin, chlorotetracycline). Pathogenic dermatophytes produce an alkaline (increased pH) product, changing the phenol red pH indicator from yellow to deep red. Dermatophytes utilize protein metabolism first (red color change) while saprophytes metabolize carbohydrates first (yellow color change). The DTM side must be checked regularly (if cultures are grown in-house); this side of the culture duet does not enhance sporulation of dermatophytes. The second side is the mycobiotic agar (sabouraud’s dextrose agar). This medium also contains antibiotics (chloramphenicol and cycloheximide) to help select for pathogenic fungi. By lacking a pH indicator (which can interfere with pigment-producing fungi), development of classical pigmented colony color and morphology of pathogenic fungal species can occur. This culture media also encourages sporulation via pancreatic digest of soy bean meal, dextrose and agar. A Derm-duet product contains RSM and DTM. RSM is rapid sporulation medium, which is similar to DTM. There is a color indicator (yellow to blue-green) which can revert from blue to light blue-green with prolonged incubation. RSM also contains antibacterial and antifungal compounds and enhances the sporulation of dermatophytes. Interpreting a dermatophyte culture is just as important as sample collection. Most dermatophytes are positive within 3-7 days, with a progressive red color change of the DTM medium (original yellowish-orange color). Most saprophytic fungi and bacteria will be inhibited on both DTM and RSM plates (if a saprophyte or contaminant does grow, it will not change the color of the culture indicator in an appropriate manner). Cultures are incubated at room temperature for 12-30 days; slow growers may take the full 3 week time period (must remember to monitor these daily to avoid growth of contaminants). If you are reading your own cultures, once you have colony growth, a tape prep of the colonies from the mycobiotic side of the fungal culture (or RSM) is prepared with a drop of lactophenol blue stain and examined under lower microscope settings. False negative cultures may be obtained as some organisms are sensitive to cycloheximide. (Cryptococcus neoformans, Pseudallescheria boyolli, Actinomyces spp.,...
**Staphylococcus**

restraint or with sedation or general anesthesia. Minimal to no preparation should be performed at the biopsy sites as this and handled gently as removed from the skin and placed in formalin. Biopsies may be collected with local

and "newer" lesions should be selected as they are more likely to be diagnostic. When possible, multiple representative samp

chronicity, determine the permanency of changes and help evaluate response to therapy. A biopsy should be performed as soon

other dis

minocycline, amikacin, etc.).

hours; a cytology is recommended at the time of culturing to ensure culture results match bacteria seen at time of collection

time to perform a culture of the skin or ear? If rods are present versus more "predictable" cocci bacteria? Based on the re

pseudintermedius, S. schleferi) lesions. Most exudative inflammatory skin diseases will be overgrown with coagulase

capillary bleeding. Certain mites are associated with sarcoptes, notoedres, demodex gatoi, cheyletiella and general surface scale; otodectes and the ear canal or periauricular region.

into the epidermis (demodex canis, demodex cati) just to the point of capillary bleeding. Certain mites are associated with more classic lesion locations: sarcoptes and ear margins, lateral elbows and hocks; demodex gatoi and the ventral abdomen, lateral shoulder; cheyletiella and general surface scale; otodectes and the ear canal or periauricular region.

An aerobic bacterial culture and sensitivity can be performe

Wood’s lamp evaluation can also help with the diagnosis of dermatophytosis (and/or help select areas to sample). A specialized UV lamp can locate affected hairs as certain dermatophytes produce a by-product that fluoresces a blue-green color (tryptophan metabolite produced on the animal). Organisms that can be Wood’s lamp positive include M. canis and M. audouinii (~30-50% of M. canis; some studies report up to 90% positive). Organisms also positive on Wood’s lamp include M. distortum and Trichophyton schoeneinii. A Wood’s lamp produces a 253.7 nanometer wavelength; the lamp must be warmed up for 5-10 minutes due to wavelength stability and intensity being temperature dependent. Positive fluorescence is indicated by a blue-green coating of the hairs; underlying skin and scales should not be affected. False positives can be obtained if certain medications or hair conditioners are present as skin and hairs will fluoresce; scaling or crust may give a yellowish color and suspect hairs should be cultured for confirmation. A negative Wood’s lamp examination does not exclude the possibility of dermatophytosis.

Wood’s lamp can locate affected hairs as certain dermatophytes produce a by-product that fluoresces a blue-green color (tryptophan metabolite produced on the animal). Organisms that can be Wood’s lamp positive include M. canis and M. audouinii (~30-50% of M. canis; some studies report up to 90% positive). Organisms also positive on Wood’s lamp include M. distortum and Trichophyton schoeneinii. A Wood’s lamp produces a 253.7 nanometer wavelength; the lamp must be warmed up for 5-10 minutes due to wavelength stability and intensity being temperature dependent. Positive fluorescence is indicated by a blue-green coating of the hairs; underlying skin and scales should not be affected. False positives can be obtained if certain medications or hair conditioners are present as skin and hairs will fluoresce; scaling or crust may give a yellowish color and suspect hairs should be cultured for confirmation. A negative Wood’s lamp examination does not exclude the possibility of dermatophytosis.

Skin scrapings should be performed when you are looking for ectoparasites (mites, mite eggs, other larval arthropods and helminth larvae burrowing into epidermis; dermatophyte arthrospores attached to hairs). A positive skin scrape means a definitive diagnosis while a negative skin scrape can be inconclusive... that particular sample did not reveal suspected parasite (so truly negative) or we missed 'em. Equipment needed for skin scrapes includes a scalpel blade (versus flat-bladed medical spatula), mineral oil, glass microscope slides +/- cover slips and a microscope (4x and 10x lenses). If you suspect a follicular mite, you can squeeze the skin, forcing the parasite into a more superficial portion of the hair follicle; scrape perpendicular to the skin surface in the direction of hair growth, scooping material from the pet onto the microscope slide. A superficial skin scrape (into the stratum corneum) will identify sarcoptes, notoedres, demodex gatoi, cheyletiella, otodectes and chiggers. A deeper skin scrape is performed for parasites burrowed into the epidermis (demodex canis, demodex cati) just to the point of capillary bleeding. Certain mites are associated with more classic lesion locations: sarcoptes and ear margins, lateral elbows and hocks; demodex gatoi and the ventral abdomen, lateral shoulder; cheyletiella and general surface scale; otodectes and the ear canal or periauricular region.

An aerobic bacterial culture and sensitivity can be performed if secondary bacteria are the cause of or complicating crusted skin lesions. Most exudative inflammatory skin diseases will be overgrown with coagulase-positive staph (Staphylococcus pseudintermedius, S. schleferi) and a positive culture confirms bacteria is present, but not diagnostic for infection. When is the right time to perform a culture of the skin or ear? If rods are present versus more “predictable” cocci bacteria? Based on the response to previously administered antibiotics? First time infection? It is best to culture the skin or ear when off systemic antibiotics for 48-72 hours; a cytology is recommended at the time of culturing to ensure culture results match bacteria seen at time of collection. With increasing numbers of resistant staphylococci, it is important to request extended antibiotic spectrum (CHPC, doxycycline, minocycline, amikacin, etc.).

A biopsy is indicated to help establish a definitive diagnosis or narrow down the category of disease(s). A biopsy may rule-out other disease processes and can be used to predict prognosis for doctor and client alike. Biopsy may help assess the degree of chronicity, determine the permanency of changes and help evaluate response to therapy. A biopsy should be performed as soon as possible as chronicity, self-trauma and topical or systemic therapy can potentially obscure diagnosis. Lesions unaffected by trauma and “newer” lesions should be selected as they are more likely to be diagnostic. When possible, multiple representative samples should be taken and, within reason, “go big or go home” attitude should be taken for biopsy size. Indications for a punch biopsy are multiple lesions, variable lesions and difficult sites (nasal planum, footpads, pinnae). Punch biopsies may be taken entirely within the boundary of the lesion or partial lesion and partial normal adjacent skin. Punch biopsy samples should be rotated in a single direction and handled gently as removed from the skin and placed in formalin. Biopsies may be collected with local anesthesia and physical restraint or with sedation or general anesthesia. Minimal to no preparation should be performed at the biopsy sites as this may disturb the biopsy results (it is all about the crust, after all!). Local 2% lidocaine and bicarbonate mix at a 50/50 ratio can be used to
undermine the biopsy sites ~5 minutes before biopsy sampling is performed. A 10% neutral phosphate buffered formalin solution is the fixative of choice; the volume of fixative should be at least 10 times that of the specimen submitted.

Audience participation

Diagnostics to consider performing

- No diagnostics, empirical therapy
- Cytology of the skin/ear
- Skin scrape- superficial vs. deep
- Dermatophyte culture
- Aerobic culture of the skin/ear
- Multiple punch biopsies for histopathology

Diseases covered in our lecture (listed alphabetically to keep things fun and interactive):

- Atopic dermatitis
- Demodicosis
- Dermatophytosis
- Discoid lupus erythematosus
- Erythema multiforme / Drug reaction
- Mycosis fungoides
- Pemphigus foliaceus
- Sebaceous adenitis

References
Light on notes, this presentation is full of clinical photographs that are more striking and, in some cases, pathognomonic for one of the dermatologic diseases listed below.

A brief description of each disease noted below will be included in our slideshow. However, the focus of this 2-hour lecture series is to highlight physical exam findings that may help put you on the path of diagnosing potentially more complicated dermatology cases or cases you may not run across every day in general practice.

**Audience participation is strongly encouraged**

**Diseases represented (in alphabetical order, to keep things fun):**

- Allergy (adverse food reaction vs. atopic dermatitis vs. flea allergy dermatitis)
- Alopecia areata
- Calcinosis cutis – natural vs. idiopathic
- Canine eosinophilic furunculosis (of the face)
- Canine leproid granuloma
- Ceruminous cystomatosis
- Color dilution alopecia
- Demodiosis – D. canis, D. injai, D. cati, D. gatoi
- Dermatophytosis
- Discoid lupus erythematosus
- Erythema multiforme
- Exfoliative cutaneous lupus erythematosus
- Feline paraneoplastic syndrome
- Hypothyroidism
- Ischemic dermatopathy
- Metacarpal fistulae of GSD
- Mycosis fungoides
- Mucocutaneous pyoderma
- Nasal arteritis
- Pemphigus foliaceus
- Perianal fistulae
- Plasma cell pododermatitis
- Pyotraumatic dermatitis
- Rabies vaccine induced vasculitis
- Sebaceous adenitis
- Solar dermatitis
- Squamous cell carcinoma
- Sterile nodular panniculitis
- Symmetric lupoid onychodystrophy
- Vasculitis / vasculopathy
- Vitiligo

**References / good dermatology books to consider:**

- Muller & Kirk’s Small Animal Dermatology
- Hnilica’s Small Animal Dermatology Color Atlas
- Gross, Ihrke, et al. Skin Diseases of the Dog and Cat
It is easy to get overwhelmed by the large number of topical products available as a prescription item as over the counter products for the skin and hair coats of our small animal patients. The goals of today’s lecture will be to provide a quick review of the foundations of atopic dermatitis and the basics of topical therapy, from a dermatologist’s perspective. This includes setting realistic treatment goals which can sometimes involve matching the client and patient with the appropriate product(s). We will review the different forms of topical products and highlight some of the newer products available.

Atopic dermatitis: defective epidermal barrier function? In humans, insufficient extrusion of lipid-containing organelles into the superficial epidermal intercellular spaces and skin lipid metabolic defects have been documented. Xerosis is the term given to an abnormal skin dryness seen with atopic dermatitis (AD) patients and is manifested as an increase in trans-epidermal water loss and defective extrusion of lamellar bodies. The outermost layer of the epidermis is composed of desquamating corneocytes surrounded by intercellular lipids. Lipids function as a major protective role in the epidermis. Linoleic acid-enriched diets are associated with decreased trans-epidermal water loss, suggesting the oral fatty acids are incorporated into epidermal intercellular lipids.

Environmental allergens contributing to symptoms of AD include dust mite and storage mite antigens, house dust, pollens from grasses, trees and weeds, mold spores, epidermal antigens and insect antigens, just to name a few. The assumption that these substances are biologically relevant to the pathogenesis of AD is inferred from several ideas. One, dogs with clinical signs of AD react to these antigens (ie. intradermal skin testing wheal and flare, in vitro elevated IgE and IgG demonstrated on Western blotting or ELISA). Two, clinical signs of AD improve with allergen immunotherapy selected from the above tests. And three, the assumption that canine AD is a similar disease to human atopy (asthma, allergic rhinitis, AD).

Atopic dermatitis: the route of allergen challenge. Allergic inhalant dermatitis refers to inhaled allergens penetrating the respiratory tract, migrating in circulation to the skin and triggering dermal mast cells. This idea comes from anecdotal clinical cases and asthma cases without skin lesions. Cutaneous absorption refers to environmental antigens transferring through the stratum corneum to contact antigen presenting cells in the epidermis, initiating cutaneous inflammation. Cutaneous absorption is sup ported by lesion location (hairless or frictional areas) and the cell types present in lesional atopic skin versus normal skin. Atopic dermatitis was initially considered an “inside-out” disease, where epidermal barrier damage was the end result of inflammatory skin disease. An “outside-in” theory is favored as the abnormal epidermal barrier is the primary cause of disease, allowing penetration of allergens and antigens, triggering inflammation in the underlying skin.

There are two ways to think of pruritus related to atopic dermatitis. The summation of effects phenomenon refers to the presence of multiple stimuli contributing to the level of pruritus (bacterial or yeast infections; flea or food hypersensitivity). Patients may tolerate some pruritic stimulus without becoming itchy, but multiple stimuli exceed the threshold and pruritus is seen due to the summation of effects of different diseases. Threshold phenomenon, on the other hand, is related to allergen load. When the allergen load is low, symptoms will not be shown; however, with heavy allergen load, clinical disease is triggered. This explains why animals that show sensitivity to multiple allergens may be successfully managed with hyposensitization that does not include every single allergen.

Topical therapy can be used as an adjunct to systemic therapy, to potentially reduce the need for systemic medications, reduce side effects and act synergistically with systemic therapy. Topical therapeutics may also be the sole therapy needed for conditions like mild sebaceous adenitis, mild atopic dermatitis, secondary pyoderma or more greasy canine patients.

Limitations of topical therapy: first and foremost, owner compliance! Topical therapy can be time-consuming and labor intensive. If our clients can fail, so can our patients! They may fight to get into the tub or shower and may lick or groom the medications off of their skin and hair coat. Localized adverse reactions are rare, but some pets may be worse after topical treatments. For example, some ingredients may be drying or irritating; some may contain perfumes or dyes; there is also a difference in the pH balance of human products versus veterinary-specific products. Anatomy… hair is a barrier, it is just doing its job! An intact stratum corneum is also a barrier to penetration of topical products. Topical therapy also increases the cost of therapy as good quality products are not cheap but may decrease money spent elsewhere (less systemic medications?) and a more “natural” approach is often preferred by many clients.

Keep in mind that a certain amount of topical medication is absorbed systemically. Increased temperature and dermal blood flow will increase this absorption while vasoconstriction decreases absorption. Iatrogenic Cushing’s is possible with topical glucocorticoids, especially in smaller breed dogs.
General principles of topical therapy: we want to obtain a definitive diagnosis (we should be doing this for systemic therapy, too!) and we want to understand the formulations of the products (choose the appropriate vehicle and agent and know the effects and potential side effects).

Shampoos are the most common delivery system in veterinary medicine. Baths function to cleanse the skin, remove scale, crust and debris and then deliver medication to the skin. Common agents in shampoos may be anti-bacterial, anti-fungal, anti-seborrheic, anti-parasitic and anti-pruritic. You may consider clipping the hair if relying heavily on topical treatments, but be careful not to further “seed” an infection into the skin with clippers and trauma. Bathe in cool, lukewarm water (hot water can exacerbate pruritus), with a minimum contact time of 8-10 minutes. Frequency of bathing may vary with the severity of lesion(s), owner compliance and product(s) used. “Lather, rinse and repeat” may be especially important if a patient is particularly dirty… oil and grease from a patient can act as an anti-foaming agent. It is a common complaint that many medicated shampoos do not lather well, but neither do baby (human) shampoos and is a consequence of lathering agents being detrimental to ingredients in the medicated shampoos. Some owners may benefit from an initial cleaning bath with a less expensive over the counter shampoo and then focus on the trouble spots with the preferred medicated product.

What is so special about a prescription shampoo? How do we justify our dog’s shampoo costing more than our own shampoo? Enhanced technology of the medicated veterinary products translates to increased efficacy and treatment success. Examples include phytosphingosine (Sogeval / Ceva), novosomes (Vetoquinol), spherulites and glycotechnology (Virbac) and various enzymes (Zymox). Phytosphingosine is a ceramide and ceramides are the major lipid component of the stratum corneum, providing the barrier property of the skin and controlling local flora. Novosomes are made up of a central holding area of water, lipids and specific shampoo ingredients that are enclosed in an outer membrane which slowly releases the solution as the layers break down. Another technique to achieve more time-released medication after the shampoo has been rinsed off are the spherulites in Virbac’s products. These spherulites are composed of multiple layers of medication and chitosanide to help form a film over the skin and hair. Glycotechnology refers to glycosugars present in the shampoo to saturate binding sites on the surface of bacteria, decreasing adherent and inflammation. Zymox products contain three enzymes derived from milk products (lysozyme, lactoperoxidase, lactoferrin) with unique antimicrobial properties that can oxidize the components of bacterial cell walls.

Soaks / wet dressings can provide good coat penetration of an anti-pruritic, drying or anti-bacterial/fungal product. These can be minimally occlusive, but need frequent application and re-application. Examples would be a warm water or saline soak, colloidal oatmeal, chlorhexidine- or iodine solution, etc. Rinses / dips are water-soluble and most applications are left to dry on the animal (ie. do not wash these off). Rinses and dips can leave a residual layer of the active agent and are good for whole body coverage. Examples would be lime sulfur dips or older flea dip products.

Powders are a pulverized solid that is applied as a thin film and are frequently used to dry the skin. These can be quite messy and may irritate the respiratory mucosa of the patient or client! There are anti-parasitic, -bacterial, -fungal and -inflammatory/pruritic powders available in veterinary medicine.

Lotions are a solid in a liquid vehicle (ie. a suspension of a powder in water). These require shaking before application and often used as a “residual” and left on the skin. Examples in veterinary medicine include anti-fungal, -bacterial and -pruritic products along with anti-histamine and local anesthetic products.

Creams are oils in water (more water than oil) and function by absorbing water from the application site. These products are best used at localized sites of non-haired skin like facial or body folds, the chin, interdigital webbing and ears. Examples include anti-bacterial, -fungal and –inflammatory products.

Ointments typically consist of water in oil (and more oil than water). Ointments are the most occlusive vehicle and allow for better penetration that lotions or creams. These are best applied over localized lesions in non-haired skin. Examples include anti-bacterial, -fungal and –inflammatory products.

Gels are clear, colorless and greaseless products that can be rubbed into the skin completely. Gels may pass through the hair coat easier than a cream or ointment and are best for localized lesions. Examples include anti-bacterial, -parasitic and –seborrheic products.

Sprays in the form of aerosols and pumps can be easy to use…but may be frightening to the patient! If the spray consists of an alcohol-based vehicle, the product may sting or cause hypersalivation. Many products are a lotion or rinse used in a spray form. Examples include anti-bacterial, -fungal, -parasitic, -pruritic and –inflammatory products.

Wipes and mousse products offer similar ingredient/vehicle combinations, but in a potentially more owner compliant product. Wipes and mousse products can be applied at the facial folds, paws, tail folds and less haired body regions and can be better tolerated by the patient….which can translate into more regular owner administration and increased treatment success.

The agent is the active ingredient in the vehicle (often more than one vehicle for a particular agent) and the selections of the agent is often based on the desired effect, the type of disease and the condition of the skin. The most common disease application in dermatology is to treat both bacterial and yeast infections, manage seborrhea, kill parasites and control pruritus.
Anti-bacterial agents: benzoyl peroxide (keratolytic, degreasing, follicular flushing; synergistic with sulfur), ethyl lactate (10% breaks down to ethanol and lactic acid, lowers pH in the hair follicles and is rarely irritating; may not be as strong), chlorhexidine (2-4% anti-bacterial, anti-fungal; not inactivated by organic debris), triclosan (usually in combination with sulfur/salicylic acid), sulfur/salicylic acid, povidone iodine, propylene glycol, silver sulfadiazine.

Anti-fungal agents are used most often in combination with systemic therapy for malassezia dermatitis or dermatophytosis and most interfere with fungal ergosterol synthesis. Examples are ketoconazole, miconazole (more effective when combined with chlorhexidine), clotrimazole, enilconazole, thiabendazole, nystatin, acetic acid, selenium disulfide, lime sulfur.

Anti-seborrheic agents are either keratolytic (decrease keratinocyte adhesion; softens and removes the scale) or keratoplastic (helps normalize epidermal cell turnover). Most frequently a mixture of keratolytic and keratoplastic agents are used. Examples include sulfur, which is mildly keratolytic and keratoplastic; can be anti-bacterial, -fungal, -parasitic, -pruritic, but STINKY! Salicylic acid is synergistic with sulfur and predominantly keratolytic (mildly keratoplastic) and anti-bacterial in spectrum. Selenium disulfide is keratolytic and both anti-fungal and anti-bacterial with potent degreasing action; do not use on cats. Tar shampoo contain hydrocarbons which are keratoplastic and keratolytic, mildly degreasing. but can be irritating and stinky. Do not use hydrocarbon-containing products on cats as they are very sensitive!

Anti-pruritic and anti-inflammatory agents are not usually successful as a sole agent (in my caseload!), but they may help reduce the pruritic load of a patient, allow lower dosages of systemic medications, can protect the skin and anesthetize peripheral nerves.

Examples include corticosteroids, anti-histamines, local anesthetics, tar, sulfur, oatmeal, etc.

There are numerous other miscellaneous topical products, including astringents like tannins, witch hazel, aluminum acetate and acetic acid. These precipitate proteins to dry the skin and decrease exudate, but do not penetrate deeply; these may irritate the skin. Emollients can soften, lubricate and soothe the skin, decreasing trans-epidermal water loss. These are often oils or lanolin-containing products that are most effective if applied after saturation of the skin with water. Humectants are hygroscopic moisturizers that are incorporated into the stratum corneum to increase water content; examples include propylene glycol, glycerin, colloidal oatmeal, urea and lactic acid. Detergents have an unpleasant taste or may numb an area with local anesthetics like lidocaine, benzocaine, capsaicin, etc. Sunscreens are either chemical (absorb UV rays and have residual activity after application) or physical (reflect and scatter light; zinc oxide, titanium dioxide).

“Newer” products aimed at restoring the skin barrier defects of our atopic patients include Dermoscent Essential 6 Spot-On and Allerderm Spot-On. These products are made up of proprietary mixtures of essential fatty acids and ceramides and act to replenish the hydrolipid film of the skin, improving skin barrier function and promoting a healthy dermal ecosystem; they also aim to decrease trans-epidermal water loss to increase moisture and protection.

Audience participation for case examples
Match the patient presentation to the most appropriate topical product(s).

References
4. Individual websites and product literature for individual medicated products.
Hyperadrenocorticism (HAC) is a common endocrinopathy in dogs traditionally characterized by hypercortisolemia. There are three classic forms of this disease:

- **ACTH dependent** (previously called pituitary dependent) – In this form of disease, the pituitary gland secretes excess adrenocorticotropic hormone (ACTH) that subsequently stimulates the adrenal cortices to produce and secrete excess cortisol. Most commonly, patients have a functional adenoma arising from the pars distalis. Most are microadenomas (<1.0 cm diameter; 50% < 3.0 mm), and only a small percentage are malignant. Macroadenomas (>1.0 cm diameter) are found in 10-20% of affected patients. Approximately 80-85% of HAC patients have ACTH dependent disease.

- **ACTH independent** (previously called primary adrenocortical tumor) – In this form of disease, one adrenal cortex autonomously secretes excess cortisol. The hypothalamus and pituitary gland are affected via a negative feedback loop. Thus, the hypothalamus and pituitary gland don’t secrete as much corticotropin-releasing hormone and ACTH, respectively. Subsequently, the contralateral adrenal gland atrophies. Approximately 15-20% of HAC patients have ACTH independent disease. Approximately two thirds of adrenocortical tumors are malignant while one third are benign.

- **Iatrogenic** – In this form of disease, patients receive systemic corticosteroids that suppress CRH and ACTH secretion from the hypothalamus and pituitary gland, respectively. The adrenal cortices are not stimulated, and thus, the adrenal glands atrophy bilaterally.

Occult (previously called atypical) hyperadrenocorticism is a controversial manifestation of this disease. Blood cortisol levels remain normal, but androgen levels (i.e.: estradiol, progesterone, 17-OH progesterone) are elevated. Some evidence exists indicating androgen derangements can mimic clinical signs of HAC.

**Signalment & clinical signs**

The classic HAC patient is middle aged or geriatric. Approximately 75% of patients with ACTH dependent disease are older than 9 years of age while 90% of those with ACTH independent disease are older than 9 years of age. Males and females are equally represented. Breeds commonly affected by HAC are Poodles, Dachshunds, terrier breeds, German shepherds, Labrador retrievers, and Boxers. Clinical signs are usually chronic and progressively, including the “4 Ps”: polyuria, polydipsia, polyphagia, and polypnea. Other common manifestations include:

- Pendulous abdomen
- Bilaterally symmetrical non-pruritic truncal alopecia
- Hepatomegaly
- Muscle weakness and atrophy
- Comedones
- Thin, inelastic skin
- Skin hyperpigmentation
- Testicular atrophy
- Calcinosis cutis
- Poor wound healing
- Poor hair regrowth
- Recurrent infections
- Neurologic deficits
- Myotonia

Clinical signs not associated with HAC include hyporexia/anorexia, vomiting, diarrhea, coughing, sneezing, icterus, pruritus, pain, lameness and bleeding.

**Diagnostic testing**

Diagnosis of HAC requires more than compatible clinical signs. Common changes found on CBC/CHEM/UA include:

- CBC: mature neutrophilia without bandemia, lymphopenia, eosinopenia
- CHEM: elevated ALP, elevated ALT, elevated GGT, hypercholesterolemia, hypertriglyceridemia, hyperglycemia, hypophosphatemia, decreased BUN
- UA: isosthenuria, proteinuria, +/- evidence of urinary tract infection (UTI)
In patients with compatible clinical signs and biochemical changes, screening for HAC is warranted. Definitively diagnosing this disease can be challenging. For this reason, proactive communication with pet owners is essential. Veterinarians must be stellar communicators, explaining to families there is no singular perfect test to screen for HAC. The screening process is, indeed, just that—a process. Traditionally, there are three screening tests for this endocrinopathy.

- Urine cortisol:creatinine ratio (UC:Cr) – urine sample must be collected at home and at least 72 hours after a veterinary visit; poor specificity & high sensitivity
- Low-dose dexamethasone suppression test (LDDST) – high sensitivity & poor specificity
- ACTH stimulation test (ACTH stim) – more specific than LDDST; less sensitive than LDDST; cannot differentiate; test of choice for iatrogenic HAC

<table>
<thead>
<tr>
<th>TEST</th>
<th>SENSITIVITY</th>
<th>SPECIFICITY</th>
</tr>
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<tbody>
<tr>
<td>UC:CR</td>
<td>Excellent 75-100%</td>
<td>Poor 24-77%</td>
</tr>
<tr>
<td>LDDST</td>
<td>Excellent 85-100%</td>
<td>Poor 44-73%</td>
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<tr>
<td>ACTH Stimulation Test</td>
<td>Less than ideal 80-95%</td>
<td>Good 86-91%</td>
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One should note screening for HAC should not be performed in sick patients, as more than 75% of patients with non-adrenal illness will have false-positive results. Exogenous steroids affect the hypothalamic-pituitary-adrenal axis, inducing adrenocortical atrophy and reducing cortisol levels. A 4-week washout period is needed to eliminate this effect. Additionally, exogenous corticosteroids like prednisone/prednisolone and hydrocortisone cause false positive results. Dexamethasone reportedly does not interfere with accurate test results.

Once a screening test has confirmed a diagnosis of HAC, localizing the disease is indicated. Tests that may be used to achieve this goal are:

- Abdominal sonography
- Endogenous ACTH level
- High-dose dexamethasone suppression test
- +/- LDDST
- Advanced imagine (MRI, CT scan)

**Treatment**

Treatment for HAC is not warranted without clinical signs. “Trial” therapy is inappropriate and not recommended. Medical treatment options currently include:

- VETORYL® - FDA-approved therapy; inhibits 3-beta hydroxysteroid dehydrogenase; no separate induction & maintenance periods; used for ACTH dependent & independent diseases; q24 hr vs. q12 hr dosing protocols
- Mitotane/Lysodren® - controlled adrenocortical necrosis; predominantly affects zona fasciculate & zona reticularis and spares zona glomerulosa; induction & maintenance phases

Surgery (adrenalectomy, hypophysectomy), stereotactic surgery/radiation therapy may also be appropriate therapies depending on the form HAC with which a patient is living.

- ACTH independent: Primary adrenocortical tumors are best treated surgically. Surgery is technically challenging with a reportedly poorer prognosis when masses are greater than 5 cm, if there is vascular invasion, if there is evidence of venous thrombosis, and if metastatic disease is present. In patients undergoing adrenalectomy for a primary adrenocortical tumor, ~15% develop intraoperative complications and 50% develop post-operative complications. The prognosis is excellent for completely excised functional adenomas.
- Hypophysectomy or radiation therapy should be considered for patients with ACTH dependent disease with neurologic signs or large macroadenomas.
Radiation therapy also offers the ability to address the pituitary mass directly. A 2007 study evaluated the use of radiation therapy in 19 dogs with pituitary macroadenomas. Mean survival time was 1,405 days with 1-, 2-, and 3-year estimated survival times of 93, 87, and 55%, respectively.

Postoperative hypophysectomy patients may experience transient or permanent diabetes insipidus-like syndrome, hypothyroidism, keratoconjunctivitis sicca, and glucocorticoid deficiency.

Less commonly employed therapies for ACTH dependent disease with neurologic signs are:

- **Cabergoline** - Cabergoline is a dopamine (D2) receptor agonist, and may help by reducing ACTH production and secretion from pituitary adenomas that express the D2 dopaminergic receptor. A 2008 study indicated cabergoline is useful in 42.5% of dogs with ACTH dependent disease.

- **Retinoic acid** – A 2006 study evaluated the use of retinoic acid in rodents and dogs with ACTH dependent HAC; results indicated a reduction in ACTH in these cases.

- **Pasireotide** – This drug is a multi-ligand somatostatin analog that binds to somatostatin receptors in pituitary adenomas to trigger anti-secretion and anti-proliferation. A 2011 study indicated good control of hypercortisolemia in a small group of dogs with ACTH dependent disease HAC.

### Monitoring

Patients receiving VETORYL® and mitotane require serial monitoring. Patients receiving VETORYL® should have an ACTH stimulation test and biochemical profile (including electrolytes) evaluated 2 weeks and 6 weeks after initiating therapy, as well as quarterly thereafter. Testing should also be performed 2 weeks after any dosage adjustment is made. Testing must occur 4-6-hours post-pill. The therapeutic range for post-ACTH stimulation test cortisol while receiving VETORYL is 1.45-9.1 ug/dL. Use of baseline cortisol as a monitoring tool for patients receiving trilostane is not very accurate. Baseline cortisol level 1.3-2.9 ug/dL predicted acceptable control in 88% of patients. Urine cortisol:creatinine ratio should not be used for monitoring therapy. Pet owners must be fully aware of the schedule for and cost of the requisite monitoring of HAC patients. VETORYL® dosage adjustments are made predominantly on a patient’s clinical signs. For this reason, families should keep a detailed journal of their pet’s clinical signs.

Similarly, mitotane/Lysodren®: Patients received mitotane typically require serial ACTH stimulation tests and electrolyte panels during the induction phase. A well-controlled patient receiving maintenance mitotane therapy should be monitored quarterly for at least one year, and then every six months thereafter.

### References

Available upon request
Hyperadrenocorticism: Treatment and Monitoring
Christopher Byers, DVM, DACVECC, DACVIM
Midwest Veterinary Specialty Hospital
Omaha, NE

Hyperadrenocorticism (HAC) is a common endocrinopathy in dogs traditionally characterized by hypercortisolemia. There are three classic forms of this disease:

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Occult (previously called atypical) hyperadrenocorticism is a controversial manifestation of this disease. Blood cortisol levels remain normal, but androgen levels (i.e.: estradiol, progesterone, 17-OH progesterone) are elevated. Some evidence exists indicating androgen derangements can mimic clinical signs of HAC.

Signalment & clinical signs
The classic HAC patient is middle aged or geriatric. Approximately 75% of patients with ACTH dependent disease are older than 9 years of age while 90% of those with ACTH independent disease are older than 9 years of age. Males and females are equally represented. Breeds commonly affected by HAC are Poodles, Dachshunds, terrier breeds, German shepherds, Labrador retrievers, and Boxers. Clinical signs are usually chronic and progressively, including the “4 Ps”: polyuria, polydipsia, polyphagia, and polypnea. Other common manifestations include:

- Pendulous abdomen
- Bilaterally symmetrical non-pruritic truncal alopecia
- Hepatomegaly
- Muscle weakness and atrophy
- Comedones
- Thin, inelastic skin
- Skin hyperpigmentation
- Testicular atrophy
- Calcinosis cutis
- Poor wound healing
- Poor hair regrowth
- Recurrent infections
- Neurologic deficits
- Myotonia

Clinical signs not associated with HAC include hyporexia/anorexia, vomiting, diarrhea, coughing, sneezing, icterus, pruritus, pain, lameness and bleeding.

Diagnostic testing
Diagnosis of HAC requires more than compatible clinical signs. Common changes found on CBC/CHEM/UA include:

- CBC: mature neutrophilia without bandemia, lymphopenia, eosinopenia
- CHEM: elevated ALP, elevated ALT, elevated GGT, hypercholesterolemia, hypertriglyceridemia, hyperglycemia, hypophosphatemia, decreased BUN
- UA: isosthenuria, proteinuria, +/- evidence of urinary tract infection (UTI)
In patients with compatible clinical signs and biochemical changes, screening for HAC is warranted. Definitively diagnosing this disease can be challenging. For this reason, proactive communication with pet owners is essential. Veterinarians must be stellar communicators, explaining to families there is no singular perfect test to screen for HAC. The screening process is, indeed, just that – a process. Traditionally, there are three screening tests for this endocrinopathy.

- Urine cortisol:creatinine ratio (UC:Cr) – urine sample must be collected at home and at least 72 hours after a veterinary visit; poor specificity & high sensitivity
- Low-dose dexamethasone suppression test (LDDST) – high sensitivity & poor specificity
- ACTH stimulation test (ACTH stim) – more specific than LDDST; less sensitive than LDDST; cannot differentiate; test of choice for iatrogenic HAC

<table>
<thead>
<tr>
<th>TEST</th>
<th>SENSITIVITY</th>
<th>SPECIFICITY</th>
</tr>
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<tbody>
<tr>
<td>UC:CR</td>
<td>Excellent 75-100%</td>
<td>Poor 24-77%</td>
</tr>
<tr>
<td>LDDST</td>
<td>Excellent 85-100%</td>
<td>Poor 44-73%</td>
</tr>
<tr>
<td>ACTH Stimulation Test</td>
<td>Less than ideal 80-95%</td>
<td>Good 86-91%</td>
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One should note screening for HAC should not be performed in sick patients, as more than 75% of patients with non-adrenal illness will have false-positive results. Exogenous steroids affect the hypothalamic-pituitary-adrenal axis, inducing adrenocortical atrophy and reducing cortisol levels. A 4-week washout period is needed to eliminate this affect. Additionally, exogenous corticosteroids like prednisone/prednisolone and hydrocortisone cause false positive results. Dexamethasone reportedly does not interfere with accurate test results.

Once a screening test has confirmed a diagnosis of HAC, localizing the disease is indicated. Tests that may be used to achieve this goal are:

- Abdominal sonography
- Endogenous ACTH level
- High-dose dexamethasone suppression test
- +/- LDDST
- Advanced imagine (MRI, CT scan)

Treatment

Treatment for HAC is not warranted without clinical signs. “Trial” therapy is inappropriate and not recommended. Medical treatment options currently include:

- VETORYL® - FDA-approved therapy; inhibits 3-beta hydroxysteroid dehydrogenase; no separate induction & maintenance periods; used for ACTH dependent & independent diseases; q24 hr vs. q12 hr dosing protocols
- Mitotane/Lysodren® - controlled adrenocortical necrosis; predominantly affects zona fasciculate & zona reticularis and spares zona glomerulosa; induction & maintenance phases

Surgery (adrenalectomy, hypophysectomy), stereotactic surgery/radiation therapy may also be appropriate therapies depending on the form HAC with which a patient is living.

- ACTH independent: Primary adrenocortical tumors are best treated surgically. Surgery is technically challenging with a reportedly poorer prognosis when masses are greater than 5 cm, if there is vascular invasion, if there is evidence of venous thrombosis, and if metastatic disease is present. In patients undergoing adrenalectomy for a primary adrenocortical tumor, ~15% develop intraoperative complications and 50% develop post-operative complications. The prognosis is excellent for completely excised functional adenomas.
- Hypophysectomy or radiation therapy should be considered for patients with ACTH dependent disease with neurologic signs or large macroadenomas.

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Radiation therapy also offers the ability to address the pituitary mass directly. A 2007 study evaluated the use of radiation therapy in 19 dogs with pituitary macroadenomas. Mean survival time was 1,405 days with 1-, 2-, and 3-year estimated survival times of 93, 87, and 55%, respectively.

Postoperative hypophysectomy patients may experience transient or permanent diabetes insipidus-like syndrome, hypothyroidism, keratoconjunctivitis sicca, and glucocorticoid deficiency.

Less commonly employed therapies for ACTH dependent disease with neurologic signs are:

- **Cabergoline** - Cabergoline is a dopamine (D2) receptor agonist, and may help by reducing ACTH production and secretion from pituitary adenomas that express the D2 dopaminergic receptor. A 2008 study indicated cabergoline is useful in 42.5% of dogs with ACTH dependent disease.

- **Retinoic acid** – A 2006 study evaluated the use of retinoic acid in rodents and dogs with ACTH dependent HAC; results indicated a reduction in ACTH in these cases.

- **Pasireotide** – This drug is a multi-ligand somatostatin analog that binds to somatostatin receptors in pituitary adenomas to trigger anti-secretion and anti-proliferation. A 2011 study indicated good control of hypercortisolemia in a small group of dogs with ACTH dependent disease HAC.

**Monitoring**

Patients receiving VETORYL® and mitotane require serial monitoring. Patients receiving VETORYL® should have an ACTH stimulation test and biochemical profile (including electrolytes) evaluated 2 weeks and 6 weeks after initiating therapy, as well as quarterly thereafter. Testing should also be performed 2 weeks after any dosage adjustment is made. Testing must occur 4-6-hours post-pill. The therapeutic range for post-ACTH stimulation test cortisol while receiving VETORYL is 1.45-9.1 ug/dL. Use of baseline cortisol as a monitoring tool for patients receiving trilostane is not very accurate. Baseline cortisol level 1.3-2.9 ug/dL predicted acceptable control in 88% of patients. Urine cortisol:creatinine ratio should not be used for monitoring therapy. Pet owners must be fully aware of the schedule for and cost of the requisite monitoring of HAC patients. VETORYL® dosage adjustments are made predominantly on a patient’s clinical signs. For this reason, families should keep a detailed journal of their pet’s clinical signs.

Similarly, mitotane/Lysodren®: Patients received mitotane typically require serial ACTH stimulation tests and electrolyte panels during the induction phase. A well-controlled patient receiving maintenance mitotane therapy should be monitored quarterly for at least one year, and then every six months thereafter.

**References**

Available upon request
Adrenal physiology
The adrenal gland can be thought of as a peanut M&M, with the peanut representing the medulla and the chocolate coating representing the cortex. The adrenal cortex can be divided into three layers with distinct functions:

- **Zona glomerulosa** (outermost layer) – responsible for mineralocorticoid (i.e., aldosterone) production & secretion
- **Zona fasciculata** (middle layer) – responsible for glucocorticoid (i.e., cortisol) production & secretion
- **Zona reticularis** (innermost layer) – responsible for androgen production & secretion

Cortisol secretion is regulated by the hypothalamic-pituitary-adrenal axis. Various stresses induce secretion of corticotropin-releasing hormone (CRH) from the hypothalamus. This hormone subsequently stimulates secretion of adrenocorticotropic hormone (ACTH) from the pars distalis of the anterior pituitary gland. Then, ACTH stimulates the adrenal cortices to secrete cortisol. Cortisol influences the hypothalamus and anterior pituitary gland via negative feedback inhibition.

Aldosterone plays a central role in the regulation of plasma sodium and extracellular potassium concentrations. Synthesis of aldosterone is stimulated by several factors, particularly increased blood ACTH and potassium levels. Physiologic doses of ACTH do not play a significant role in the regulation of aldosterone secretion. Aldosterone binds to mineralocorticoid receptors within principal cells of the renal distal tubule and collecting ducts. Subsequently, Na+/K+ pumps in the basolateral membranes are upregulated and activated to pump three sodium ions out of the cell into the interstitial fluid and two potassium ions into the cell from the interstitial fluid. The resulting concentration gradient promotes the absorption of sodium (and water) into the blood and secretion of potassium into the lumens of the collecting ducts.

Pathophysiology
Primary hyperadrenocorticism is the result of immune-mediated destruction of more than 90% of the adrenal cortex. Rarely, neoplasia, infectious diseases, or inflammatory infiltration affecting both adrenal cortices can result in clinical illness. Secondary hyperadrenocorticism is the result of ACTH deficiency, and results in cortisol deficiency. Iatrogenic administration of glucocorticoids is the most common cause of secondary hyperadrenocorticism, but pituitary pathology is possible. Pituitary gland disease that results in ACTH deficiency should not result in aldosterone deficiency. Tertiary hyperadrenocorticism arises from disease in the hypothalamus, and is exceedingly rare. Atypical hyperadrenocorticism is a unique manifestation characterized by glucocorticoid deficiency with normal mineralocorticoid secretion and function. The etiology of this form is not known, and major theories include:

- Immune-mediated destruction of adrenal cortices that spares the zona glomerulosa
- Patients can compensate for mineralocorticoid deficiency via unknown mechanism(s)
- Atypical hyperadrenocorticism is an early manifestation of the typical disease

Clinical presentation
There is no pathognomonic presentation for patients living with hyperadrenocorticism. For this reason, this disease is often referred to as the Great Pretender. Patients may present acutely in various stages of hypovolemic shock. Others have chronic, waxing & waning, vague gastrointestinal signs, including vomiting, diarrhea, anorexia/hyporexia, melena, etc.

For those patients presented in shock, clinical signs vary depending on the stage of shock:

- **Compensatory** (CO increases due to catecholamine release) – normal vital signs to slight tachycardia, injected mucus membranes, rapid CRT, normal BP, normal mentation
- **Early Decompensatory** (blood preferentially distributed to heart/brain) – tachycardia (dogs > cats), pale mucous membranes, prolonged CRT, depression, hypothermia, hypotension
- **Late Decompensatory** (auto-regulatory escape) – bradycardia (dogs > cats), severe hypotension, absent CRT, weak/absent peripheral pulses, hypothermia, oliguria, obtundation

Rarely, patients may have a history of regurgitation due to diffuse megaesophagus. Generalized seizure activity has been documented secondary to hypoglycemia. Polyuria / polydipsia has been reported in some patients with atypical hyperadrenocorticism, but the mechanism has not yet been determined.
Diagnosis

Initial screening
A thorough diagnostic investigation is of paramount importance. A minimum database often yields clues that raise suspicion for hypoadrenocorticism. For example:

- Complete Blood Count (CBC): normal neutrophil count or neutropenia, normal lymphocyte count or lymphocytosis, normal eosinophil count or eosinophilia
- Serum Biochemical Profile (CHEM): hypoaalbuminemia, azotemia, elevated hepatocellular & cholestatic enzymes, hypercholesterolemia, hypoponatremia, hyperkalemia, total hypercalcemia, hypochloremia, hypoglycemia
- Venous Blood Gas (VBG): metabolic acidosis
- Urinalysis (UA): isosthenuria or minimally concentrated urine
- Electrocardiography: bradycardia, tented T waves, absent p waves, increased P-R interval
- Thoracic Radiography (CXR): microcardia, narrow caudal vena cava, megaesophagus
- Abdominal sonography (AUS): bilaterally small adrenal glands

The use of basal cortisol levels has been advocated as a clinically useful screening test for hypoadrenocorticism due to its reportedly high negative predictive value. In 2007, Lennon EM et al studied 110 dogs with non-adrenal illness and 13 dogs with hypoadrenocorticism. Basal cortisol concentrations <2 ug/dL had 100% sensitivity and 78.2% specificity. These results suggested patients with basal cortisol concentrations <2 ug/dL who are not receiving corticosteroids, mitotane, or ketoconazole were highly unlikely to have hypoadrenocorticism. In 2014, Bovens C et al evaluated 450 dogs with non-adrenal illness and 14 dogs with naturally occurring hypoadrenocorticism. A basal cortisol level ≤2 ug/dL had a 100% and 63.3% sensitivity and specificity, respectively; these results corroborated the results of Lennon EM et al. Most recently in 2016, Gold AJ et al evaluated 532 dogs (163 with hypoadrenocorticism, 351 with non-adrenal illness, 8 with equivocal results). A basal cortisol level of 2 ug/dL results in a sensitivity of 99.4%, confirming this value was useful for excluding a diagnosis of hypoadrenocorticism. The author finds measuring basal cortisol levels helpful for screening patients with normal sodium and potassium concentrations but who are suspected of living with the atypical form of the disease. Rarely patients with hypoadrenocorticism may have basal cortisol concentrations between 2-3 ug/dL.

Patients with basal cortisol levels <2 ug/dL require an ACTH stimulation test to confirm a diagnosis of hypoadrenocorticism.

Definitive testing
An ACTH stimulation test is the gold standard test for diagnosing hypoadrenocorticism. To perform the test:

- Collect a baseline cortisol sample
- Administer synthetic ACTH (cosyntropin / Cortrosyn®) @ 5 ug/kg IV
- Collect a 1-hour post-cosyntropin cortisol sample

Pre- and post-ACTH stimulation cortisol levels <2 ug/dL are consistent with hypoadrenocorticism. Due to the relatively high cost and/or lack of availability of cosyntropin, some investigators have evaluated other means of accurately diagnosing hypoadrenocorticism:

- Lathan P et al studied endogenous ACTH and cortisol concentrations before and after ACTH stimulation in 8 healthy dogs, 19 dogs with non-adrenal illness, and 15 dogs with hypoadrenocorticism. Baseline cortisol and ACTH concentrations were significantly lower and higher, respectively, in dogs with hypoadrenocorticism compared to the other groups (although overlap between all groups was documented). Cortisol-to-ACTH ratios were significantly lower in those with hypoadrenocorticism (median 0.000714) compared to the other 2 groups (healthy median: 2.27; non-adrenal illness median: 2.84). There was no overlap of those with hypoadrenocorticism with the other 2 groups.
- Zeugswetter FK et al retrospectively evaluated 145 dogs with clinical signs that raised suspicion for spontaneous hypoadrenocorticism. An endogenous ACTH level >50 pmol/L has a 96% sensitivity and 100% specificity. A Na⁺:K⁺ ratio ≤ 22 had a 92% sensitivity and 91% specificity. Neutrophil to lymphocyte ratio performed poorly. However, 68% had Na⁺:K⁺ ratios and neutrophil to lymphocyte ratio <2.3.
- Boretti FS et al evaluated 23 dogs with hypoadrenocorticism, 79 dogs with diseases mimicking hypoadrenocorticism, and 30 healthy dogs. Cortisol to ACTH ratios were significantly lower in dogs with hypoadrenocorticism compared to healthy dogs; however, there was a considerable overlap between those with hypoadrenocorticism and those with disease mimicking the condition.

Treatment
The interventions required for patients with hypoadrenocorticism is largely dependent on that patient’s presentation. Patients presented in shock – a so-called Addisonian crisis – require efficient stabilization. Therapies needed include intravenous fluids, calcium gluconate for cardioprotection, regular insulin and dextrose supplementation to address hypokalemia and hypoglycemia, respectively. Glucocorticoid supplementation is a key intervention in patient with both typical and atypical hypoadrenocorticism. Initial supraphysiologic supplementation with dexamethasone (0.05-0.07 mg/kg IV q24 hr) is appropriate until a patient is cardiovascularly stable.
stable and willing to eat/drink on its own without complications. At that time, patients should be transitioned to prednisone at supraphysiologic doses (0.3-0.45 mg/kg PO q24 hr). After several days of supraphysiologic supplementation, provision of physiologic doses (0.01-0.25 mg/kg PO q24 hr) is needed lifelong. The required physiologic dose should be adjusted based on a patient’s clinical signs, including corticosteroid-induced side effects. Owners should be advised to increase their pet’s glucocorticoid dose during stressful periods and/or unrelated illness.

Replacement of mineralocorticoid is indicated for patients with typical hypoadrenocorticism, that is pets with documented hyperkalemia and hyponatremia. Patients with atypical hypoadrenocorticism do not require mineralocorticoid replacement. Mineralocorticoids can be replaced with either oral desoxycorticosterone pivalate (DOCP / ZYCORTEL Suspension ®; 2.2 mg/kg SC; some clinicians use an extra-label initial dosing of 1.7 – 2.2 mg/kg) or fludrocortisone (Florinef®; 0.01 mg/kg PO q12 hr) administered via injection every 25-30 days. Most Addisonian patients are dehydrated upon presentation, and thus, proper absorption of subcutaneous injections may be adversely affected by dehydration. ZYCORTEL Suspension is labeled for subcutaneous injection. However, clinicians need to ensure proper patient hydration prior to subcutaneous injections; alternatively, they may elect extra-label intramuscular administration to ensure proper absorption. Fludrocortisone has glucocorticoid activity. Approximately 50% of dogs receiving this drug do not require additional glucocorticoid supplementation. DOCP has no glucocorticoid activity. Thus, all patients receiving this drug require physiologic glucocorticoid supplementation. The author prefers to supplement mineralocorticoids with DOCP. Electrolytes should be assessed 12-14 days post-DOCP administration, and these results may be used to adjust dosing in the future.

Prognosis
Patients with hypoadrenocorticism who survive an initial crisis usually have an excellent prognosis. The author frequently tells pet parents during counseling that if he must diagnose patients a lifelong disease, he prefers to diagnose them hypoadrenocorticism. Owners must be educated about the requisite monitoring for patients with this disease. Furthermore, those with the atypical manifestation of this disease should be monitoring regularly for the development of electrolyte derangements necessitating mineralocorticoid supplementation.

References
Diseases of the Neck
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With ever-improving imaging technology, recognition and diagnosis of performance issues related to diseases of the equine neck continue to increase. Wobblers Syndrome (also known as cervical vertebral stenotic myelopathy (CVSM), cervical vertebral compressive myelopathy, cervical vertebral instability, and/or cervical vertebral malformation) and cervical osteoarthritis are the most common diseases of the neck and can occur simultaneously or independently. CVSM is often divided into two types which encompass two rather different diseases. Young horse CVSM is a developmental abnormality seen in a weanling age horse with stenosis of the vertebral canal (usually mid-cervical) that is worse on flexion. The second type of CVSM involves bony and soft-tissue impingement on the spinal cord due to remodeling of the articular facets that is present statically or worsened by hyperextension. This syndrome occurs in middle age and older horses, and generally involves C5-C7. Middle aged and older horses can also have degenerative joint disease of the articular facets that causes lameness and performance issues without overt ataxia and spinal cord compression but with possible nerve root compression.

Clinical signs of CVSM are classically seen in horses 6-18 months old, however those with disease due to remodeling or DJD of the articular facets may be seen at any point in life. Ataxia and paresis typically develop and progressively worsen over several months although they may wax and wane or develop suddenly. The pelvic limbs are generally more affected than the thoracic limbs due to the more superficial location of the proprioceptive tracts from the pelvic limbs within the spinal cord. An exception would be that lesions at C6-T1 may result in more severe signs in the thoracic limbs. Typically signs are symmetrical with dynamic stenosis, but may be asymmetrical with static stenosis/asymmetric facet enlargement. Clinical signs may include stumbling/toe scuffing, stiff incoordinated walk, truncal sway, decreased resistance to walking tail pull, hypometric forelimb gait when head elevated (“tin soldier” gait), circumduction of outside pelvic limbs when circling, and difficulty backing. Conscious proprioceptive deficits may also be present. Repeated pulling of front shoes or shearing of front heels may be a sign of CVSM due to overreaching stride length. Neck pain, muscle atrophy, and negative slap test can also be seen. Horses with cervical facet arthritis but no spinal cord compression may show neck pain, abnormal head and neck posture, and reduced neck flexion. With nerve root compression, thoracic limb lameness or “root signature” (forelimb raised and held off the ground) may be seen. Neck problems may also be manifest as behavior changes under saddle or difficulty with maintaining a frame, bending, or lateral work.

Plain lateral radiographs are the first diagnostic to pursue and may be adequate to document stenosis of the vertebral canal or cervical osteoarthritis. With developmental CVSM, osteochondrosis of the articular facets and metaphyseal growth plates result in the classic radiographic findings of caudal flare of the vertebral body, relative elongation of the dorsal laminae, and enlargement of the facet joints in addition to narrowing of the vertebral canal. Other findings may include mal-articulation of vertebrae. Intervertebral sagittal ratios can be performed on plain lateral films to identify narrowing of the vertebral canal (Figure 1). Intravertebral sagittal ratios are calculated as the [minimal sagittal diameter of the vertebral canal] ÷ [maximal sagittal diameter of the cranial half of that vertebral body]. The original description found 89% sensitivity and specificity using values of <0.52 from C4-C6 and <0.54 at C7 to predict compression. In practice, most clinicians use cutoffs of 0.5 for C3-C6 and 0.52 for C7. Intervertebral sagittal ratios look for fixed flexion or extension angles that might result in spinal cord compression. The numerator is the shorter of either the shortest distance from the caudal aspect of the dorsal lamina to the craniodorsal body of the next vertebra or the shortest distance between the caudodorsal vertebral body to the cranial point of the dorsal lamina of the next caudal vertebra. The denominator is the maximal sagittal diameter of the cranial half of the vertebral body of the caudal vertebra. Values have been published for each location (Hahn CN, Vet Radiol Ultrasound 2008;49:1-6). Sagittal diameter ratios of ≤0.485 at any inter- or intra-vertebral site from C2-C7 could correctly classify a horse has having CVSM. While cervical osteoarthritis is easy to recognize radiographically, it can be difficult to determine the clinical relevance as age-related remodeling is very common. Asymmetrically enlarged joints or those that extend ventrally below the bottom of the vertebral canal are generally considered clinically significant. When indicated, oblique radiographs can also be taken to better evaluate each articular facet for DJD or fractures. Ultrasound examination of the articular facets is also described primarily in terms of ultrasound-guided injection, but can be useful to look for DJD, fractures, or synovial proliferation.

Figure 1. Plain standing lateral radiograph of C4-C5 of a 6 month old TB weanling colt demonstrating flare of the caudal epiphysis and overriding dorsal lamina, all suggestive of CVSM. The intravertebral ratio is calculated as 2.3/4.9 = 0.4. The intervertebral ratio is 2.1/4.9 = 0.42. Both of these are also
suggestive of compression.

Myelography may be used to support the diagnosis of compression and establish the number and location of sites involved. This is especially important if surgical stabilization is going to be pursued as different sites may compress on myelography than those that were suspected on plain radiographs. Lateral views are taken in neutral, flexed, and extended positions and then several criteria can be evaluated, none of which are perfect. Complete loss of the dorsal and ventral dye columns is clear evidence of compression. Greater than 50% compression of the dorsal dye column in a flexed position compared to the dorsal dye column at the adjacent cranial or caudal body with complete attenuation of the ventral dye column has adequate sensitivity but poor specificity (i.e. too many false positives, especially in the caudal neck) (Figure 2). Greater accuracy can be obtained by measuring the dural (or sagittal) diameter at the site of interest (B) compared with the mid-body dural diameter of the cranial vertebrae (A) with ≥ 20% or greater reduction being compressive. Unfortunately, myelography primarily measures dorsoventral compression so transverse or oblique compression may be missed. Advanced imaging with computed tomography or magnetic resonance imaging are becoming possible at some hospitals, including standing CT development using different technologies to overcome the limitations of gantry size. Cervical vertebral canal endoscopy has also been described but is not widely performed. Ultimately, necropsy examination remains the gold standard.

Figure 2. Myelogram of C2-C5.

Treatment for CVSM depends on the age of animal at diagnosis, type of changes, severity of clinical signs, and expectations of the owner. Horses less than one year of age may be placed on a “paced growth” diet to either prevent CVSM or treat those with mild clinical signs. If still suckling, foals are immediately weaned and then fed a diet 65-75% of NRC requirements (usually grass hay with a ration balancer high in micronutrients and low in calories). Exercise restriction and no access to pasture are also enforced. Vitamin E and selenium supplementation should also occur. Surgical correction with ventral interbody vertebral fusion (“basket surgery”) can relieve dynamic stenosis and may improve static stenosis by allowing atrophy of the articular facets over time. The aim is to stop the repetitive trauma and associated inflammation in and around the spinal cord. On average, an improvement of 1 to 2 neurologic grades is expected with 12-62% of horses returning to athletic function. Recent unpublished numbers from a large referral hospital found that 75% of horses improved by 2 grades with 63% returning to athletic function, 15% being suitable for breeding, 10% being safe for turnout, and 12% failing to improve. Even 3-site stabilizations can be successful. Techniques involving locking compression plates or pedicle screw and rod construct have also been described. Obviously humane euthanasia is also a consideration in many cases based upon the severity of clinical signs, safety of the animal to itself and others, intended use of the animal and financial resources of the owner.

Ultrasound-guided articular facet injection with a combination of corticosteroid +/– antibiotics and hyaluronic acid may decrease inflammation and stabilize or prevent further bony proliferation. Joint injections are most beneficial in horses with zero or minimal ataxia (< grade 2). Systemic non-steroidal anti-inflammatories or corticosteroids may also improve clinical signs.

Diseases of the neck remain an important cause of ataxia and performance issues in horses. Improved diagnostic imaging may allow earlier and more accurate diagnosis which will result in increasingly more successful treatments in the future.
Equine Herpes Myeloencephalopathy
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Equine herpesvirus-1 is ubiquitous in most equine populations causing primarily respiratory disease in young horses. However, it can also cause abortion in pregnant mares, early neonatal death in foals, and myeloencephalopathy. While reports of equine herpesvirus-1 myeloencephalopathy (EHM) may appear to be on the rise, EHM fortunately remains a sporadic and relatively uncommon disease.

Infection with EHV-1 occurs by inhalation with virus then attaching to and replicating within the nasopharyngeal epithelium and associated lymphoreticular tissues. Virus is detected in the respiratory lymph nodes within 24-48 hours post-infection. Fever generally occurs 24 hours before nasal shedding of virus. Infection of peripheral blood mononuclear cells results in a viremia that allows EHV-1 to be delivered to other tissues. Infection of endothelial cells in the small vessels of the central nervous system or uterus results in vascular compromise and associated clinical signs. Cell-associated viremia can persist for at least 14 days. While viremia is common post EHV-1 infection, delivery to the CNS and development of EHM is rare with only 10% of horses developing neurological signs even during an EHM outbreak. It is unclear what determines which horses may develop EHM after EHV-1 infection. Virus, host, and environmental factors may all play a role. The D752 “neuropathogenic” strain of EHV-1 results in higher magnitude and duration of viremia which may result in increased ability to cause EHM. However, not all cases of EHM are associated with D752 as approximately 14% of EHV-1 isolates from horses with EHM do not have that “neuropathogenic” marker (N752). Older horses are generally more susceptible to EHM which may indicate a role for immunologic status in the pathogenesis of EHM, but that is not fully elucidated.

Clinical signs of neurologic disease are due to vasculitis, hemorrhage, thrombosis, and ischemic neuronal injury to the central nervous system. The spinal cord grey and white matter are most commonly affected with the brainstem being infrequently affected. EHM cases may be preceded by a recent history of fever, abortion or respiratory disease in the affected horse or herd mates, but this is not always the case. Neurological signs include sudden onset of ataxia, paresis, and urinary and fecal incontinence.

Diagnosis of EHM is primarily based on molecular detection of EHV-1. Hematologic abnormalities are inconsistent, and cerebrospinal fluid typically reveals xanthochromia but is non-specific. Quantitative polymerase chain reaction (qPCR) has become the test of choice due to its high sensitivity and specificity. Submission of both nasopharyngeal swab and uncoagulated blood in an EDTA tube is recommended to look for both nasal shedding and viremia. Viral load testing is beneficial to better assess the risk of exposure to other horses as well as response to therapy. qPCR has recently been used to document differences in EHV-1 viral loads in blood and nasal secretions between horses in the febrile and neurological stages of disease and between clinically affected and subclinically infected horses. qPCR also allows the determination of strain type (D752 vs N752), although the distinction between neuropathogenic and non-neuropathogenic does not guarantee the presence or absence of neurological signs. It is important to note that the random testing of normal horses for EHV-1 should be avoided as this may complicate quarantine decisions.

Treatment of horses with EHM has historically been supportive including nursing care, fluid and nutritional support, and bladder and rectal evaluation. Anti-inflammatories in the form of NSAIDs or steroids are frequently used but with little evidence that they alter outcome. In vitro work indicates that the anti-inflammatory drugs firocoxib, lidocaine, and dexamethasone decrease infection of endothelial cells by reducing contact between EHV-1 infected peripheral blood mononuclear cells and endothelial cells. While further in vivo work is necessary, these results suggest that earlier use of anti-inflammatory drugs in EHV-1 infections might decrease endothelial cell infection at the CNS-barrier and therefore prevent EHM. Recent work has also provided encouraging evidence for the use of heparin in cases of EHM. During an outbreak of EHV-1, metaphylactic heparin (25000IU SQ q12h for 3 days) was used in 31 febrile horses (the first 30 febrile horses in this outbreak were untreated). There was a lower EHM incidence in heparin-treated horses (1/31; 3.2%) than untreated horses (7/30; 23.3%); however randomized, prospective studies need to confirm this finding. Another drug under investigation is valacyclovir. Valacyclovir is an antiviral drug with in vitro activity against EHV-1 that has been used in outbreaks previously but with little clinical in vivo evidence. Maxwell, et al investigated the protective effects of valacyclovir (loading dose of 27 mg/kg PO q8h for the first 48 hours, then 18 mg/kg PO q12h) whether given prophylactically 1 day prior to EHV challenge (and continued for 1 or 2 weeks after viral inoculation) or post-challenge at onset of fever (treatment initiated at fever and continued for 1 or 2 weeks). Horses receiving prophylactic valacyclovir for 1 or 2 weeks and horses in the febrile treatment group treated for 2 weeks had significantly reduced amounts of viral DNA in nasal swabs for 2 weeks post inoculation. Drug treatment also reduced viremia in both groups. While clinical disease still occurred, the severity of fever and ataxia were decreased in both treatment groups versus controls. The protective effect was greatest when treatment was started prior to inoculation and continued for 2 weeks but still showed benefit even when given 2 days post-inoculation. This suggests that valacyclovir treatment may be indicated in outbreak situations with horses at various stages of infection. Administration of a zinc-containing supplement was found to be associated with decreased risk of EHM in one owner-reported epidemiologic study.
There is no vaccine licensed for the prevention of EHM. Vaccination has been cited by some as a potential risk factor for development of EHM, although evidence to support this opinion is far from conclusive. In contrast, field experience in North America strongly suggests that regular revaccination of pregnant mares and other horses on breeding farms reduces the risk of EHV-1 induced abortion and is well justified. Therefore, vaccination may be used to minimize EHV spread and viremia in a population of horses. Vaccination in the face of an outbreak should be discussed carefully as it is a risk-based decision. Biosecurity cannot be overemphasized, however, as reducing introduction, dissemination, or recrudescence of latent EHV-1 infections may decrease viremia and likelihood of clinical disease. As a reportable disease, quarantines associated with EHM will be decided by the state veterinarian. However, horses returning from events where they may have been exposed to EHV-1 should ideally be isolated for 21 days and have temperatures taken twice daily to mitigate the risk at their home facility.

Good communication between owners, barn managers, veterinarians, and state authorities cannot be overstated in cases of EHM. Prompt recognition, diagnosis, quarantine and treatment will hopefully result in the best case scenario.
Horses can be affected by a variety of neurologic diseases with potentially career or life-ending implications. The importance of a complete neurologic examination cannot be understated. The goals of the neurologic exam are to first determine if neurologic deficits are indeed present and if so, to use them to localize the lesion(s). Neuroanatomical localization is key to creating and ranking differential diagnoses, which then guide the diagnostic and treatment plan. Thus, a systematic and repeatable neurologic examination is an invaluable tool for all equine practitioners.

A complete examination begins with a thorough history including signalment, vaccination history, travel history, presence of signs in more than one horse or other animal on the farm, diet, and medication history. The duration and progression of signs as well as response to any treatments should be noted. A complete physical examination should also be performed. Presence of fever is especially important, but evaluation of the cardiovascular, gastrointestinal, ophthalmic, and musculoskeletal systems may also reveal significant abnormalities.

Initial observation of the horse should include assessment of both mentation and behavior. Mentation describes the patient’s level of awareness or consciousness and is affected by diseases involving the ascending reticular activating system in the brainstem or diffuse forebrain disease. Abnormalities of behavior are termed dementia and are generally signs of forebrain disease. Moving caudally, assessment of the cranial nerves provides further assessment of the brain and brainstem.

<table>
<thead>
<tr>
<th>Name</th>
<th>Function</th>
<th>Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Offactory</td>
<td>Smell</td>
<td>Difficult to assess and dysfunction uncommon</td>
</tr>
<tr>
<td>II Optic</td>
<td>Vision</td>
<td>1. Menace (also CN V)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. PLRs (also CN III)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Dazzle response</td>
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<tr>
<td></td>
<td></td>
<td>4. Obstacle course</td>
</tr>
<tr>
<td>III Oculomotor</td>
<td>Extraocular muscles Pupil diameter</td>
<td>1. Eye position (also CN IV, VI)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a. Lesion=ventrolat strabismus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Pupil size and symmetry</td>
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<tr>
<td></td>
<td></td>
<td>3. PLRs (also CN II)</td>
</tr>
<tr>
<td>IV Trochlear</td>
<td>Extraocular muscle (dorsal oblique)</td>
<td>1. Eye position (also CN III, VI)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a. Lesion=dorsomed strabismus</td>
</tr>
<tr>
<td>V Trigeminal</td>
<td>Facial sensation</td>
<td>1. Palpebral reflex (also CN VII)</td>
</tr>
<tr>
<td></td>
<td>Motor to muscles of mastication</td>
<td>2. Nasal septum, face, ear sensation</td>
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<tr>
<td></td>
<td></td>
<td>3. Corneal reflex</td>
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<td></td>
<td></td>
<td>4. Jaw tone</td>
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<td></td>
<td></td>
<td>5. Masseter, temporalis, pterygoid muscle mass</td>
</tr>
<tr>
<td>VI Abducens</td>
<td>Extraocular muscles (lateral rectus and retractor bulbi)</td>
<td>1. Eye position (also CN III, IV)</td>
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<tr>
<td></td>
<td></td>
<td>a. Lesion=medial strabismus</td>
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<tr>
<td></td>
<td></td>
<td>2. Globe retraction, corneal reflex</td>
</tr>
<tr>
<td>VII Facial</td>
<td>Motor to muscles of facial expression</td>
<td>1. Palpebral reflex (also CN V)</td>
</tr>
<tr>
<td></td>
<td>Parasympathetic to salivary and lacrimal glands</td>
<td>2. Lip/ear droop, ptosis, nasal deviation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Dry eye</td>
</tr>
<tr>
<td>VIII Vestibulocochlear</td>
<td>Posture and balance Hearing</td>
<td>1. Head tilt, leaning, circling, falling</td>
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<tr>
<td></td>
<td></td>
<td>2. Eye position</td>
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<td></td>
<td></td>
<td>3. Normal physiologic nystagmus</td>
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<td></td>
<td></td>
<td>4. Abnormal resting or positional nystagmus</td>
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<tr>
<td></td>
<td></td>
<td>5. Response to noise</td>
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<td></td>
<td></td>
<td>6. BAER</td>
</tr>
<tr>
<td>IX Glossopharyngeal</td>
<td>Sensory and motor to pharynx</td>
<td>1. Ability to swallow (also CN X)</td>
</tr>
<tr>
<td>X Vagus</td>
<td>Laryngeal and pharyngeal function</td>
<td>1. Ability to swallow (also CN IX)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Slap test (endoscopy)</td>
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<tr>
<td></td>
<td></td>
<td>3. Respiratory noise</td>
</tr>
<tr>
<td>XI Spinal accessory</td>
<td>Sensory and motor to cervical epaxial muscles</td>
<td>1. Skin sensation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Muscle atrophy of neck</td>
</tr>
<tr>
<td>XII Hypoglossal</td>
<td>Motor to tongue</td>
<td>1. Tongue symmetry</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Tongue strength</td>
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</tbody>
</table>
Continuing caudally, reflexes such as the cervicofacial, cutaneous trunci, spinal flexion, perineal sensation, anal tone, and tail tone can be used to assess the spinal cord. A retractable ballpoint pen is generally sufficient, although hemostats may be used. Careful assessment of any changes in muscle mass or limb posture can also be useful.

Finally, the moving examination is used to more finely localize any spinal cord lesions. The moving exam should be performed first walking in a straight line on level ground. Abnormalities such as toe dragging, inconsistent limb placement, inconsistent stride length, and limb interference should be noted. Neurologic horses generally have an irregularly irregular gait (as opposed to a regularly irregular gait which can be seen with lameness). Elevating the head may exacerbate subtle proprioceptive deficits. Normal horses should shorten their stride, those with UMN deficits will have a long, floating stride, and those with LMN deficits may be unable to raise their head or will have a severely shortened gait. Both standing and walking tail pull should be performed: standing to assess lower motor neuron strength and walking to assess upper motor neuron function. The horse should be circled in both directions. Horses with UMN lesions may show circumduction, interference, or fall. Those with LMN deficits may plant the limbs and pivot. When possible, the exam should also include walking the horse over obstacles such as a curb and up/down a hill. Horses with LMN deficits will have trouble picking their feet up high enough to step onto the curb or climb up a hill. Horses with UMN deficits may misplace their feet coming off a curb and have a long gait coming down a hill. Backing should be performed as a coordinated diagonal gait. Hopping can be used to assess forelimb strength. Blindfolding can be performed to remove visual input and unmask compensated vestibular disease, but should only be done in a safe location with soft footing.

<table>
<thead>
<tr>
<th>Neurologic Deficit</th>
<th>Localization</th>
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<tbody>
<tr>
<td>UMN in all 4 limbs</td>
<td>C1-C6</td>
</tr>
<tr>
<td>LMN in forelimbs and UMN in hind limbs</td>
<td>C6-T2</td>
</tr>
<tr>
<td>LMN in forelimbs with normal hind limbs</td>
<td>Ventral grey column C6-T2, nerve roots, peripheral nerves</td>
</tr>
<tr>
<td>UMN in hind limbs only</td>
<td>T3-L3</td>
</tr>
<tr>
<td>LMN in hind limbs only</td>
<td>L4-S2, nerve roots, peripheral nerves</td>
</tr>
<tr>
<td>LMN in all 4 limbs</td>
<td>Diffuse neuromuscular</td>
</tr>
<tr>
<td>Loss of anal/tail tone, urinary/fecal incontinence</td>
<td>Sacrocaudal spinal cord, nerve roots, peripheral nerves</td>
</tr>
</tbody>
</table>

With the overall goal to provide neuroanatomical localization, the first objective is to decide if any neurologic deficits were observed. If so, list all the abnormalities and where within the nervous system they could occur (cerebrum, brainstem, cerebellum, spinal cord, or peripheral nerve). If they can be described by one lesion, disease should be assumed to be focal. If there are multiple lesions, disease may be multifocal or diffuse. Any asymmetry left-to-right or front-to-back should also be noted. This information is all used to create a list of differentials and diagnostic plan.
Equine protozoal myeloencephalitis (EPM) is a neurologic disease of horses caused by one of two different protozoal organisms. The life cycle of *Sarcocystis neurona* is well described with the definitive host being the opossum and the intermediate hosts being raccoons, skunks, armadillos, sea otters, and cats. Within the horse, sporocysts become merozoites infecting the central nervous system, but the horse is an aberrant host and cannot complete the life cycle. The seroprevalence of *S. neurona* in the United States varies from 15-89% with the highest prevalence in areas with large opossum concentrations and the lowest prevalence in cold or arid climates. The complete life cycle including definitive and intermediate hosts of *Neospora hughesi* is unknown, but there is evidence that it can be passed transplacentally in horses. *N. hughesi* shows no regional differences in its seroprevalence which is approximately 30% throughout the US. It is important to remember for either cause of EPM that seroprevalence does not equal disease as not all horses that are exposed will develop clinical signs. In fact, the annual incidence of clinical cases of EPM in horses in the United States is less than 1%.

EPM can look like many other neurologic or musculoskeletal diseases. Clinical signs seen with EPM can be caused by both direct neuronal damage as well as secondary damage from inflammation. Signs can range from subtle and slowly progressive to acute and severe. They can be focal or multifocal and can involve any portion of the central neurologic system from brain to spinal cord, including white or grey matter. The constellation and severity of signs depends on the location of infection, number of organisms, and duration of disease. The classic signs of EPM include ataxia and muscle atrophy, either of which is often asymmetric. Forebrain and brainstem lesions can also be seen with changes in behavior, seizures, and cranial nerve deficits.

Definitive diagnosis of EPM is challenging, with necropsy remaining the gold standard. Physical exam, lameness exam, and neurologic exam should all be performed to confirm and localize neurologic deficits. Complete blood count and chemistry can be performed to look for any concurrent diseases. For specific diagnosis of EPM, several serologic tests are available, but it is important to remember that the presence of antibodies in the blood only confirms exposure not infection of the neurologic system. Horses without neurologic signs should NOT be tested. Specific antibody-based testing options include indirect fluorescent antibody (IFAT, UC Davis) and SnSAG2,4/3 ELISA (Equine Diagnostic Solutions, Lexington KY). The SnSAG 2,4/3 measures antibodies against 3 different surface antigens of *S. neurona*, while IFAT tests for whole organism targets and can cross-react with *Sarcocystis fayeri*. If using the IFAT on serum alone, it is important to remember that the predictive values provided by the laboratory are only valid in California where the seroprevalence is lower than most of the country. Cerebrospinal fluid (CSF) must be collected to most accurately make an ante-mortem diagnosis as well as rule out other diseases. CSF indices (color, clarity, white blood cell count, and protein concentration) are not consistently abnormal with EPM. The most accurate diagnosis will be made using a serum:CSF ratio - this ratio allows comparison between antibody produced in the CSF (thus indicating active infection) and antibody in the blood just indicating exposure. An SnSAG 2.4/3 serum:CSF ratio of <100 has the highest sensitivity and specificity of any available test. If using the IFAT, a serum:CSF ratio <64 is indicative of EPM. Both UC Davis and EDS offer testing for *N. hughesi* as well, and considering that 15% of EPM cases are due to *N. hughesi* veterinarians should consider testing for both protozoa.

There are 3 FDA approved treatments for EPM. Combination treatment with pyrimethamine and sulfadiazine (ReBalance®, PRN Pharmaceutical, 1 mg/kg and 20mg/kg respectively) synergistically inhibits folate metabolism. Because pyrimethamine also has slight inhibitory effects on mammalian dihydrofolate reductase, this drug combination can have potential side effects including bone marrow suppression (anemia, leukopenia, and thrombocytopenia), teratogenesis, and glossitis. Therefore, routine complete blood count monitoring is suggested at least monthly during treatment with these drugs. While anemia usually recovers within several weeks of discontinuing the drug, folate supplementation will not prevent or treat these side effects. Tetrahydrofolic acid must be used. While CSF steady state is rapidly reached within 4-6 hours, absorption is affected by dietary folate, so it is recommended that treatment not be given within 1-2 hours of a hay meal. Treatment with Pyr-SMZ usually lasts 90-270 days. The triazine anti-coccidials include ponazuril, diclazuril, and toltrazuril. These drugs target the merozoite stage of the organism, affecting the apicoplast organelle although their precise mode of action is unclear. Due to their selective action on a non-mammalian organelle, there is minimal toxicity seen with any of these drugs. Marquis® (ponazuril, Boehringer Ingelheim) was the first FDA-approved treatment for EPM. With the initial label dosing of 5 mg/kg SID, it required approximately 7 days to reach CSF steady state. With a one-time loading dose of 15 mg/kg, CSF steady state is now reached after the first dose. Label treatment should then continue for at least 28 days. Co-administration of either copper or DMSO has also been shown to increase bioavailability. The drug has not been tested in breeding animals or with concomitant therapies. Diclazuril is available as a top dress feed (Protazil®, Merck), Steady state is reached after 10 days, however the label states therapeutic levels are reached 6-8 hours after a single dose. The label recommends treatment for at least 28 days. Signs of toxicity are rare with ponazuril or diclazuril, but reported effects may include inappetance or soft feces. The efficacy studies for all 3 FDA approved drugs are reasonably similar with approximately 60% response rates. It is important to note these
studies were all done using the Western blot for diagnosis and on referral or university hospital populations. While there are many non-FDA approved products being sold to treat EPM, none have gone through safety or efficacy studies or been subjected to the scrutiny of published, peer-reviewed literature.

Adjunctive therapies include anti-inflammatories such as corticosteroids and non-steroidal anti-inflammatory. Short term use of corticosteroids may be of benefit for their anti-inflammatory effects, but long term use is discouraged due to their immunosuppressive effects. Vitamin E may be used for its anti-oxidant properties. Immunomodulators are an under-researched area in the field of EPM, however many clinicians attempt to promote cell mediated immunity. While levamisole may have such effects, it important to note that its metabolite aminorex will result in positive drug tests in racing or performance horses.

Prognosis for improvement is routinely quoted at 50-80%, with an estimated 20% making full recoveries. The likelihood of recovery is inversely related to the delay in instituting treatment. The most significant improvement is generally seen within the first four weeks. Improvement or recovery should be based on neurologic exam, not retesting, as 80% of horses will remain positive on CSF Western blot with treatment even if they appear clinically normal. Perhaps one of the most frustrating and puzzling aspects of EPM is the 10-20% of horses who “relapse” within 2 years of treatment. It is unclear whether this is due to reinfection or recrudescence, but treatment is generally considered less successful.

Prevention of EPM should be primarily through environmental control to minimize exposure of horses to opossum feces. Maintaining horse health through proper nutrition, deworming, and veterinary care for concurrent diseases (such as PPID) cannot be understated. If N. hughesi is confirmed in a mare, she should ideally be removed from the breeding herd as there is a risk of transplacental infection. Various chemoprophylactic or metaphylactic protocols have been investigated, but all are off-label. While development of resistance of the protozoa to drugs is unlikely due to the life cycle, the lack of development of protective immunity may leave horses more susceptible to future infection if chemoprophylaxis is ever stopped.

EPM remains a frustrating disease for equine veterinarians and horse owners alike. Ongoing research will hopefully shed more light on the life cycle, accurate diagnostic modalities, new treatments, and effective prevention.
Colic is a general term for abdominal discomfort. However, the signs consistent with colic are very nonspecific and certainly do not represent specific lesions and diagnoses without further evaluation. This talk will go through a variety of common and unique cases of colic. The history, workup, treatments, complications, outcome, and interesting facts and tricks will be discussed throughout each case. In general, the tenants of the approach to a colicky case are the same and will be constantly reinforced. This approach is detailed in the proceedings for Colic: Treatment options and when to refer. This session will be interactive and audience participation is highly encouraged.
Approach to the Horse in Shock
Jarred Williams, DVM, PhD, DACVS, DACVECC
University of Georgia
Athens, GA

All cells need ATP to survive and function. Within each cell, the mitochondrion is able to manufacture ATP in 2 main ways; with and without oxygen. The process utilizing oxygen to create ATP, known as aerobic respiration, is very efficient and can make up to 32-36 ATP. The efficiency of this process lies with the electron transport chain and oxidative phosphorylation, which collectively account for 28-32 of the total 32-36 ATP. The remaining 4 ATP are created through glycolysis and the Krebs cycle, oxygen independent processes. The creation of ATP without oxygen, known as anaerobic metabolism, is therefore much less efficient. For a cell to survive on such little ATP, its usage or cellular metabolism, known as oxygen utilization (VO$_2$) must be minimal. Unfortunately, in almost every scenario, a cell’s metabolism is not low enough to sustain such minimal ATP production, as is created by anaerobic metabolism, and thus, the cell dies under conditions in which oxygen delivery (DO$_2$) is not adequate. The balance between DO$_2$ and VO$_2$ is vital, such that DO$_2$ must always be greater than VO$_2$ for sustained (>5 minutes) cellular life. When VO$_2$ exceeds DO$_2$, cells will die, leading to organ failure and ultimately death. Shock is the term that describes that this is happening and there can be a number of reasons for it to happen. When attending a horse that may be in shock it is vitally important to identify that the patient is in shock, and why it is in shock.

In most cases of shock, the problem is oxygen delivery, rather than an inability to use the oxygen once it is delivered. A primary function of the cardiovascular system is to provide a circuit by which oxygen is gathered in the lungs, transported via vessels to the heart, pumped to cells, and then returned to the heart for pumping back to the lungs. Any problem that arises in the circuit represents a potential reason for shock. A problem with the pump represents cardiogenic shock. A problem with vascular tone, or the vessels, represents distributive shock. An occlusion of the vessels, or pump, represents obstructive shock. And, a problem with the volume of the vessels represents hypovolemic shock. When the cardiovascular circuit is completely functional, a patient could still be in shock if the cell is unable to process or utilize the oxygen properly, this is known as metabolic or cytotoxic shock. The attending veterinarian should always start with an examination that seeks to identify abnormalities in aspects of the cardiovascular system, or circuit, that could lead to a patient in shock.

When first examining a horse that may be in shock, a triage examination should be performed. The components of the triage examination include evaluation of mentation, assessment of the temperature of the extremities (ears and legs), assessment of mucous membrane color and capillary refill time, palpation of peripheral arterial pulses, jugular refill time, cardiac auscultation for rate and rhythm, respiratory rate and auscultation for breath sounds, and systemic temperature. Abnormalities in aspects of this examination provide information to the clinician that there is, or at least could be, a problem with systemic circulation. Blood work can also be very beneficial in recognizing that the patient is in shock. Lactate is a byproduct of anaerobic metabolism. When a patient is systemically hyperlactatemic, a good assumption can be made that oxygen delivery is insufficient. However, it is very important to remember that in this scenario oxygen delivery can be insufficient because delivery is decreased beyond cellular need, or, that delivery is normal or even increased and cellular demand/utilization has increased beyond the cardiovascular system’s ability to supply enough oxygen. Packed cell volume and total protein both give a rough estimate of volume status, as can creatinine. Creatinine, however, can also be elevated for renal or post renal reasons, so total reliance on this value for volume status can be difficult.

Assessing specific regions can also be very helpful. For example, cool extremities indicate poor perfusion to the periphery and suggest shunting of blood flow to the core. Absence of urine production, suggests that glomerular filtration is diminished from lack of volume or renal perfusion. Evaluating urine specific gravity can give insight into the kidney’s ability to concentrate as well as hydration status. Altered mentation is also a very valuable indicator of perfusion as it can suggest that cerebral blood flow could be decreased.

For horses, the most common type of shock is hypovolemic. Therefore, prompt venous access and commencement of intravenous fluid therapy is essential. The most rapid way to expand vascular volume is through hypertonic saline (7.2% NaCl), though this is only a temporary bridge as the hypertonicity of the fluid primarily helps for extravascular fluid recruitment. The dose for hypertonic saline is 2-4 ml/kg. Caution should be taken in administering hypertonic saline to patients with a history of chronic diarrhea. Following the hypertonic saline with isotonic crystalloid is recommended. The shock dosage is 90 ml/kg; however, it is most prudent to begin with 20ml/kg boluses of isotonic fluid and reassess cardiovascular status before increasing to the next 20 ml/kg bolus. Oftentimes, patients in hypovolemic shock may also be hypoproteinemic to the point of poor colloid oncotic pressure. These cases frequently benefit from vascular expansion in the form of colloid (plasma, hetastarch, etc.).

Horses in cardiogenic shock need the exact opposite of what patients with hypovolemic shock need. These cases have a detrimentally low cardiac output and need volume taken off of the cardiovascular circuit. Therefore, intravenous fluids are contraindicated in most situations. Furosemide is a good way to emergently decrease vascular volume via increased urination. Many of these cases will also benefit from oxygen supplementation and a vasodilator, such as nitroglycerin, if they have pulmonary edema.
Horses in distributive shock are typically foals. These patients are hypotensive and are in need of medications that improve mean arterial pressure. Drugs such as dobutamine, norepinephrine, and vasopressin each work differently to improve this value and hopefully ameliorate the effects of the hypotension and distributive shock. Horses in obstructive shock need the obstruction removed. The most common example of a horse with obstructive shock would be the patient with a large colon volvulus. In this scenario the colon can expand to the point that venous return is diminished from an extraluminal occlusion of the vena cava. Cardiac output is drastically diminished from the poor venous return and decreased preload. Finally, horses in metabolic or cytopathic shock need the cause for cellular dysfunction identified and removed, if possible. In horses, endotoxemia is a potential cause for cellular dysfunction and anti-endotoxic therapies such as polymixin B and low dose banamine are recommended.

In summary, identification that the horse is in shock is the most important step in managing these cases. The triage examination and initial blood work can go a long way in instituting a plan. In most scenarios of shock the patient is hypovolemic and intravenous fluid therapy should be initiated. However, the importance of recognizing cardiac dysfunction before instituting intravenous fluid therapy cannot be overstated.
The decision to refer a horse with signs of abdominal pain to a tertiary center is case dependent based on a number of factors evaluated together. Any colic workup should include a systematic approach to determine the section of the gastrointestinal tract involved and the broad category of disease: non-strangulating obstruction, strangulating obstruction, or inflammation. Based on the section involved and category of disease, the decision to treat at the farm or refer can be efficiently made. However, it is essential to consider that history and degree of pain may necessitate early referral, before a complete examination can be performed by an attending veterinarian. Finally, before the decision to treat at the farm versus at a referral center is made, it is important that the owner and veterinarian have a firm grasp on the capabilities, financial and medical, of the respective parties.

There are so many factors that go into a veterinarian recommending referral to a tertiary facility, and an owner accepting that recommendation. There are no set criteria for this recommendation, and each veterinarian has their own findings that they are comfortable managing or not. The most important take away point from this talk is to be sure of your findings before deciding to manage the patient at the farm, as delayed referral can negatively affect outcome. A substantial advancement in colic management and surgery has been early referral. One important note to consider is that oftentimes patients are referred to a secondary facility to more efficiently receive medical management from a veterinarian rather than repeated visits to a farm. For the remainder of this proceeding, “referral” will be defined as referral to a tertiary facility from either a farm setting or a secondary facility capable of administering medical management. The objective of this report is to address the 3 main aspects of referral: the client/owner, the farm visit, and the referral center.

A colicky horse can be a very stressful event for an owner to manage. Many owners are unfamiliar with the typical signs of colic, and thus, can range in response from a delay in recognizing them to completely blowing them out of proportion. For this reason, the best start to management of colic is owner education prior to having a horse actually colic. The owner education should include ensuring they know what the different signs of colic are, establishing a standard operating procedure for when a horse does colic, and making sure the client has access to transportation for their horse. The SOP should be tailored to the individual veterinarian’s preference for treatment prior to arrival at the farm, the owner’s ability to administer medical therapy, and the veterinarian’s comfort with the client. This frequently includes walking, removing food, taking a heart rate, assessing degree of abdominal distension, gauging degree of pain, and flunixin meglumine administration. It is so important to recognize that this approach may or may not work for each individual veterinarian or client. Great communication is imperative to outcome, and the more that has been set up prior to an event, the smoother things may go once a situation arises. Along those lines, it is important to discuss with the owner which referral center would be desired should the need arise, and to establish with that center what the average cost of referral, general medical management, and surgical management are. It should not be expected that such estimates would need to be definitive; however it is very important that the owner have a rough idea of the cost of things. Additionally, conversations regarding insurance policies and payment expectations/types at the referral center are beneficial to have prior to an emergency situation. Oftentimes, the delay in referral or surgery is due to a misunderstanding of cost and outcome, which frequently leads to higher bills and worse outcomes.

Few aspects of colic surgery have influenced outcome more than the farm visit. Early and accurate identification of the general problem, coupled with appropriate referral based on these results, have drastically improved postoperative success. In other words, the healthier the patient and gastrointestinal tissues are, the less the complications. When an attending reaches a farm, it is best if they immediately establish themselves as the leader of the scenario while making sure a few important tasks are achieved: ensure the safety of anyone involved, alleviate the patient’s discomfort, even if it is only temporary, and perform a few necessary diagnostic tests. Whenever possible, the colic examination should begin with a triage examination to identify if the patient is cardiovascularly stable or in shock. This examination should be brief, efficient, and include evaluating mucous membranes, capillary refill time, jugular fill, facial artery pulse, mentation, palpation of extremities (ears and distal limbs), heart rate and rhythm, and respiratory rate and effort. If the patient is deemed cardiovascularly unstable immediate referral is recommended. If the facility is nearby (within 30 minutes), referral without treatment for the cardiovascular instability is reasonable; however passage of a nasogastric tube prior to shipment is still recommended.

If the referral center is not nearby, stabilization with intravenous fluids (1-2 liters hypertonic saline and/or 5-10 liters of isotonic fluids) via jugular vein catheterization is reasonable, particularly if the fluids are administered while in transit, as well as passage of a nasogastric tube. The decision to keep the nasogastric tube in place while transporting is dependent on the estimated length of travel, the estimated rate and volume of gastric fill, and the patient’s temperament. In general, if there are concerns that the gastric volume could exceed 6-8 liters within the period of shipment, the safest course of action would be to leave it in place. Securing the tube in
place via taping it to the halter or rostral muzzle, protecting the cornea from reflux, and placement of a one way valve, such as a glove with a finger removed, are all important factors to consider when shipping a horse with a nasogastric tube in place.

If the patient is cardiovascularly stable, the triage examination should be expanded to include gastrointestinal sounds, digital pulses, and temperature. If the patient is too painful to allow for a complete examination to be completed, sedation should be administered to facilitate the remainder of the examination. Following the physical examination, a nasogastric tube should be passed. This procedure allows for 3 important aspects of the work up to happen: to alleviate gastric distension if present, to assess aboral movement of ingesta, and to provide a route for intragastric fluid, electrolyte, or laxative administration.

When possible, a rectal examination should be performed. Limiting factors precluding a rectal examination include patient size, temperament, degree of pain, and location to safely perform the procedure. Findings of the rectal examination can help to determine whether a case should be referred early or attempted to be managed at the farm; though it should not be the only determinant of this decision. Generally speaking, rectal palpation findings of small intestinal distension and very tight large viscus gas distension are those in which early referral should be considered. Large and small colon impactions, as well as, mild to moderate large viscus gas distension, are findings consistent with lesions that may be amenable to treatment at the farm.

While not always performed at the farm, transabdominal ultrasound and an abdominocentesis can also provide valuable information for determining the lesion. Like with the findings of the rectal examination, transabdominal ultrasound should be used to gain information to help the decision making for referral, but should not be the only piece of information used. Evidence of small intestinal distension, a diaphragmatic hernia, a target lesion suspicious of an intussusception, intramural thickening of any section of the GI tract, particularly the small intestine or large colon, and a hemoabdomen are findings that warrant early referral. Any abnormal abdominal fluid collected via abdominocentesis should be recommended for early referral, particularly if it is sanguineous, serosanguinous, or purulent.

The goal of the physical examination, nasogastric intubation, and rectal examination is to determine if the lesion is small intestinal or large intestinal. As a basic recommendation, cases in which referral is an option, nasogastric reflux is obtained and distended small intestine is palpated per rectum should be referred regardless of pain. It is difficult to predict whether the reflux and small intestinal distension are transient, and without an abdominocentesis, impossible to know if the lesion is strangulating. Therefore, early and immediate referral gives the patient the best chance of survival and minimizes what could be numerous farm visits by the referring veterinarian. When the lesion is a large intestinal lesion, the options would be a strangulating obstruction, non-strangulating obstruction, or inflammation.

Inflammation of the colon is colitis, which is frequently associated with diarrhea and an infectious agent, therefore referral to an isolation facility is often in the best interest of all involved. The differentiation between a strangulating and non-strangulating obstruction of the large intestine is largely based on degree of pain and history. The lesions that fit within a strangulating obstruction include a large colon volvulus or an intussusception (cecocecal or cecocolic). With both lesions the patient is typically very painful despite sedation. Furthermore, the typical history of a postpartum broodmare is so suggestive of a large colon volvulus, that referral without even seeing the mare is a reasonable approach. Thus, non-strangulating lesions are really the only recommended large intestinal diseases that should be attempted to be treated at the farm. All other lesions are best suited for early referral.

When referral is an option per the owner, these specific non-strangulating obstructions are reasonable to treat initially at the farm: large colon impaction, small colon impaction, nephrosplenic entrapment, right dorsal displacement, and spasmodic tympanic colic. However, if medical management is not successful in leading to resolution or improvement in discomfort, referral to a tertiary facility should then occur. It is very difficult to assign time recommendations for medical management at the farm prior to referral, because certain conditions can take days to resolve (impactions) while others should resolve more quickly with medical management if they are to respond (displacements). When surgery is an option for these patients, the postoperative outcome will likely be more positive if the patient is not sick and the gastrointestinal tissues are healthy. Therefore, medical management at the farm should inhibit these factors.

As a general rule, if the patient remains stable and relatively comfortable, treatment at the farm for 12-24 hours is reasonable. If the rectal examination worsens during this period of time (increased gas distension, worsening of an impaction, or the development of small intestinal distension) the patient should be referred. After 12-24 hours, it is still reasonable to continue to treat a horse with impaction colic medically at the farm if they remain cardiovascularly stable, are still only very mildly uncomfortable, and the rectal examination and fecal output indicate that the condition is improving and the treatment is effective. With that being said, the vast majority of cases are non-strangulating large intestinal obstructions that respond to a dose of flunixin meglumine, a walk, perhaps a dose of sedation for the evaluation, and just a small tincture of time (1-2 hours).

Clinicians at a referral center are trained to evaluate the “worst of the worst”, just like most field veterinarians are experts at managing colic without going overboard on diagnostics, treatments, and cost. What is a common case to a referral facility may in fact be a relatively uncommon case to the rest of the community, so taking a second to remember that fact typically allows for an understanding of what has been done at the farm and what will then be done upon arrival.
In general, cases that are referred to a tertiary facility are those that need an isolation facility, are in shock, have a strangulating lesion and need immediate surgery, or are non-strangulating lesions that have not responded to initial medical management. However, it is common that this delineation has not been definitively determined prior to arrival, so the work up is based upon trying to figure this out. Upon arrival, the triage examination, colic examination, nasogastric intubation, and rectal examination will be repeated. Depending on the age, pain, and history, the attending clinician may choose to perform bloodwork, abdominal radiographs, transabdominal ultrasound, and an abdominocentesis. If, based on these examinations a strangulating lesion is suspected, surgery is recommended; however if it is believed that the lesion is non-strangulating, efforts to get the horse through the colic with medical management will be made.

Taking a horse with a strangulating lesion to surgery is an easy decision, as is not taking a horse with colitis to surgery. The difficulty of management of colic at a referral practice is deciding when to take a horse with a non-strangulating lesion to surgery. Regardless of the other factors, persistent pain is the number one reason to take these cases to surgery. Other reasons would include a change in systemic health, a negative change in the belly tap, worsening of rectal examination findings, increase in nasogastric reflux, or the lack of progress despite no other changes.

The decision to refer can be very simple or agonizing. As bottom line guidelines, if the horse is in shock, in severe pain despite sedation, is a postpartum broodmare, is reflexing, or has distended loops of small intestine on rectal, it should be referred without hesitation. If there is suspicion that the lesion is a non-strangulating obstruction of the large intestine (large colon impaction, nephroplenic entrapment, right dorsal displacement, small colon impaction, or spasmodic/tympanic colic, then field management is reasonable.
Conservative Fracture Management
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Few calls in veterinary medicine conjure up negative thoughts like those involving the words fracture and horse. Diagnosing displaced and/or open long bone fractures is usually not a challenge, and, based on the bone involved, severity or configuration of the fracture, long and short term prognosis for recovery, and financial interest of the owners, the recommendation for referral and treatment or euthanasia is oftentimes clear. This scenario differs dramatically when the fracture is non-displaced. The patients, like those with displaced fractures, are oftentimes acutely very lame on the affected limb. However, unlike patients with displaced fractures, horses with non- or minimally displaced fractures are usually still able to bear weight, albeit reluctantly, and may not have obvious areas of swelling. In those scenarios, the field diagnostics can be a challenge. Often an area of the limb is suspected as a site of interest based on the physical examination, and is radiographed and diagnosed. Other times, the site of interest is more difficult to identify, and the patient is referred to a clinic with the ability to do more advanced diagnostics such as nuclear scintigraphy or radiography with units that have greater output capabilities. Regardless of whether the diagnosis is made in the field or in hospital, once the diagnosis is made, instituting a realistic and achievable plan for treatment is necessary for a successful outcome. This session is focused on accurately identifying which patients with non- or minimally displaced long bone fractures that are best treated medically versus surgically, how to manage these patients as they heal, and to determine if this is something that can be managed at the farm.

When it comes to determining how to manage a minimally or non-displaced fracture, most decisions come down to cost, well-being/comfort of the patient, and prognosis; in other words, what is the cheapest option that will maximize comfort and prognosis. There are a number of factors that go into determining how to achieve this. Above all else is comfort. If a patient is reasonably comfortable on the affected limb, than the clinician has options regarding treatment choice. If the patient is unacceptably uncomfortable despite analgesia and external coaptation, than methods of improving comfort through surgical stability must be considered, as prolonged periods of time in this given state of discomfort can and will lead to the patient’s demise. Assuming comfort, the next most important factor to consider is long term outcome; is this patient going to be able to return to their intended use. In consideration of this concept, joint involvement must be ascertained. In cases of minimal displacement, when the displaced fracture line extends to the joint surface an incongruity in cartilage can be created. Such cartilage involvement can lead to arthritis, increased resorption of the fracture line with synovial fluid, and potentially subchondral bone cyst formation; conditions that can all limit the patient’s ability to return to intended level of function. When it appears that the fracture does not extend into the joint, or that the fracture line extending into the joint is either non-displaced or minimally displaced with minimal joint incongruity, the option for non-surgical management can produce a similar long-term outcome as surgical management.

The next level on the decision making tree involves factors that may or may not affect outcome that should be considered. Hypothetically, if the patient is comfortable without surgical management and the long-term outcome will not change based on treatment choice, than why would one ever consider the more costly surgical option. Surgical treatments that involve internal fixation take advantage of bone healing principles that allow for a stronger bone in a shorter period of time. Thus, if an earlier return to function is needed by the owner, than the more costly option for surgery may be more beneficial. This opens the door for decision making that is based on desire and not necessity, which is routine and acceptable, as long as other elements are weighed in the decision making process; these include access to the affected bone, additional soft tissue trauma that could be encountered in the repair, risk of complication upon anesthetic recovery, and risk of surgical complication such as infection or implant failure. When the added risks of elective surgical management of the fracture outweigh the benefits of surgical intervention, medical management can be elected in the aforementioned scenario. Of course medical management of fractures is not without complications, therefore the remainder of this discussion will address concepts of the management that should be considered and addressed to maximize the potential for a successful outcome.

When attempting to manage these cases, there are a few aspects of care that must be addressed: analgesia, stability of the limb, and the systemic health of the patient. Maintaining comfort can be one of the most challenging tasks. Failure to do so can lead to a number of problems such as support limb laminitis, ileus, colic, gastrointestinal ulceration secondary to the stress of pain, anorexia, and complications secondary to not eating or drinking adequately. Providing adequate analgesia can be achieved with the singular systemic administration of any number of drugs within their respective drug classes, such as non-steroidal anti-inflammatories, opioids, or alpha-2 agonists, or via a multimodal approach with any number of combinations between these drug classes. Local analgesia can be achieved with the use of perineural catheters or occasional nerve blocking. Hindlimb injuries have the advantage of being able to benefit from epidural analgesia either through continual injections or an epidural catheter.

An equally challenging task to maintaining adequate comfort is ensuring that fracture does not displace further. Depending on the fracture configuration this can be achieved with external coaptation in the form of casting or bandaging. However, many of the long
bones that you would attempt to treat medically cannot be adequately coapted, therefore, methods to minimize increased force on the healing bone can be employed. These methods include placing the patient in a sling, tying them to the front of the stall, or placing them on “a wire”. The overall goal is to prevent the patient from lying down and minimizing movement in the stall to decrease the force placed on the bone associated with movement and standing. Along with the affected limb, attention needs to be given to the contralateral foot. A variety of treatments are used that include, but are not limited to, removing an existing shoe if applicable, shortening and beveling the toe or hoof wall, placing the hoof in a Soft-Ride boot, packing the sole and frog with the Equine Digital Support System, elevating the heel, casting the foot, or placing the foot in an elevated heel wedge.

Finally, paramount to success is maintaining the overall systemic health of the patient. Fresh hay and water should constantly be provided, especially if the patient is in a sling or tied and these items are hung. Ensuring that the patient is defecating regularly and that the feces is of normal consistency is a very important monitoring step and abnormalities should be identified and treated early before signs of colic are observed. The patients are frequently receiving NSAIDS, thus total protein and creatinine should be routinely monitored to assess trends indicating drug side effects. These patients are going to be stall confined; therefore ensuring fresh bedding, minimizing dust and flies, and maximizing good ventilation are important. Patients that are tied or on a wire are unable to drop their heads properly and allow their sinuses and nares to drain mucus collected from the actions of the mucociliary apparatus, so 1-2 times per day they should be allowed to drop their head for 10-15 minutes. This temporary relief must be monitored. Finally, numerous enrichment tools should be used to stimulate the patient’s mental health such as toys (i.e. jolly ball and sweet lick), treats, and constant grooming and attention.

When all aspects of the patient’s well-being are addressed, difficult cases such as long bone fractures can be managed successfully in the short and long term when accurate case selection has occurred. These cases do not always need to be referred to a specialty clinic, and can be managed at the farm or at smaller clinics when attention to detail and identification of problems can be achieved quickly and precisely.
There are a number of reasons to recommend surgical removal of one or both ovaries (ovariectomy); most commonly are behavior modification, hormonal manipulation, and removal of neoplastic tissue. For all the reasons above, pharmacologic intervention is commonly the method of medical management, and is frequently successful in alleviating the unwanted behavior. Unfortunately, medical management is not always permanently successful, thus the next method of treatment is surgical removal. The purpose of this presentation is to explain the most recent information regarding when and why to perform an ovariectomy, describe the different techniques currently recommended and performed, and discuss the overall cost and expectations to the client.

The primary reason a client begins to inquire about hormonal manipulation or ovariectomy of their mare is due to behavior. The exception to this situation is the mare that has normal ovaries and a bilateral ovariectomy is desired to hormonally manipulate her for the purpose of a teaser mare. The undesired behavior described is usually one of three scenarios; the mare is consistently difficult to manage or “grumpy” around the time she is ovulating, the mare has recently experienced a consistent change in personality regardless of the timing of ovulation, or the mare has begun to display signs of “stallion-like” behavior, such as herding, mounting, or aggressive tendencies (kicking, biting, etc). Clinically, these mares may begin to have increased muscle mass in their necks (cresty), may vocalize or squeal more frequently, guard their flank, flank watch, colic around the time of ovulation, or exhibit signs of poor performance (lamesness, refusal, reluctance to open up gate, etc). Reproductively, the mares may show anestrus, intermittent or continuous estrus, or may continue to cycle without issue.

Transrectal palpation of the ovaries will yield a variety of outcomes. The most obvious abnormality that can be palpated is one very large ovary ranging in size from a softball to a beach ball, with the opposing ovary palpating smaller than normal. It is also common that both ovaries can palpate normally, or perhaps one ovary seems normally sized and the other one is subjectively smaller. Rarely, both ovaries can palpate larger than normal. In all scenarios, ultrasonography is recommended. The ultrasonographic appearance may be polycystic, especially in the case of neoplastic tissue, though this will vary. The ovaries may also have the presence of hematomas, or rarely, an abscess, increased hyperechoic tissue, large follicles, or appear normal.

The most common pathology associated with the ovary leading to many of the aforementioned abnormalities is the presence of a sex cord-stromal tumor; more specifically, a granulosa cell tumor (GCT). By definition, a GCT is composed primarily of granulosa cells, though it is common for these tumors to also contain theca cells, and is termed a granulosa-theca cell tumor (GTCT). While histologically distinct, these tumors are clinically regarded as similar. Collectively, GCTs retrospectively represent approximately 85% of all equine reproductive tumors and about 2.5% of all equine neoplasms in general.

As expected, the gold standard diagnostic test to confirm the presence of neoplastic tissue is histopathology. In most scenarios, the recommendation for obtaining a tissue sample for histopathology is via a biopsy; however, in the case of equine ovarian tumors, the recommendation is removal of the entire ovary via ovariectomy. Because of the invasiveness and absoluteness of removing the ovary, preoperative testing to guide the decision making process is very important and needs to be accurate.

There are a number of blood tests that vary in their accuracy. Like any assay, the accuracy is dependent upon the methodology of the test and the laboratory the test is performed in. Historically, testosterone, estradiol, progesterone, follicle stimulating hormone, luteinizing hormone, and inhibin have been measured. These endocrinologic assays have been evaluated and compared to a gold standard, histopathologic confirmation of a GCT, in a number of studies. Through these efforts, it was determined that testosterone and inhibin were the most accurate indicators of the presence of a GCT, with elevated serum concentrations being detected in 67% and 87%, respectively.

Recently, antimullerian hormone (AMH) expression has been evaluated in normal equine ovaries, as well as those with histologically confirmed granulosa-cell tumors. In normal cyclic mares and in pregnant mares, there was no effect of cycle stage or month of gestation on serum AMH concentrations. However, in GCT mares, serum concentrations of AMH (1901.4 ± 1144.6 ng/mL) were higher than those in cyclic (0.96 ± 0.08 ng/mL) or pregnant (0.72 ± 0.05 ng/mL) mares, and serum AMH concentrations decreased after tumor removal.

AMH is a transforming growth factor beta (TGF-β) / bone morphogenic protein (BMP), and is produced prepartum by both male and female embryos, as well as in the granulosa cells of reproductively capable females and in the testis of males. AMH serves a variety of functions including inhibition of the development of the paranes nephric (muellarian) ducts in male embryos, inhibition of excessive follicular recruitment in females, and as a measure of the health of the ovary and the remaining reproductive potential of an individual (females).

When compared to testosterone and inhibin, AMH is advantageous as a marker for equine ovarian neoplasia due to its increased sensitivity for detection of GCTs. AMH has a sensitivity of 98%, versus 80% (inhibin) or 48% (testosterone) or both (84%) in...
detection of GCTs later confirmed via histopathology. Thus, the most sensitive and recommended hormone to evaluate is currently AMH. The Clinical Endocrinology Laboratory at the University of California, Davis offers an endocrinology panel which includes AMH, testosterone, and inhibin. The current cost for this panel is $130.00, and they require at least 3 ml of serum. If only the results of AMH are desired, the cost is approximately $60. The website, phone, and fax numbers are as follows:

Despite the reasons for an ovariectomy, there are a number of approaches for removal. The decision for approach is based upon size of the ovary, cost, available equipment, surgeon preference, owner preference, recovery time, risk, invasiveness, and anesthetic considerations. The described procedures that can be performed in the standing, sedated mare include a colpotomy, ovariectomy via flank laparotomy, laparoscopic ovariectomy via a flank approach, and a transvaginal endoscopic ovariectomy. If the ovary is very large (>10cm in diameter), it may need to be decompressed or morsellized in a laparoscopic retrieval bag prior to removal via a flank incision. Alternatively, in the anesthetized patient, the ovary may be removed via a caudal ventral midline or paramedian laparotomy.

Postoperative recovery time varies by approach and surgeon preference. Minimally invasive techniques typically allow 2-4 weeks of rest before returning to pasture turnout and/or exercise. Ovariectomy via flank laparotomy may require 4-6 weeks of rest prior to pasture turnout and/or exercise. Ventral midline or paramedical laparotomies may require 8-12 weeks of rest prior to pasture turnout and/or exercise. The postoperative prognosis for these procedures depends on the reason for the procedure. For bilateral ovariectomy for the purposes of hormonal manipulation for a teaser mare, the prognosis is excellent. For behavioral changes associated with ovarian pathology, the prognosis is also excellent. For behavioral changes with no pathology associated with the ovary, the prognosis is guarded for resolution of the behavior.

As to be expected, the cost of the procedure changes with the approach and these costs will certainly vary based on the hospital. The cost of a colpotomy is the most inexpensive option ($500-1000 range), but certainly carries a higher risk of serious complication (hemorrhage, sepsis, evisceration). Minimally invasive techniques involving the laparoscope in the sedated, standing mare are most typically in the $1500-2500 range, and are the least associated with complications. Ovariectomy via flank laparotomy may cost somewhere in between (~$1000-2000), with a higher degree of morbidity associated with the larger flank incision. Finally, the ventral midline or paramedical laparotomy, which requires recumbent anesthesia and oftentimes stapling equipment, can have a wide range of cost ($2500-5000).

Overall, ovariectomies are common and effective procedures for correcting a definitive problem in the mare and allowing a return to full form and function. They are generally considered an affordable procedure and are typically associated with minimal morbidity and mortality.
Disorders of the reproductive tract in captive birds are one of the most common reasons for seeking veterinary care. While reproductive disorders such as chronic egg laying, oviductal disease, ovarian disease, and testicular tumors are relatively easy to diagnose in avian patients, the treatment of these disorders remains challenging.

The female avian reproductive tract is characterized by a single left ovary and left oviduct. The oviduct can be subdivided into the infundibulum (site of fertilization), the magnum and isthmus (albumin and egg membrane production), the shell gland (shell formation), and the vagina, which enters into the urodeum of the cloaca. The ovary and oviduct will substantially enlarge in size in reproductively active female birds and the oviduct often fills the majority of the caudal coelomic cavity.

Environmental triggers are very important to initiate female reproductive activity in birds. In particular, in pet birds, overstimulation will lead to chronic and excessive egg laying and associated health problems. Exposure to long periods of day light (>12 hours), will trigger egg laying in most bird species. The availability of excessive amounts of food, offering food high in carbohydrate (e.g. corn, bread), fat (nuts), or protein (e.g. beans) will trigger egg laying. In addition to the nutritional content of the food offered, the consistency is also a very important trigger. Warm and soft food simulate the feeding of crop content by a mate, which is commonly performed as part of courtship. Therefore, food should never be offered warm or cooked. The availability of a nesting site, either as as designated nest box, a dark hiding space (e.g. space under furniture. Oviposition (expulsion of the egg) occurs approximately every 24 hours in chickens, and can take up to 48 hours in clutch laying psittacines.

Diagnosis of Reproductive Disorders

Signs of reproductive activity can be easily missed during a physical examination. Female reproductively active birds will often gain body weight, due to enlargement of the oviduct and liver. The pubic bones will be wider in birds with high circulating blood estrogen. The pubic bone width should be determined all avian patients. The cloacal opening is usually more relaxed and swollen in female birds which are reproductively active. Coelomic palpation may reveal an increased filling, mineralized eggs or distension with fluid. A brood patch, a featherless area of skin over the ventrum develops in many bird species with the onset of reproductive activity. Radiographs are very helpful for the investigation of reproductive diseases in birds. Proper radiographic position is important in order to increased calcium deposition in the long bones of female birds (i.e. medullary bone) is caused by high estrogen levels, which lead to deposition of calcium into the bone as a reservoir for future egg shell production. Medullary bone can be easily identified on radiographs. An increase in soft tissue density is seen in the caudal to mid coelom and is usually caused by an enlarged oviduct. Mineralized eggs can be easily visualized on radiographs. Unshelled eggs (e.g. retained unshelled eggs in the oviduct), cannot be visualized by radiographs but coelomic ultrasound or preferably computed tomography is required.

Routine blood work, consisting of a complete blood count and plasma biochemistry profile may reveal abnormalities which can indicate reproductive activity and/or disease. The white blood cells count is often increased in cases of oviductal infections (salpingitis) and egg-yolk coelomitis. However, in some species (e.g. macaws) white blood cell counts tend to increase during reproductive activity, without and underlying disease process being present. Increases in blood calcium are very common in female birds, and this hypercalcemia is caused by increased amount of calcium being mobilized and transported to the oviduct for egg shell formation. Plasma triglyceride and cholesterol levels will also increase, and blood may appear lipemic. These lipids are transported from the liver to the ovary during ovarian follicular development. Plasma proteins can also be increased due to the increased amount of protein transported to the oviduct for egg formation.

Treatment of Reproductive disorders

Environmental and Dietary Changes

Neither medical nor surgical treatments alone or combined are unlikely to completely resolve disorders due to chronic gonadal activity in birds. Identifying and correcting the environmental factors which may trigger continued reproductive activity, such as the availability of nesting sites, shredding of paper, feeding warm and soft foods (e.g., hand-feeding formula, oatmeal, cooked vegetables), excessive availability of food, foods of high energy density (seeds, corn, grains, table food), excessive day light exposure, and inappropriate petting is critical in helping manage these challenging cases. Without address the environmental cues all other chosen treatment (i.e. medical and surgical treatments) will most likely fail. Frequently dietary corrections are one of the most important steps to take for owners. Another important aspect is ensuring an appropriate bird-owner bond. Inappropriate petting (anywhere below the neck) should be avoided. If a bird gives preference to one particular family member, then increased interactions...
with other family member should be encouraged. Making a bird feel less “comfortable”, for example by moving the cage location, changing the cage interior, etc, may also lead to a decrease in reproductive activity. Reducing exposure to light (keep at < 12 hours) and removal of all potential nesting materials and sites is necessary.

Medical and Surgical Treatment Options
In contrast to many diseases seen in dogs and cats, no scientific studies are available which compare outcome and survival times of medical vs. surgical treatments in birds suffering from reproductive disorders. Therefore it remains challenging to make scientifically sound recommendations to clients, which are seeking to make an informed decision about the best treatment options available. Surgical treatment options as the sole therapy for reproductive disorders in birds carry a guarded to poor prognosis in most cases. While surgical correction is curative for certain disease presentations such as oviductal prolapse or oviductal impaction, the inability to completely remove the ovary surgically can predispose birds to secondary complications such as egg-yolk coelomitis due to persistent ovarian activity. Therefore, significant environmental adjustments to minimize triggering of reproductive activity as well as medical treatments are required to prevent continued ovarian activity and ovulation. In other reproductive diseases, such as ovarian neoplasia or ovarian cysts surgical intervention will not achieve cure, but may only lead to temporary improvement of the clinical condition. Considering the significant surgical and anesthetic risks of coelomic surgery, particular in small avian patients, medical treatment options are often more feasible, less expensive and therefore more frequently considered by veterinarians and clients. Over the past years, the clinical use of GnRH-agonists for treatment of avian reproductive disorders has been increasingly reported and several prospective research studies evaluating the efficacy of long-acting GnRH-agonists in birds have been published.  

GnRH acts as the main link of the central nervous system, which received environmental visual and tactile (e.g. day light exposure, food availability, tactile stimulation, courtship display) with the endocrine system represented by the anterior pituitary and the gonads. These environmental cues lead to increased release of short-lived GnRH (half-life of 3-4 minutes) in a pulsatile fashion from the hypothalamus into the hypophyseal portal vasculature and reaches the anterior pituitary gland where GnRH receptors are expressed. GnRH receptors on the anterior pituitary gland, and control synthesis and release of gonadotropins and therefore gonadal function and hormone production.

Long-acting GnRH supra-agonists used in birds

Deslorelin acetate
Commercial forms of sustained-release deslorelin acetate are available. A recent study in cockatiels showed that a 4.7 mg deslorelin implant suppressed egg laying for > 180 days. However, in clinical practice duration of effects is often shorter and not effective in all treated birds. In female Japanese quail (Coturnix japonica), in which deslorelin implants were only effective in 60-78% of the treated birds, In 60% (6/10) quail egg production was significantly decreased from 2 to 12 weeks after administration of a single 4.7 mg deslorelin implant. In a second study by the same authors 70% of the quail implanted with two 4.7mg deslorelin implants, produced significantly less eggs between week 1-15 following implantation. In contrast quail implanted with a single 9.5 mg deslorelin implant showed no reduction in egg production until week 12 following implantation. Egg production was significantly less compared to the control group, from week 12-26. However, the study was terminated after 26 weeks, and therefore a possible longer effect could not be excluded. In another study in male and female Japanese quail found that a single 4.7mg deslorelin implant was effective in 89% of the birds. In female quail egg production was reduced in 7/9 birds (78%) within the 1-5 week post-implantation. Egg reduction was reduced for 7-18 weeks, with 5/7 birds not laying eggs for more than 14 weeks. In male quail the duration of the effect was reported to be 2-13 weeks, based on, a substantial reduction in circulating testosterone and estrone sulfate levels (10/10 animals) and other parameters, such a size of the cloacal gland. Testosterone levels were significantly reduced in all male quail within 3 days after implant administration. No adverse effects were reported in studies performed in chicken, quail or cockatiels and have also not been witnessed by the author in clinical practice. From these prospective studies it can be concluded that significant species differences in the efficacy and duration of deslorelin implants exist and that in chicken 9.5 mg implants provide longer suppression compared to the 4.7 mg implants. Retrospective case series reporting the use of deslorelin implants in psittacine birds have in the non-peer reviewed literature. In female birds suffering from chronic egg laying a single 4.7mg deslorelin implant suppressed egg laying for around 3 months in all treated 32 psittacine birds. Deslorelin has also been used in cases with reproductive driven feather destructive or aggressive behavior.

Deslorelin acetate implants have also been reported to lead improvement of clinical signs in a case series 10 budgerigars (Melopsittacus undulatus) tentatively diagnosed with hormone producing gonadal tumors (i.e. Sertoli cell tumors). The tentative diagnosis was made based on brownish discoloration of the cere, polyostotic hyperostosis and a soft tissue mass in the coelomic cavity. 4.7 mg deslorelin acetate implant were placed subcutaneously in the knee fold under general anesthesia. A response to treatment was reported in 7/9 budgerigars, evident by a change of cere color from brownish to blue. Further improvements included resolution of previously diagnosed lameness (3/3) and an improved general condition (7/8). Initial clinical improvement was noted...
after several days to 4 weeks. Reoccurrence of clinical signs was noted on average 19 weeks after initial deslorelin administration. Deslorelin implants were repeatedly administered after reoccurrence of clinical signs and the effect of deslorelin lasted on average 20 weeks. Treatment of suspected Sertoli cell tumors in budgerigars with deslorelin implants was found to be effective by temporarily treating the clinical signs, secondary to abnormal estrogen production and no significant side effects were reported.

References

Dental disease in rabbits and rodents

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Dental disease is one of the most common disorders diagnosed in rabbits and rodents. Rabbits and all rodents have continuously growing (elodont) incisor teeth. Rabbits and hystricomorphous rodents (e.g. guinea pigs, chinchillas, degus) are strict herbivores and in addition to continuously growing incisor teeth, also have continuously growing cheek teeth. In contrast myomorphous rodents (e.g. rats, mice, gerbils, hamsters) do not have continuously growing cheek teeth and are omnivorous. Continuously growing teeth in rabbits and rodents do not have a anatomical crown (aradicular) and instead a very large crown (hyposodont), which is divided into the “clinical crown” portion visible above the gingival margin, and the so called “reserve crown” below the gum line.

The dental formula of rabbits is 2I 0C 3P 3M / 1I 0C 2P 3M, and in hystricomorphous rodents: 2I 1I 0C 1P 3M, and in myomorphous rodents: 2I 1I 0C 0P 3M. The author prefers to call the premolar and molar teeth “cheek teeth (CT)”. Rabbits therefore have 6 cheek teeth (CT1-6) in the maxillary arcades and 5 cheek teeth (CT1-5) in the mandibular arcade. Hystricomorphous rodents have 4 cheek teeth (CT1-4) in each arcade, while myomorphous rodents have 3 cheek teeth (CT1-3) in each arcade, which are short crowned (brachydont) and have an anatomical root (radicular).1

There is no single etiology of dental disease in rabbits and hystricomorphous rodents, but instead a variety of underlying cause may lead or contribute to the development of dental disease in these species. Congenital disease is usually diagnosed early in life, while acquired dental disease can be caused by trauma, infection or nutritional disorders. Historically feeding a diet predominately consisting of grass hay has been proposed to prevent the development of dental disease, due to the high fiber content and the required increased attrition in order to ingest such diet. However, besides the high fiber content, the high Ca:P ratio in hay may play an equally important role in prevention of dental disease. An inappropriate Ca:P ratio has been shown to induce dental disease in degus2, and while not proven in other rodent species or rabbits, yet, it should be assumed that feeding a diet with excessive phosphorus may lead to dental disease in other species as well. Therefore feeding grains, sugar-containing treats or other dietary items high in phosphorus is not recommended.

Clinical signs associated with dental disease include selective food intake, refusal to eat hay, weight loss, hypersalivation, fur quality, ocular discharge (due to nasolacrimal duct obstruction due to apical cheek teeth elongation), unilateral exophthalmos (due to retrobulbar odontogenic abscessation) or facial swellings (due to odontogenic abscesses). Physical examination should pay attention to facial asymmetry, and appearance of the incisor teeth.

Examination of the cheek teeth is not recommended in conscious rabbits and rodents, since the information gained may be misleading, and the presence of dental disease and associated intraoral pain can never be ruled out in a conscious patient. A complete intraoral examination can only be performed under general anesthesia, just like in cats or dogs. To aid in the visualization of the teeth specialized dental tools such as the mouth gag, inserted between the incisor teeth in order to open the mouth from top to bottom, and cheek dilators that have spatulated wings that open the mouth from side to side with a spring action should be used.

The use of a rigid endoscope of video-otoscope is highly recommended for intraoral examination under general anesthesia. Endoscopy (stomatoscopy) provides focal illumination, magnification, and allows for documentation of normal and abnormal findings, which will aid in client education and medical record keeping. Using an endoscope for intraoral exams will reduce the risk of missing intraoral disease, in particular in chinchillas periodontal disease and buccal spurs of the last two maxillary cheek teeth can be easily missed if no endoscopy-guided intraoral examination is performed.

Skull radiographs, preferably 5 views that evaluate lateral, dorsoventral, rostrocaudal, and right and left lateral oblique projections, in order to more critically assess the teeth and jaw bones. If available a CT scan of the head is preferred to skull radiographs, since it allows for detailed evaluation of the teeth and surrounding bone without the summation effects seen on skull radiographs. In addition a complete evaluation of the retrobulbar space, middle ears and nasal cavities is possible and provides valuable information.

All intraoral dental treatments should be performed under general anesthesia. Performing intraoral procedures in awake animals is not acceptable, ineffective and carries a high risk of iatrogenic trauma. Treatment of coronal overgrowth as well as dental spurs should never be performed using so-called “molar clippers” or rasps, but are still sold as tools for treatment of dental disease in rabbits and rodents. The clippers will result in sharp edges and carry the risk of inducing vertical fractures of the teeth. The dental rasps, are highly inefficient and carry the risk of inducting soft tissue trauma. Instead, for adjustment of coronal height and occlusal surfaces, a
low speed hand-piece straight nose cone (1:1) attachment should be used in combination with a diamond bur tip and a soft-tissue protector.  

It is important to communicate to pet owners that coronal height adjustments or removal of spurs will require repeated anesthesia and treatment and therefore a substantial financial commitment of the client is required. It is important, however, to not perform dental treatments on a set schedule, but rather to monitor for changes in behavior or food intake, in order to avoid unnecessary anesthesia and dental treatments.

Extraction of incisors is routinely performed in rabbits, mainly for treatment of mandibular prognathism. The procedure is well tolerated and rabbits do well without incisors. Extraction of cheek teeth should be avoided, unless teeth are severely infected, fractured or non-viable. Pack the extraction site with Doxirobe™ gel in order to avoid impaction with food.

Odontogenic infection (infections originating from the teeth and surrounding structures) are commonly diagnosed and most commonly occur in form of periapical abscesses (“tooth root” abscesses) in rabbits and guinea pigs. In chinchillas periapical abscesses are uncommon and instead periodontal disease with gingivitis, caries and secondary tooth resorption are frequently diagnosed. Periapical abscesses can be associated with the incisor teeth or cheek teeth and may be palpable or visible as facial swellings. However retrobulbar abscessation in rabbits and guinea pigs is common and usually present as unilateral exophthalmos.

For diagnostic work-up of periapical abscesses diagnostic imaging in form of skull radiographs or preferable CT should be performed in all cases. Failure to identify the source and extend of the odontogenic infection will result in a higher chance of treatment failure. A complete intraoral examination under general anesthesia and sample collection for bacterial cultures (aerobic and anaerobic) as well as cytology is necessary in order to completely evaluate patients with periapical abscesses. It should be considered that strict or facultative anaerobic bacteria, such as Bacteroides, Prevotella, Fusobacterium and Actinomyces species, play an important role in odontogenic infections. Therefore if empirical antibiotic treatment is initiated pending bacterial culture results, appropriate antibiotics, which are effective against anaerobic bacteria, should always be prescribed. Most odontogenic infections are mixed aerobic-anaerobic infections, and aerobic bacteria such as Streptococcus, E.coli or Pseudomonas are also frequently isolated concurrently.

Different therapeutic techniques have been recommended in the literature for treatment of periapical abscesses in rabbits and rodents. It should be considered that most of the recommended techniques have not been evaluated in randomized clinical trials in order to determine their efficacy and risk of reoccurrence or treatment failure. It should also be considered that severity of the infection usually correlates with prognosis and the patient’s systemic health status and immune system will influence the prognosis. Regardless of the technique used, clients should be thoroughly educated about the prognosis, risks of treatment failure and reoccurrence of infection, the associated usually substantial costs of therapy, and side effects. Therapeutic techniques reported include complete surgical excision of the abscess and removal of the involved diseased teeth and bone followed by either placement of antibiotic impregnated beads or marsupialization of the surgical site to allow for continued drainage and open wound management. An alternative technique described in the literature, which has also been evaluated in form of a retrospective study, involves lancing of the abscess and packing with antibiotic-impregnated gauze, followed by surgical closure of the wound and replacement of the gauze strips every 7 days until resolution of infection. Retrobulbar abscesses in rabbits can be successfully treated without the need of enucleation, by removal of the involved maxillary cheek teeth to allow for drainage followed by systemic antibiotic therapy. Regardless of the surgical technique used for treatment of periapical abscesses the author strongly recommends systemic appropriate antibiotic therapy, which is effective against anaerobic bacteria, such as metronidazole, penicillin (parentally only) or azithromycin, in addition to treatment against aerobic bacteria if isolated. Enrofloxacin and trimethoprim-sulfa drugs are not effective against most anaerobic bacteria. Supportive care and pain management are important measures in the post-surgical period.
References


Incortrect husbandry accounts for the majority of diseases encountered in captive reptiles. Knowledge about the natural history and unique environmental and nutritional requirements is important for veterinarians to diagnosis and treat husbandry related disorders. This lecture will discuss the common husbandry-related disorders and provide information on their treatment and prevention. Reptile patients have specific environmental and nutritional requirements in order to remain healthy. Due to the variety of reptile species maintained in captivity, veterinary treating reptiles should be familiar with the basic concepts of reptile husbandry, as well as the specific needs of commonly kept reptile species.

Environmental temperature
Reptiles are ectothermic and therefore the body temperature is directly affected by the environmental temperature. In their natural environment reptiles regulate their body temperature, by behaviors such as basking (increasing body temperature) or hiding under cover, in order to maintain their body temperature within their preferred optimal temperature zone (POTZ). Most physiological processes, such as immune function, growth, and digestion are directly affected by body temperature. In captivity, the lack of an appropriate temperature gradient within an enclosure often prevents reptiles from regulating their body temperature. For most species, a focal basking light should be positioned at one end of the enclosure, resulting in an area with the highest environmental temperature. If sufficient ventilation is provided, the opposite end of the enclosure will remain cooler. Hence a temperature gradient is created, allowing the reptile to choose the correct temperature zone, in order to modify it body temperature as needed for various physiological processes (e.g. digestion, etc). In addition, a day-night temperature change is important. Night time temperatures should be lower than day-time temperatures in most species. The author prefers to use bright light emitting bulbs as heat source for the basking spot, since in the wild radiant heat is associated with visible sunlight. The use of ceramic heaters or red infrared bulbs as the primary heat source for basking spots is therefore discouraged. Other heat elements (e.g. under tank heat mats, tape or cables) can be used to modify the environmental temperature accordingly, but in most cases are not necessary. Species-specific shelter should be provided at various temperature areas within the enclosure. In their natural environment, hiding spots are usually cooler, since they are not exposed to sunlight, and therefore care should be taken to provide shelter at the cool end of the enclosure. Chronic exposure to high environmental temperatures without the availability of a temperature gradient, will lead to various health problems, including chronic dehydration, kidney disease, bladder stones in certain lizards and tortoises, and problems shedding.

UVB light exposure
Insufficient exposure to UV-B radiation (280-315nm) is well known to lead to secondary nutritional hyperparathyroidism and metabolic bone disease in many reptile species, in particular herbivorous ones. The biological active form of vitamin D₃ is 1,25 dihydroycholecalciferol (syn. calcitriol), which regulates calcium metabolism by increasing calcium and phosphorus absorption from the intestine, mobilizing calcium resorption from by bone. UV-B is necessary to activate the cholecalciferol pathway in species, which rely not on dietary vitamin D₃ intake, which is the case in herbivorous reptiles. If vitamin D₃ is predominately of dietary origin or endogenously synthesized differs between reptile species based on their nutritional (carnivorous, omnivorous, herbivorous) strategy and natural behavior (e.g. nocturnal, vs. diurnal) and natural habitat. In omnivorous and carnivorous reptiles the need to provision of artificial UV-B radiation in captivity remains controversial. In most reptile species evaluated, exposure to UV-B radiation will lead to increased plasma vitamin D levels. Exposure to artificial UV-B in panther chameleons (Furcifer pardalis) had a significant effect on plasma 25-hydroxycholecalciferol. Red-eared sliders (Trachemys scripta elegans) had significantly higher plasma 25-hydroxycholecalciferol concentrations if turtles were exposed to artificial UV-B radiation for 4 weeks. In corn snakes exposure to artificial UV-B radiation for 4 weeks significantly increased plasma 25-hydroxycholecalciferol levels. In contrast, exposing ball pythons (Python regius) to artificial UV-B radiation for 70 days had no significant effect on 25-hydroxycholecalciferol or ionized calcium levels. As a nocturnal species ball pythons might not need to utilize UV-B radiation for synthesis of cholecalciferol as compared to diurnal species. In addition dietary intake of vitamin D₃ affects basking behavior and therefore exposure to UV-B radiation. Panther chameleons adapt their basking behavior and exposure to UV-B radiation based on their dietary vitamin D₃ intake. Chameleons with less dietary vitamin D₃ intake chameleons spend more time basking. It needs to be remembered that most forms of artificial UV-B radiation provided in captivity are only an inadequate supplement for natural sunlight most reptiles are exposed to in their natural habitat. Hermann’s tortoises (Testudo hermanni) exposed for 35 days to either mercury or fluorescent UV-B radiation emitting light bulbs, had significantly lower 25-hydroxycholecalciferol plasma levels compared to days 0. While tortoises exposed to natural sunlight maintained their plasma 25-hydroxycholecalciferol levels. In bearded dragons (Pogona vitticeps) dietary supplementation of vitamin D₃, even at high doses, was insufficient to maintain plasma 25-hydroxycholecalciferol levels, compared to bearded dragons exposed to artificial UV-B radiation.
Direct unfiltered sunlight is preferred over artificial UV-B radiation sources, whenever possible, but challenging to accomplish in most captive housing situations. A variety of UV-B emitting light bulbs are available, which vary in intensity of the emitted UV-B rays as well as heat emission (i.e., mercury vapor vs fluorescent bulbs). It is important to note that UV-B rays are completely blocked by most glass and plexiglass products and can be significantly reduced by fine metal mesh. It is important to follow manufacturer guidelines in regards to optimal distance between light bulb and the animal, since with increasing distance the amount of UV-B reaching the animal is progressively reduced.

Insufficient exposure to UV-B radiation leads to well-reported clinical signs associated with Nutritional secondary hyperparathyroidism (NSHP). Lethargy, reduced appetite, constipation, dystocia or preovulatory stasis can be seen in animals with calcium deficiencies. However, these clinical signs are non-specific and other disease processes or husbandry problems should be ruled out. Skeletal deformities or fractures due to demineralization of the bones are common in reptiles suffering from calcium deficiencies. Fractures of the limbs and ribs are most commonly seen in lizards. Once total body calcium stores are depleted enough, so that blood calcium levels cannot be maintained at adequate levels, muscle twitching, tremors, paresis, and neurological signs can be seen. This hypocalcemic crisis, and is considered as an acute decompensation of chronic NSPH.

**Humidity**

Reptiles originate from a variety of natural environments with highly different degrees of relative humidity. In addition, within each natural environment humidity can vary greatly, and is usually higher in hiding spots, not exposed to sunlight as well as in areas with organic materials (e.g., soil, moss, etc.). In captivity, the humidity in reptile enclosures is affected by several factors, including heat, ventilation, and presence of organic material. Regular misting or fogging can aid in keeping humidity at the desired higher levels of tropical and subtropical species. Insufficient humidity can lead to a variety of health problems in reptiles, in particular to problems shedding (dysecdysis). If humidity cannot be maintained at sufficient levels in the entire enclosure, then hiding boxes filled with damp newspaper, coconut fiber, or other absorbent materials should be offered. Snakes and lizards will frequently use these “shedding boxes” during the shedding period and the risk of dysecdysis is reduced.

**Substrate**

A variety of substrates can be used in reptile enclosures. Newspaper or paper sheets offer the most hygienic option for substrate, as it allows for easy cleaning and monitoring. However, in species which require higher environmental humidity or like to dig in their substrate paper is not suitable for long-term use. Potting soil, mulch, and coconut fiber can be used as substrate, but often will retain a lot of moisture and the risk for molt overgrowth is increased. Sand is sometimes used for desert species and usually does not cause problems. However, accidental ingestion of sand (or any other substrate) can lead to GI impaction and obstruction. Therefore, food items should never be offered directly on the substrate, but in a flat bowl or stone, which reduces the risk of accidental substrate ingestion. Some reptiles will purposely ingest substrate (pica), which is frequently seen in lizards and chelonians. In such cases the substrate should be replaced with a material, which cannot be ingested. In addition, underlying causes for pica should be investigated.
Sedation of dogs and cats in veterinary practice is daily routine for a variety of procedures, such as radiographs and ultrasonography, or other non-painful, but potentially stressful procedures. However, historically for exotic pet patients, either manual restraint of conscious animals or general anesthesia used to typically performed, in order to complete most clinical procedures. General anesthesia predisposes patients to cardiovascular and respiratory depression, and may cause aspiration of gastric or crop contents, lead to hypothermia and result in prolonged recoveries. In contrast, manual restraint in conscious patients may be simple to perform, but can have negative consequences, including stress to the patient and/or handler, negative conditioning to the clinic environment (e.g., the person restraining or the towel used for restraint), hyperthermia, and the predisposition of trauma to the handler and/or patient. Several recent studies demonstrated that manual restraint of birds and small mammals causes increased body temperature and respiratory rate. In sick, old, or very stressed patients, acute collapse and death secondary to manually restraint may occur. Therefore, sedation techniques provide a useful alternative for reducing stress in exotic pets undergoing non-painful clinical procedures. Furthermore, sedation provides easier restraint and increases the safety of many clinical procedures (e.g., blood collection, radiography, ultrasonography) and allows for a more complete examination, which would otherwise only be achieved under general anesthesia, in particular in birds and small mammals. Using safe and effective sedative protocols in exotic pet patients, provides substantial benefits to the patients as well as the veterinarian and staff, and should be considered for a variety of clinical procedures.

Route of Drug Administration
Historically sedative drugs have been most commonly administered by intramuscular injection. However, the subcutaneous route should also be considered, since injection by this route result in less discomfort. Recently the intranasal administration of sedative drugs in birds, as well as mammals and reptiles has gained increased attention. Almost all drugs used for sedation, such as benzodiazepines, ketamine, alpha-2-agonist, and opioids can be administered intranasally. Intranasal drug administration offers an alternative, non-invasive technique for drug administration in birds. It is characterized by its ease of administration, high bioavailability, rapid onset of action, and reduced pain compared to intramuscular administration. Elevation of muscle enzymes in biochemistry panels secondary to intramuscular drug administration is avoided, if intranasal administration is used instead. In addition, clients perceive the intranasal route in birds as non-invasive, compared to intramuscular injection, which leads to better client compliance in cases in which sedation is recommended. The time of onset to sedation is rapid, typically within 3 - 5 minutes. However, limitations of intranasal administration include incomplete drug delivery, due to sneezing during administration, physiologically narrowed nostrils (e.g. cockatoos) or upper respiratory disease (e.g. blocked or stenotic nostrils). In some cases in larger birds (e.g. macaws) the drug volume can also be limiting the effectiveness and produce excessive sneezing, therefore leading to incomplete drug delivery. Higher concentrated drugs (e.g. midazolam 50 mg/ml, Zoopharm, Windsor, CO) are available, but intramuscular administration might be more feasible in these cases. In small mammals the intranasal route is rarely used, since administration is frequently challenging, and in particular in obligate nasal breathers, it may induce anxiety. In reptiles the intranasal route can be used for induction and reversal of sedation, but the subcutaneous route is generally preferred by the author.

Midazolam
Midazolam is currently the most commonly used drug for sedation of exotic pet species and has a wide margin of safety. Midazolam has sedative, muscle relaxing, anxiolytic, amnestic and appetite stimulating properties in birds. The injectable form of midazolam (midazolam hydrochloride (5 mg/ml); e.g. Hospira Inc, Lake Forest, IL) or a more concentrated form (50 mg/ml, Zoopharm, Windsor, CO) can be administered intranasally, subcutaneously and intramuscular, without side effects. Dosages commonly used in pet birds range from 0.5 - 3 mg/kg. At the University of Wisconsin, we routinely use 2 mg/kg of midazolam in pet birds, if administered intranasally and as the sole sedative agent. In smaller birds such as finches or budgerigars we routinely use 4-6 mg/kg of midazolam if administered alone. In small mammals midazolam is rarely administered alone, but usually combined with an opioid and frequently ketamine. In reptiles midazolam administered alone leads to inconsistent sedative effects and is therefore usually administered in combination with dexmedetomidine, ketamine or alfaxalone by the author. The author strongly recommends the use of concentrated (50mg/ml) midazolam if the intramuscular route is used in order to limit the discomfort caused by administration of large volumes of standard concentration midazolam.
Diazepam

Diazepam is of similar efficacy as midazolam in birds following intranasal administration, but has a longer onset time and duration of action. While less commonly used for intranasal administration in birds, diazepam represents a suitable alternative, in cases in which midazolam might not be available. The intramuscular administration of diazepam should be avoided in any patient, due to delayed absorption and muscle irritation. Dosages commonly used in pet birds range from 0.2 - 2 mg/kg, if used as a sole sedative agent. Dosages as high as 10-15 mg/kg have been administered to finches and budgerigars, without significant side effects. The author does not recommend the use of diazepam in small mammals or reptiles.

Butorphanol

Butorphanol frequently combined with midazolam for sedation in birds and small mammals. Besides its analgesic effects (except in reptiles), butorphanol has sedative effects, which are potentiated by midazolam, diazepam. The combined administration of midazolam and butorphanol is recommended in birds and small mammals for which midazolam alone provides only an insufficient level of sedation or which require deeper sedation for certain clinical procedures such as radiographic positioning. Butorphanol can be given in combination with midazolam, drawn into a single syringe and can be given parenterally as well as intranasally. No side effects of intranasal administration of butorphanol at a dose range of 1 - 3 mg/kg are seen in psittacine birds. At the University of Wisconsin-Madison we routinely use butorphanol 1 - 3 mg/kg combined with 2-6 mg/kg of midazolam in pet birds administered intranasal or by intramuscular injection. In rabbits midazolam at 0.5 mg/kg plus butorphanol at 0.25-0.3 mg/kg results in sedation of most patients, suitable for a variety of clinical procedures such as CT scans, or other diagnostic imaging techniques. Butorphanol is not recommended for use in reptiles by the author. Ketamine can be added to this protocol at 5-10 mg/kg, in order to provide deeper sedation, dependent on the patient and scheduled procedure.

Ketamine

Ketamine, a dissociative agent with dose-dependent anesthetic, sedative and analgesic properties, is frequently used in in small mammal and reptiles to induce sedation. However, ketamine is rarely indicated for sedation in avian patients. High dosages of ketamine should be avoided due to the likelihood of prolonged recoveries with this non-reversible drug. Ketamine should not be used alone, and instead administered with an alpha-2-adrenergic agonist (e.g. dexmedetomidine) and/or a benzodiazepine (e.g. midazolam), which allows for reduction of the ketamine dose. These protocols have the benefit of partial reversibility, leading to more rapid recoveries and increased safety, compared to the administration of ketamine alone. Even at lower (5 mg/kg), ketamine can provide additional sedation and analgesia if combined with other anesthetic drugs in reptiles and small mammals.

Alfaxalone

Alfaxalone is a short-acting steroid anesthetic, which is labeled for induction of anesthesia in dogs, cats and rabbits (in the UK) by intravenous administration. However, alfaxalone can be administered by either the intramuscular or subcutaneous route at higher doses. Alfaxalone is rapidly cleared and its metabolism is independent of organ function. Similar to propofol, alfaxalone administration is associated with dose-dependent cardiovascular and respiratory depression in mammals. Recovery from alfaxalone induced anesthesia is dose-dependent, and at high dosages, prolonged recoveries are to be expected. The major advantage of alfaxalone over propofol is that it can be administered intramuscular as well as subcutaneously, in addition to the intravenous route in birds, reptiles and small mammals. However, significant interspecies differences exist. For example, in guinea pigs the intramuscular administration of alfaxalone at 5mg/kg IM resulted in sedation suitable for radiographic positioning and other non-invasive procedures. However, the large injection volumes are a concern if the intramuscular route is used. Therefore, the subcutaneous route should be considered when possible, which has been shown to lead to a rapid onset of sedation in various reptile species. However, in chinchillas the subcutaneous route was found to be ineffective (compared to the intramuscular one) at 5-10 mg/kg. Alfaxalone can be combined with midazolam in reptiles or mammals or butorphanol in small mammals, which results in smaller injection volumes, because the alfaxalone dose can be reduced. In addition, the combination with midazolam or butorphanol often provides more desirable sedation, by reducing muscle twitching, and rough recoveries in some species.

Dexmedetomidine

Alpha-2-adrenergic agonists, such as medetomidine and dexmedetomidine, provide sedation, muscle relaxation and analgesia. Their use in avian sedation has been reported, but the author does not recommend their use in most avian patients. In small mammals dexmedetomidine and medetomidine may be used in healthy animals without underlying cardiovascular disease, since dose-dependent cardiovascular depression is well documented. Alpha-2-adrenergic agonists are commonly used in combination with ketamine, especially in chelonians, for safe, reliable, and reversible sedation but can also be combined with benzodiazepines for procedural sedation in all reptile species. Combining ketamine with medetomidine, or dexmedetomidine, allows reduction of both drug dosages, and reversibility of the alpha-2-adrenergic agonists with atipamezole will lead to faster and more predictable recoveries. Concentrated
formulations of medetomidine (e.g. 20 mg/ml, Zoopharm Inc) are available and are more suitable for large reptiles, such as large tortoises. Alpha-2-adrenergic agonists are fully reversible by using atipamezole, which usually results in rapid recovery from sedation.

**Reversal of Sedation**

Sedation should be reversed (if the protocol used included reversible drugs) in most exotic pet patients, in order to allow for a more rapid return to normal behavior (e.g. food intake, thermoregulation, etc). Only if patients were sedated for treatment of seizures or for application of an e-collar, bandages or splints, sedation should not be reversed. In these cases, birds should be carefully monitored and reversal considered, if the level of sedation is perceived too deep or the duration of sedation is prolonged, and might interfere with physiological behavior, particular food intake. It is important not to discharge sedated patients, as owners do not tend to appreciate to have a partially sedated pet, which might be imbalanced, sleepy and refuses to eat.

**Flumazenil**

Flumazenil is a benzodiazepine antagonist, and is used to reverse the sedative effects of midazolam and diazepam. The injectable form of flumazenil (flumazenil hydrochloride (0.1 mg/ml), Abaxis Pharmaceutical Products, Schaumburg, IL) can be administered intranasally, subcutaneously, intramuscularly or intravenously, without side effects. The recommended dosages range from 0.01 - 0.1 mg/kg. Alternatively, a flumazenil to midazolam ratio of 13:1 has been recommended, but it has been shown that lower doses of flumazenil achieve complete recovery from midazolam induced sedation in birds. The author prefers to administer 0.05 mg/kg initially in most patients. If reversal is deemed unsatisfactory, then the same amount of flumazenil can be administered repeatedly. Recovery from sedation is usually complete within 10-15 minutes in most birds, but can be prolonged in small mammals, in particular if high doses of midazolam were used.
Small mammals: Tips and tricks
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Small mammals, such as rabbits and rodents are becoming increasingly popular as pets. This lecture will provide clinically relevant information on managing common clinical challenges in these species successfully, including abdominal radiograph interpretation, IV catheter placement, sedation protocols, and providing effective nutritional support.

The value of blood biochemistry evaluation in rabbits
Rabbits are frequently presented for evaluation of sudden onset anorexia and lethargy. Possible underlying causes include stress, and underlying disease process or pain. It can be challenging to identify the correct underlying cause in rabbits and this may result in the misdiagnosis of potential life-threatening conditions, such as GI obstructions or liver lobe torsions. Blood biochemistry offers a simple and cost-effective way to rule out important underlying diseases in a short period of time. In most clinics, these tests can be performed in-house and allow the veterinarian to provide the best possible treatment options for rabbit patients.

Blood glucose has been shown to be significantly elevated (> 360 mg/dL) in rabbits with severe painful disease condition, such as GI obstructions, while with other conditions or stress, glucose values remain significantly lower. Running a blood glucose, allows the veterinarian to inexpensively identify cases, which require more diagnostic and therapeutic interventions, and avoid prescribing treatments, which may be detrimental (e.g. syringe feeding or prokinetics in cases of GI obstruction).

Liver lobe torsion is another easily missed underlying disease causing rabbits to present with unspecific signs of anorexia and lethargy. Radiographs cannot reliably diagnose this condition. However, liver enzyme levels measured on commonly used biochemistry profiles are a sensitive method to screen for rabbits with liver lobe torsion. In the authors experience ALT is usually substantially increased in cases with liver lobe torsion. Ultrasound of the liver is used to confirm the suspicion of liver lobe torsion in rabbits.

Other common disorders, which can be investigated by plasma biochemistry include azotemia and electrolyte imbalances in rabbits.

Abdominal radiograph interpretation in rabbits
Performing abdominal radiographs in these patients is routinely performed, but the interpretation of the radiographic finding may be challenging. All abdominal radiographs should be systematically evaluated. The stomach should not be extending beyond the last rib and should not be touching the ventral body wall. Small intestinal loops should never be visible in healthy rabbits. The cecum is located in the ventral mid-caudal abdomen and should have be filled with ingesta intermixed with small gas bubbles. The ascending and descending colon may or may not be visible in healthy rabbits. In rabbits with small intestinal obstruction, the stomach is enlarged and the small intestinal loops are gas filled. The degree of gastric dilatation and small intestinal tympany depends on how long the obstruction has been present. In cases of GI obstruction, the cecum show little to know gas content. Most rabbits with GI obstruction get initially treated with aggressive medical therapy, including intravenous fluids and opioids analgesics. Repeated radiographs are recommended in order to assess, if the obstruction is resolving. This may take > 12 hours, but once hair pellets, the most common cause for small intestinal obstruction in rabbits has moved into the cecum or colon, increased amount of gas should be present in the cecum and the stomach should be smaller in size.

Intravenous catheter placement
Obtaining and maintaining intravenous access is one of the most important steps in the care of critically ill small mammals. Due to the small size of some of these animals, or their limited tolerance for manual restraint, intravenous catheter placement can be challenging or impossible. If intravenous access cannot be obtained, an intraosseous catheter should be considered. In rabbits the author prefers to place intravenous catheters in the marginal ear veins. This technique is simple and well tolerated by rabbits.

Nutritional support
Nutritional support is a critical component of supportive care for any patient, but in particular in herbivorous rodents (e.g. chinchillas, guinea pigs) and rabbits, providing nutritional support is critical in order to avoid development of secondary GI complications (e.g. hypomotility, tympany, dysbacteriosis). Provision of a high fiber diet which can be administered by syringe at sufficient quantities is important for these species. Several commercial products are available, which can be reconstituted with water into a slurry. The author prefers to use 1 ml syringes for syringe feeding rabbits and rodents, since it allows for more reliable oral administration, by limiting the amount of food which drools out of the mouth during feeding. One ml syringes can be placed deeper in the oral cavity and therefore will trigger a swallowing reflex. The author usually recommended to syringe feed 50-80 ml/kg body weight per day of a
high-fiber critical care diet, divided into 3-4 feedings. For example, a rabbit with a body weight of 2 kg, should receive 25-30 ml per feeding.

**Sedation**
Sedation increases the tolerance of patients towards stressful procedures. Sedation is routinely used in human and veterinary medicine and is increasingly considered for small mammal patients as well. Since most small mammals are prey species, they are less tolerant to handling, manual restraint and other stressful or painful procedures. In particular, in small mammals in respiratory distress, sedation should be considered prior to prolonged manual restraint, for example for radiographic positioning. A variety of drugs can be used to induce effective sedation in small mammals. Some of these drugs have sedative, anxiolytic and amnestic properties (e.g. midazolam), while others have mainly analgesic and sedative properties (e.g. butorphanol). The combination of two drugs (e.g. midazolam with butorphanol) usually results in better sedation with less side effects, as compared to using a single drug at a higher dose. Most drugs used for sedation can be reversed, which allows for a more rapid recovery from sedation. In addition, in case of complications, the administration of reversal agents may help improve the outcome. Drugs, which cannot be reversed (e.g. ketamine) should be used at low doses only, in particular in animals with reduced liver and/or kidney function. Oxygen flow-by should always be provided, since most sedation protocols will result in a reduction of respiratory rate.
Fear Free Doesn’t Require a Longer Office Visit
**IF You Know These 10 Tricks**

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Veterinary Medical Center of Fort Mill
Rock Hill, SC

The goal of Fear Free℠ is to improve patient health, welfare, and well-being as well as enhance the client and team experience. For many people change is hard, and one of the reservations many individuals have about implementing Fear Free is that it will take too long for a busy, high-volume practice. My practice is a busy 4 doctor operation that sees on average 80-120 patients/day for medical reasons. We have been able to successfully integrate Fear Free techniques into our 30-minute appointment schedule with doctors maintaining a $200 ACT and generating between $4000-$7000 in revenue per shift. Many veterinary practices have found that Fear Free℠ Certification creates many benefits: it promotes positive practice culture, client loyalty, and revenue generation.

**Tip 1: Understand organizational change**
- Fear Free℠ is change & change is a process
  - All organizational change has an emotional and productive impact on the staff
  - Initially staff may experience excitement, fear, or even denial
  - As the change process is underway resentment or negative emotions are often expressed
    - This coincides with a brief period of decreased productivity
  - As the change process matures staff reengage and the practice experiences higher morale and productivity than before the change
- Use positive peer pressure to encourage everyone along

**Tip 2: Understand human behavior change**
- Emotional intelligence is a balance between the rational and emotional center of the brain
- Human behavior change usually requires an individual to be reacting with their rationale brain
- How do you get an individual to switch from the emotional side (no I do want to change, Fear Free is just not for me) to the rationale side?
  - Tell Story
  - Ask open ended Questions
    - What does an ideal day in veterinary medicine look like to you?
    - What is an ideal patient experience in our practice?
    - What are your thoughts on how we can achieve this together?

**Tip 3: Pre-appointment communication**
Prior to the appointment support staff clearly communicates at least the following 3 criteria:
- Bring in a fresh stool sample
  - Technicians can begin processing in house puppy/kitten fecals before even stepping in the room to get a history
  - Doctor time is not required to collect a stool sample during exam
- Bring pet appropriately dressed
  - Dog on 6 foot leash with buckle collar
  - Cats in carrier that opens from top and front
- Bring the pet hungry!
  - The pet will exponentially be more likely to engage in treats if they are hungry prior to the exam

**Tip 4: Appointment staggering**
If a practice has multiple doctors seeing appointments at the same time consider offsetting the scheduling.
- Dr A will start seeing appointments every 30 minutes beginning at 8 am
- Dr B will start seeing appointments every 30 minutes at 8:15 am

**Tip 5: Stock the exam room appropriately**
- Make sure that all items for common procedures are available in every exam room
- Consistency in placing teams in similar places in the exam room are very helpful for staff in large hospitals.
Tip 6: Perform more procedures in the exam room
- Really objectively watch the amount of time that is wasted the next time a patient is removed from the exam room for a simple blood draw
- Completing simple procedures in the exam room eliminates the time it takes to explain to the owner why the pet is being removed, the time it takes to coax the pet away from the owner, and the time it takes to “find a friend” to hold

Tip 7: Easy to use and reach positive reinforcement in all areas of the hospital
- Peanut or soy butter cups, pretzel rods, meat flavored treats, etc near the scale front door, exam rooms, treatment areas, and animal wards eliminates employees having to travel to retrieve items
- Food treat dispenser that mentally engage patients can be highly distracting which allows procedures to be completed quickly

Tip 8: Disinfect quickly
- Shutting exam rooms down for extended period to allow 10 minutes of disinfectant contact time really hurts the efficiency of the hospital
- Consider disinfectant products that require 606 seconds of contact time or less and get the exam rooms opened up more quickly

Tip 9: Be Pro-active with pre-visit pharmaceuticals
- If a patient is rising above a 3 on the FAS scale, we do not proceed without adding in sedation or rescheduling the procedure once the pre-visit pharmaceutical is on board
- Go to PVP doses:
  - Trazodone (preferred first pick for dogs) 8-10 mg/kg the night before and then 2 hours before appointment
  - Gabapentin (preferred first pick for cats, will combine with trazodone in dogs if needed)
    - Cat: 100 mg/10 pound of cat (can reduce to 50 mg/cat if very petite cat)
    - Dog 20-30 mg/kg the night before and then 2 hours before appointment
- PVP’s are given to all or in house drop off patients and surgical patients

Tip 10: Exam room checkout
- This eliminates clients trying to check out at the front desk while others are checking in
- Consider rear reception work space near the exam rooms to allow clients to check out right in the room.

References
Reversing the Decline in Veterinary Care Utilization: Progress made, challenges remain. AAHA-AVMA white paper 2014
Joseph Pine II, James H. Gilmore. The Experience Economy. 1999
For more information on the Fear Free℠ certification program and resources visit: www.fearfreepets.com
Fear Free- Happy Pets, and People, Healthier Pets and Profits
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It can be intimidating to invest in Fear Free® certification for a significant percentage of your staff. When a practice invests in a new piece of equipment or offers a new service it is easy to track and monitor revenue generation in practice software systems. You may worry that your practice will not see a direct return on investment with Fear Free® certification; furthermore, you may be concerned that incorporating Fear Free® techniques will lead to a decrease in workflow efficiency. Admittedly, revenue generation from Fear Free® certification cannot be demonstrated with simple reports run on invoice line items. It will take a little persistence and patience to reap the full financial benefits of Fear Free® commitment, but the veterinary practices that persevere will reap the ultimate revenue generator: engagement!! Fear Free® carves a new path in veterinary medicine that leads to employee engagement as well as customer engagement.

Employee engagement

One significant predictor of a successful veterinary practice is the engagement level of the staff. A practice that experiences high employee turnover, staff members calling out, gossip, or client complaints regarding service/attitude is not harnessing the power of an engaged staff. Employee engagement has received very little attention in veterinary medicine, and our profession is suffering because of this oversight. Our profession struggles to retain licensed veterinary technicians, many individuals deal with burn out daily, and many experience compassion fatigue. If veterinary practices take a step back and realize that “what gets done is as important as how it gets done,” then we stand a chance to reverse some of those concerning trends.

Millennials are quickly becoming the main portion of the modern workforce. In 2020, millennials will be 50% of the workforce. Veterinary practices need to create a plan of action to connect with this new generation. Simon Sinek’s video on millennials in the workplace provides insight on why millennials struggle with satisfaction in the workplace. This generation expects the workplace to provide options for personal growth, contributing to society (or making an impact), and a sense of community. Interestingly, the latest research from the Harvard positive psychology department shows that it’s not just millennials craving these factors in employment; it’s all of us! We are told from an early age that when you work hard you will become successful, and once you are successful you will be happy. After years of education, training, and commitment it can be a huge disappointment to discover that happiness and fulfillment are not waiting for us with open arms. Positive psychology has discovered that the formula for happiness is back wards. Research demonstrates that if you are happy then success will follow. Organizations that promote positivity and optimism generate 37% more revenue. Interestingly, doctors are 19% more accurate and faster in their diagnosis when the organization promotes positivity and has an engaged staff.

How does a practice cultivate an environment that fosters happiness when our profession is fast paced and dealing with life and death issues multiple times per day? The first step is creating the opportunity for personal development. Providing Fear Free® certification for staff members is a direct signal that a practice is committed to the personal development of its employees. Since additional content will be available with yearly Fear Free® renewal, your staff will continuously benefit from the investment in their education and training. A second way to build engagement is to communicate and provide opportunities for all levels of staff to feel they are making a difference or contributing to society. Vet practices that embrace Fear Free® are very in tune with the role and importance that each staff member has in creating Fear Free® experiences for pets. A doctor will fail to deliver a Fear Free® veterinary visit if the receptionist does not understand or is not empowered to create a relaxed, stress free check in process. Fear Free® helps each employee understand that they have a key role in eliminating fear, anxiety, and stress during the patient visit. Fear Free® also fosters the feeling of community. Staff members will be learning and forming new habits together. Fear Free® professionals also have access to community social media platforms and share the connection of “doing well by doing good” in veterinary medicine.

Customer engagement

What does the modern veterinary customer want in a veterinary practice? It may seem logical that price convenience, and speed are the top three most influential aspects pet owners use to select a veterinarian practice. Millennials are now the largest pet owning population and the modern pet owner uses more than just customer service to select their veterinarian. Modern pet owners are looking for “why” you do what you do, the ability to deliver and experience, and authenticity.

Does your practice lead with “why?” Imagine a pet owner walking into 2 different veterinary clinics. The first clinic states that they practice high quality veterinary medicine with the use of experienced staff members and the latest equipment. In closing, the staff member mentions that the staff members love pets and providing stress free veterinary visits. When the pet owner walks into the second practice the staff member that greets them opens with, “Welcome to our veterinary clinic we are so passionate about delivering Fear Free veterinary visits for you and your pet. We are able to do this by investing in Fear Free certification amongst our staff and utilizing the latest veterinary equipment to provide high quality veterinary care.” The first practice began the conversation with “what”
they do, then “how” they do it, and finally explained “why” they exist. The second practice opened with “why” they exist and later explained “how” and “what” they offer. Fear Free is a very easy way for clients to recognize “why” you do what you do!

The economy is a rapidly changing environment. 20 years ago, the major profit center of a veterinary practice was the pharmacy. When the internet and online pet pharmacies became mainstream, veterinary practices had to shift and find a new revenue center. Veterinary services are now the number one profit center of most veterinary practices. Unfortunately, there is an expiration date on services being profitable enough to sustain a veterinary practice in the future. It will become imperative for practices to create an experience that enhances and protects the human animal bond. Fear Free provides practices the opportunity to create individualized veterinary care that focuses on physical and emotional wellbeing. Clients will delight in selecting bandanas, calming scents, and menu options for their pet’s positive reinforcement.

If you listen to economic futurists or ted talks, then you will likely hear that the next phase of the economic development is authenticity. What is “authenticity?” Simply put, being authentic means staying true to who you are, what you do and who you serve. In an environment in which more human elements matter it creates value and benefits for your followers as well as improving your business.

Authenticity works because…

- It elevates your practice above the competition
- It builds your identity and image into something influential
- It gives substance to your practice, services and products
- It enables people to relate to your practice
- It helps people understand how what you offer is of benefit to them
- It tells people that what you offer is of high quality
- It marks you out as a reliable, trustworthy company
- It encourages engagement and can turn audiences into advocates

The modern consumer will want to see that a veterinary practice consistently delivers what is says it does. By investing in Fear Free℠ certification for all staff and/or Fear Free℠ Practice Certification a practice will be outwardly demonstrating their authenticity and commitment to reducing fear, anxiety, and stress in pets.

**Conclusion**

In today’s world, veterinary practices will be rewarded when they learn to harness the power of engagement. An engaged staff will have low turnover, increased innovation, and increased productivity/revenue generation. An engaged consumer will be loyal to the veterinary practice and very trusting of recommendations and treatment plans.

**References**

Reversing the Decline in Veterinary Care Utilization: Progress made, challenges remain. AAHA-AVMA white paper 2014

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Fear Free Exam Experience
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When you implement Fear Free it is important to take a high-level view of the client and patients exam room experience:

- Physical Well-being Experience
- Emotional Well-being Experience

A key thought in creating an exam experience
“The Experience of being understood, versus interpreted, is so compelling you could charge admission”. – Joseph Pines, Experience Economy Pioneer
Efforts to make your appointments an experience instead of a transaction is not just to increase profits, but also to increase compliance. If the pet parents have positive memorable experiences in your practice, then they are more likely to return for rechecks and wellness visits.

Economic transitions
With the advancement of technology, increasing competition, and the increasing expectations of consumers, services today are starting to look like commodities. Products can be placed on a continuum from undifferentiated (referred to as commodities) to highly differentiated. Just as service markets build on goods markets which in turn build on commodity markets, so transformation and experience markets build on these newly commoditized services.

The classification for each stage in the evolution of products is:

- A commodity business charges for undifferentiated products.
- A goods business charges for distinctive, tangible things.
- A service business charges for the activities you perform.
- An experience business charges for the feeling customers get by engaging it.
- A transformation business charges for the benefit customers (or "guests") receive by spending time there.

Most brick and mortar veterinary practices rely on services as their top revenue generator. But once services (lab testing, spay neuter surgeries, etc) become a commodity, then we must at taking out practices into the experience economy.

How to create an exam experience
1. Ease of Communication prior to appt: text and email reminders, exchange of information via email. If clients know their wellness options in advance, then they can make more informed decisions ahead of time.
2. Assessing FAS prior to the appt: Am Assigned staff member to review the patient’s FAS (Fear, Anxiety, Stress) level prior to appt. If 1-3 discuss nutraceutical prior to visit. If 3 or higher then discussion PVP (pre-visit pharmaceutical)
3. Layering pheromones will enhance your patient’s response
   a. Wall plug ins
   b. Direct patient application (spray feline pheromone on towel, place right in front of carrier, spray bandanas with canine pheromone- can be reused or given as memorable gift)
      i. Spray fabrics 15 minutes before patient interaction allows alcohol smell to dissipate
   c. Wardrobe changes
      i. Consider colors and if coats increase patient fear in your practice
      ii. Spray feline pheromone from neck to waist (7 sprays) & canine pheromone from waist to ankle (7 sprays)
         1. Reapply every 4-5 hours
         2. Don’t forget the stethoscope: feline pheromone wipe can be used in between patients
4. Calming music
   a. Could simply be playing from phone in exam room, computer in exam room, pet specific music playing device
   b. Types of music: studies show soft playing instrumental music, audio books, or reggae music can reduce heat rate levels
5. Examine patient where they choose
   a. Sometimes that will be on the floor, in the client’s lap, or up on the exam table
   b. Be mindful of nonslip footing for every type of footing your patient will experience in the clinic
      i. Footing should be nonslip as well as not cold to the touch (remember our patients are always walking barefoot!!)
6. Try to reduce separation between patient and client
   a. Work with your support staff team to stock the exam rooms and increasing training so that injections, blood draws, and blood pressure readings can all be performed in the exam room with the client present.
7. Share the knowledge
   a. Talk, talk, talk, during that exam
   b. Talk about your medical findings as well as your body language observations of the pet
   c. Looks for ways to commend the client for their current efforts, this will make them much more receptive to your recommendations

8. High Reward Treats
   a. This can be pretzel rods, peanut butter, soy butter, marshmallows, dog treats, cat treats, frozen broth, tuna, etc
   b. It can also be balls, squeakers, and feather toys
   c. Anything that is a positive reinforcement tool that help the pet forget that they are in a medical facility will translate into an experience for both pet and client

When trying to create a memorable experience for the pet and pet owner remember that the client is looking for validation from the veterinary professional that they are a loving, caring pet parent. Truthfully, there are times that we can instantly see that the pet parent could be doing more. But, in order for us to successfully deliver medical recommendations that the client will consider we must first ensure that they feel validated. Look for anything to initially compliment. It could be a lovely new collar, nicely smelling coat from a fresh bath, or even their effort to bring the pet in that day.

By validating the pet parent and setting the stage for a positive patient exam you will ensure that you and your practice have entered the experience economy!

References
Joseph Pine II, James H. Gilmore. The Experience Economy. 1999
Elisabeth Kubler-Ross. On Death and Dying. 1969
There are approximately 10 fold more bacteria than cells in the body. Thus, mammals and not the microbiota could be considered as the parasites! Regardless, it is clear that both provide benefit to each other. Mammals provide a nutrient-rich environment for the microbiota to live and, in turn, intestinal bacteria provide a number of benefits to the host such as synthesis of nutrients (e.g. SCFAs) and vitamins (B vitamins), regulation of the intestinal epithelial barrier, promotion of digestion, crosstalk with the host immune system, influence of host-cell proliferation, vascularization, and neurologic signaling, and protection against pathogens. Many factors influence the microbiome in humans including genetic background, infections and its associated immune response, environmental exposures, and most importantly, diet, sex and age, previous xenobiotic administration, and intestinal biopsy sample collection. Perhaps it comes as no surprise, given the ratio of bacteria to host cells, that “dysbiosis” or alteration of the native microbiota is associated with a wide variety of diseases. The onset of microbial shifts and reduction in biodiversity are well-described in canine and feline enteropathies. For this reason, manipulation of intestinal bacteria with probiotic administration represents a potential therapeutic target for a variety of diseases. Probiotics are live microorganisms, which when administered in appropriate concentrations, are intended to colonize and interact with the host intestinal epithelium and immune system and confer a physiological health benefit to the recipient (e.g. anti-inflammatory activity, antagonize enteric pathogens, etc). Probiotics, therefore, must survive not only processing and storage in vitro but also gastric and bile acid degradation in vivo. Most probiotics contain lactic acid-producing bacteria (i.e. *Bifidobacterium*, *Lactobacillus*, *Enterococcus spp*). Lactic acid-producing bacteria are normal inhabitants of the colon. They maintain anti-microbial properties as a result of secretion of bioactive compounds and induction of changes in environmental pH that may be unfavorable to certain pathogens. These bacteria tend to be decreased in inflammatory bowel disease. Although a variety of veterinary probiotics containing lactic-acid producing bacteria are now available, many animals are still treated with probiotics intended for human use as these are more widely available. Thus, practitioners should have a good understanding of the probiotics that are available, both those intended for human and animal use. Probiotic strains derived from dogs can adhere to the human and canine GI tract similarly. Moreover, the use of probiotics intended for humans can transit the canine and feline GI tract. Thus, despite differences in resident bacteria among species (e.g. cats have more anaerobic bacteria in their intestine compared to dogs and humans), probiotics do not necessarily need to be derived from the species being treated. However, they must be shown to survive GI transport and colonize the intestinal tract of the species of interest. Practitioners should also be aware of dosing and storage guidelines for each probiotic as they vary greatly between products. Practitioners should make clients aware that probiotics are classified as dietary supplements, not pharmaceuticals, and therefore are not regulated by the FDA. Proof of efficacy is not required. Several studies had demonstrated that a substantial number of probiotics on the market for human or animal use may not contain the claimed organism, may contain additional species not listed on the label, and/or may contain markedly lower concentrations than stated on the label. Thus, practitioners and clients should scrutinize probiotic products and only choose probiotics produced from companies with good quality control measures.

**Probiotics in gastrointestinal diseases**

Evaluation of the effect of probiotics as adjunctive therapies for the treatment of animals with naturally occurring gastrointestinal diseases is still in its infancy. Most work to date has been focused on the use of probiotics for the treatment of acute idiopathic diarrhea showing the most promise. For example, administration of the probiotic *Enterococcus faecium* SF68 to shelter cats resulted in a significantly lower percentage of cats with diarrhea for ≥2 days compared to cats that received placebo. Administration of the canine-derived probiotic containing *Bifidobacterium animalis* AHC7 to dogs with acute idiopathic diarrhea resulted in significantly reduced time to resolution of diarrhea and reduced percentage of dogs administered metronidazole compared to dogs receiving placebo. Similar results were found in a study investigating the effects of a probiotic cocktail orally administered to dogs with acute vomiting and diarrhea. In this study, dogs who received the probiotic cocktail had a quicker resolution of diarrhea, but not vomiting, compared to dogs who received placebo. Probiotic administration has also been demonstrated to decrease the incidence of diarrhea in dogs and cats entering animal shelters. Probiotic administration may also lessen gastrointestinal signs induced by antibiotic administration. The beneficial effects of probiotics and microbial therapy for infectious diarrhea (e.g. *C. diff*-associated diarrhea) has been well established in people. However, the beneficial effects of probiotics for infectious diarrhea in dogs and cats have been underexplored. To the author’s knowledge, only one published report has described the use of probiotics for infectious diarrhea in dogs wherein treatment of dogs with subclinical chronic giardiasis with the probiotic *Enterococcus faecium* SF68 did not reduce Giardial cyst or fecal antigen shedding compared to dogs receiving placebo. This lack of efficacy may be related to timing of administration or type or concentration of probiotic bacteria administered. In unpublished work supported by the Winn Feline...
Foundation, using an in vitro model of infection, our lab has demonstrated that Enterococci decreased *Trichomonas foetus* (TF) cytopathogenicity but only when the Enterococci were administered prior to TF colonization of the intestine. Moreover, *Enterococcus hirae* were more effective than *Enterococcus faecium* in preventing TF intestinal adhesion.

It stands to reason the probiotics would also be helpful in the treatment of intestinal disorders where dysbiosis is thought to play a major role (i.e. antibiotic-induced diarrhea, antibiotic-responsive diarrhea, inflammatory bowel disease (IBD)). Unfortunately, at the time of this writing, there are only a handful of studies evaluating probiotics or synbiotics for the adjunctive treatment of chronic enteropathy in dogs or cats. These studies suggest that probiotics may play a beneficial role as adjunctive therapy in a subset of dogs and cats with chronic enteropathy. However, the benefit of probiotic therapy for dogs and cats with food responsive diarrhea (FRD) has not been demonstrated likely because dogs and cats with FRD often respond quickly to diet alone making it difficult to evaluate for a potential beneficial effect of the probiotic. A small pilot study demonstrated a possible benefit to adjunctive probiotic therapy in cats with chronic idiopathic constipation.

**Cautions with use**

Probiotics are considered supplements and therefore are not subject to regulatory oversight by the FDA. Demonstration of effectiveness is not mandated. Generally speaking, most probiotics are safe and are associated with few to no side effects. However, probiotics might be inappropriately labeled, contain organisms at the incorrect concentration, or contain organisms that might be pathogenic or have not been demonstrated to possess probiotic properties. Clients should be notified of these concerns as well as the lack of efficacy data in veterinary medicine. Some probiotics intended for human use are manufactured in enteric-coated capsules to prevent premature acid degradation and to assist in delivery of bacteria to the distal intestine prior to activation. A study performed in cats with CKD demonstrated that opening an enteric-coated synbiotic capsule and sprinkling its contents on food or delivering as a slurry resulted in ineffectiveness of the synbiotic. Thus, this form of delivery, unless otherwise indicated by the manufacturer, is not recommended.

**Conclusions**

The culmination of this early work suggests that probiotics might have a place in the adjunctive treatment of gastrointestinal disease in dogs and cats. However, much work is needed to determine which diseases will respond and which type and how much of the probiotic is needed to induce such a favorable response. At this time, the presenter does use probiotics for the treatment of acute idiopathic diarrhea, antibiotic-induced diarrhea, and as an adjunctive treatment in dogs and cats with chronic enteropathies other than food-responsive disease.

**Selected reading**

What’s on the horizon for detecting food or steroid responsive enteropathy? What factors increase the risk of heartworm positivity in cats? What long-term consequences can be anticipated in declawed cats? What is the “go to” anti-thrombotic drug in cats at risk for thromboembolic disease? Some of the answers may surprise you! In this 50 minute interactive seminar, we will discuss the top studies selected by specialists in a variety of fields including emergency/critical care, surgery, neurology, dermatology, and general practice. In each published study, there will be at least one finding that can be applied immediately to clinical practice.
Causes of gastrointestinal erosive and ulcerative disease are numerous, but the end result is a compromised mucosal barrier and, often, continued tissue injury beyond the primary insult as a result of exposure of the compromised mucosa to gastric acid, bile acids and pepsin. Left untreated, this ongoing injury can lead to increased morbidity and poor quality of life. Gastroprotectants are widely used for the prevention and treatment of erosive and ulcerative gastrointestinal diseases. Gastroprotectants include acid suppressants (e.g., histamine-2 receptor antagonists, proton pump inhibitors), coating agents (e.g., barium, sucralfate), and prostaglandin analogs (e.g., misoprostol). More recent studies suggest that we have been using gastroprotectants ineffectively in dogs and cats. This presentation will review the available veterinary literature, provide insights from the use of these drugs in human medicine, and, when available, introduce newer gastroprotectant dosing recommendations and indications which may improve the outcome of dogs and cats with and at risk for erosive and ulcerative disease.

Acid suppressants

Gastric acid secretion is triggered by hormonal and neural stimulation of parietal cells, the acid producing cells of the stomach. Acid suppressant drugs, which take aim at these physiological targets on the parietal cell surface, have largely supplanted antacids (acid-neutralizing drugs) as the drugs of choice for upper gastrointestinal erosion and ulceration. Histamine-2 receptor antagonists (H2RAs) are competitive inhibitors for the interaction of histamine with its receptor but differ in potency with famotidine being the only H2RA shown to significantly increase gastric pH compared to placebo in dogs and cats. Famotidine can be administered with food and is effective with minutes to hours after administration. In contrast, proton pump inhibitors (PPIs) form irreversible bonds with the acid-producing pump of the stomach and take longer to reach peak effect. However, recent studies suggest that omeprazole is as effective as famotidine by day 1 of administration and more effective by day 4. Moreover, prolonged daily administration of famotidine results in tolerance to the medication in dogs and cats. This has been demonstrated to occur by day 11 of treatment but may start to occur as early as day 3 of administration. Many formulations and types (e.g., omeprazole, pantoprazole, esomeprazole) of PPIs are available. Omeprazole is the most extensively studied in veterinary medicine and has been shown to be effective when given as a liquid, paste, as well as tablets. Despite the presence of an enteric coating, omeprazole is still effective when split and administered to cats orally. To the author’s knowledge, there are no comparative studies evaluating the effect of differing types of PPIs on gastric pH when given orally to dogs and cats. However, a recent pilot study evaluating esomeprazole in dogs suggest that it may have a superior effect on gastric pH. When severe erosive or ulcerative disease is documented or suspected, a PPI should be administered at a dose of 1 mg/kg twice daily.

Guidelines for acid suppressant prophylaxis in dogs and cats to reduce risk for GUE have not been created. Use of guidelines for the treatment of GUE in humans for the treatment of GUE in dogs and cats is not possible. However, some recommendations are sensible and should be adopted until appropriate guidelines have been established for veterinary patients. Acid suppressant prophylaxis should be discouraged unless patients have multiple risk factors for the development of clinically important GUE or have a previous history of GI bleeding. Risk factors may include critical illness, mechanical ventilation, coagulopathy, acute kidney injury, liver failure, shock, severe head or spinal cord injury, acute lung injury, or major surgery lasting more than 4 hours. Patients being treated with warfarin that are concurrently receiving NSAIDS or anti-platelet drugs may also benefit from acid suppressant therapy to reduce the risk of warfarin-induced GI bleeding.

Coating agents

Coating agents include barium and sucralfate. Barium has mucosal protecting effects and hemostatic properties. Barium enemas are effective for treatment of lower GI bleeding in people. To the author’s knowledge, no published studies have evaluated the efficacy of barium in the treatment of dogs and cats with GUE, however, studies in the treatment of rectal bleeding in people and anecdotal information in dogs and cats suggest that it may be an effective adjunct treatment of GUE. The dose recommended for mucosal hemostasis can be occasionally challenging to administer especially in a patient with a history of inappetance or vomiting. Although barium is inert, aspiration of barium with gastric fluid contents can be fatal. Discontinue barium for at least 24 hours prior to gastrointestinal endoscopy and do not use in animals where GI perforation is suspected. Sucralfate, a polyaluminum sucrose sulfate, forms a protective layer on the proximal GI mucosa. It is only effective when administered as a liquid or slurry. It forms a gel-like substance to cover tissue injury in the esophagus and proximal GI tract. Sucralfate may also stimulate the production of prostaglandins, which may help bolster the GI mucosal barrier through increased mucus and bicarbonate production and secretion. Sucralfate may be more effective in the adjunctive treatment of duodenal ulcers compared to gastric ulcers. It significantly reduces overt upper GI bleeding in humans, and likely, dogs and cats. Sucralfate may also be helpful for oral lesions when used as a mouth wash. Sucralfate is associated with very few adverse effects aside from constipation; however, it should not be used as a phosphorous...
binder in cats with CKD. Sucralfate alters gastric pH and, therefore, may interfere with the metabolism of drugs that are dependent on an acidic gastric pH such as acid suppressants. It also interferes with drugs affected by the aluminum component of sucralfate (e.g. tetracyclines, ciprofloxacin). Therefore, these drugs should be administered at least two hours before or after sucralfate administration.

**Prostaglandin analogues**

The most commonly used prostaglandin agonist in veterinary medicine is misoprostol, a PGE1 analog. By simulating endogenous eicosanoids, misoprostol increases mucosal blood flow and epithelial repair and stimulates mucus and bicarbonate secretion. Despite its mechanism of action, misoprostol is only effective for NSAID-induced injury and has no effect with steroid-associated ulceration. Its use is discouraged with other cause of GI erosion and ulceration because its use in these conditions likely cause more harm than provides benefit. As mentioned for PPIs, prophylactic use of misoprostol for NSAID-induced GUE is effective but not recommended unless other risk factors are identified.

**Suggested reading**


Degenerative joint disease (DJD) is a common but often unrecognized condition in cats, causing chronic pain and inability to perform normal feline behaviors. If untreated, patient welfare is impacted, often secondary to chronic and neuropathic pain, emotional pain, and changes in social relationships. Awareness of DJD, its incidence, affected joints, and how to recognize and develop a comprehensive treatment plan leads to successful outcomes.

Prevalence of feline DJD
In a random study of cats in different age groups, 91% of cats had radiographic evidence of DJD, occurring as early as 6 months of age, and with equal frequency in all age groups. Despite radiographic evidence in young cats, signs may not be evident until disease worsens with age. There is a dramatic increase in prevalence of signs and burden by 10 years of age.

Etiology
The cause in the majority of cases of feline DJD is unknown. However, injury and genetic conditions can predispose, the latter including hip dysplasia, patellar luxation, and osteochondrodysplasia. Hip dysplasia and patellar luxation can occur genetically in both non-purebred and purebred cats. 18-24.9% of Maine Coon cats have hip dysplasia, with 56% having bilateral disease. Patellar luxation is more common in Abyssinian and Devon Rex breeds. Osteochondrodysplasia in Scottish Folds is a dominantly inherited condition with malformation of limbs and progressive joint destruction. Another suggested role in feline DJD is immune system dysfunction.

Obesity impacts joints in two different ways, with excess weight increasing the burden on joints and increased adipokines and obesity-related inflammation leading to breakdown of articular cartilage. Concurrent disease is common in older cats, and DJD often occurs with, but unrelated to, other chronic conditions. On the other hand, chronic kidney disease (CKD) and DJD occur concurrently in cats of all age groups - between 6 months and 20 years - with 68.8% of cats with DJD having concurrent CKD as compared to 50% of cats with CKD alone.

Joints affected by DJD
Multiple joints are often affected with feline DJD. Both appendages and/or spine can be impacted. Spinal or axial DJD is more frequently found between thoracic vertebrae T7-T10, but the lumbar vertebrae are affected more severely. Axial DJD increases with age. The most commonly affected appendicular joints are the hips, elbows, knees, and hocks. As opposed to axial DJD, appendicular occurs equally through the ages. Axial disease can occur without appendicular disease, but spondylosis commonly coexists with osteoarthritis in older cats.

Is it DJD or Osteoarthritis?
DJD is an over-arching term that includes osteoarthritis as well as trauma, inflammation, and other types of degeneration of cartilaginous joints, including spondylosis of the intervertebral joints. Whereas DJD includes both axial and appendicular disease, osteoarthritis is defined as a non-inflammatory disease of appendicular joints. Although osteoarthritis and DJD may be used interchangeably in journal articles, the presenter chooses to use the term DJD due to the common occurrence of axial disease in cats.

Diagnosis
The challenge to diagnose Feline DJD is difficult because of the cat’s tendency to hide pain as a protective mechanism. Cat owners think their cats are slowing down or “just getting old”. As opposed to the dog, most cats with DJD don’t limp because the disease usually impacts the same joints bilaterally. Changes in behavior are the most common signs of DJD, but these also occur with other physical pain, either acute or chronic, non-painful illness, as well as with emotional pain or distress. Also complicating recognition is the waxing and waning of clinical signs of DJD. Gait analysis in the practice is also challenging in feline patients. Although many cats have radiographic evidence of DJD, radiographic signs do not equate with pain. Additionally, painful DJD can occur prior to obvious radiographic changes. History: Owner input is critical

Studies indicate that clients often recognize the pain of DJD in their own pets more accurately than veterinarians because they know their cat’s normal behaviors and changes to them. However, clients frequently think the changes are associated with “old age” rather than pain. History should include questions about changes in that individual cat’s behavior, including jumping, the height of the
jump (e.g., does the cat still jump up to favored high locations), hesitation to jump up or down, and changes in climbing up or down steps. Other signs may include decreased grooming, appetite, increased or decreased vocalization, house soiling, changes in interactions with other pets or people, and aggression. A cat may present with one or multiple changes in behaviors. The Feline Musculoskeletal Pain Index is a good questionnaire to use.

Interestingly, a study demonstrated that cat owners placed more importance on non-physical outcomes (60%) for quality of life (e.g., grooming and comfort during resting), in contrast to the hypothesis that physical activity (mobility) would be more significant to owners.

**Examination**

Prior to handling, observe the cat from a distance, assessing for stiffness and muscle atrophy over back and limbs. Assessing a cat’s gait in he practice is challenging, but observing the cat’s mobility in the exam room or upon returning to the carrier at the end of the appointment can be helpful. Video clips of the cat’s mobility at home are an ideal means to evaluate mobility and gait as owners often do not recognize the gradual changes. Ask owners to take short video clips with smart phones and send electronically to provide easy access and ability to link to electronic records. Specifically request short videos of a cat jumping up and down from the bed or other favored piece of furniture and/or climbing up and down stairs. Comparison with previous examinations and videos can be very helpful.

Hands-on examination includes palpation of back and limbs to identify painful axial and appendicular DJD respectively. Spinal pain is most commonly located over the lumbar and lumbosacral regions. Palpation of thickening of the elbow or knee joints is not uncommon with DJD. Other signs are crepitus, effusion within the joint capsule, and discomfort with or decreased range of motion. Watching the gait following palpation is also helpful and can be done as the cat goes back to the carrier. One study indicated that joint palpation failed to differentiate between cats with DJD and those without, and that gait was most reliable to diagnose, but more studies are needed.

**Other diagnostic modalities**

Research studies use other criteria to diagnose, some of which may be used within the home, including pressure mats, accelerometers, and paw withdrawal thresholds. Pressure mat use determines changes in weight distribution from an affected limb, which has been noted in cats with DJD. Accelerometers identified changes in activity pre-, during, and post-treatment, demonstrating impaired mobility prior to treatment and treatment efficacy.

Radiographs are important to confirm suspicion of DJD and to rule out neoplasia, fractures, and other abnormalities. There is correlation between cartilage damage and the presence of osteophytes and joint-associated mineralization. However, cats commonly have DJD without radiographic evidence of disease, occurring in the knees (71% of knee joints affected with DJD appeared normal) hips (57%), elbows (57%), and tarsal joints (46%).

**Treatment**

Even when diagnosed, cats often receive inadequate treatment due to veterinary concerns about adverse drug effects in this species and owner difficulty to administer medication. DJD impacts the cat’s quality of life and the relationship that owners have with their cats, and requires adequate treatment with both owner input and follow-up appointments. Multi-modal therapy and ensuring an environment that allows cats to perform their normal behaviors is essential to pain management and patient welfare.

**Multi-modal management**

Treatment includes the need for both medical and environmental modifications to allow the cat to perform its normal behaviors and maintain comfort. Multi-modal medication targets multiple sites along the pain pathways, and potentially reduces the doses of each drug, subsequently reducing the potential for adverse effects.

**Pharmacologic treatment**

NSAIDs: Non-steroidal anti-inflammatory drugs (NSAIDs) are the mainstay of pharmacologic treatment to date for DJD in cats as well as other species. Administering NSAID’s that are specifically labeled for use in cats is important. However, none of the approved NSAIDs for cats are approved for long-term use within the United States. Meloxicam is however approved for long-term use in cats in Europe, Canada, and several other countries. Although veterinarians are often concerned about NSAID use in cats with concurrent chronic kidney disease (CKD), Meloxicam has also been used effectively in studies. and Robenacoxib in 1 study in cats with concurrent DJD and CKD without negative ramifications. Patients in the studies were cats with stable IRIS stage 1, 2, and 3 chronic kidney disease, normovolemic, and without GI signs. If meloxicam or robenacoxib are used long-term in the US, it is recommended that owners sign a waiver. Dosing should be based on lean body weight. Owners should be warned to stop medication and call the veterinary practice if the cat is not eating, is vomiting, or any other changes. The patient should be reassessed for comfort as well as for diagnostic monitoring. The author does taper meloxicam’s dose and frequency, with cats often comfortable with low dose NSAIDs given every other or every third day.

**Central activity**
The chronic pain of DJD is associated with changes in the central nervous system related to central sensitization. Approximately 25% of cats with DJD did not respond to NSAID therapy alone because of the neuropathic pain. Gabapentin is used for chronic and neuropathic pain without causing serious side effects. It is easy to administer, and it is a great drug to use in conjunction with other treatments, such as NSAIDs. Tramadol also decreases central desensitization, increasing mobility and weight bearing at 3mg/kg twice daily. However, Tramadol is bitter and there is also risk of potential human abuse.

**NV-O2, franzemetab**

NV-02, a feline anti-nerve growth factor antibody that appears to be a safe, long-term analgesic in cats with degenerative joint disease-related pain, but additional studies are indicated.

**Treatment efficacy**

Many cats appear to do well with treatments, but the placebo effect is very high, with owners noting improvement in both placebo and treatment groups. Also of note is that the placebo group often had a high rate of adverse events reported. In one study, differentiating between placebo and treated cats was only recognized once treatment was withdrawn. Ideal studies are double blinded or have other monitoring such as activity monitors, treadmills or pressure mats.

**Non-pharmacological treatment**

Weight optimization and acupuncture are excellent non-pharmacological treatments that can be used as a component of multi-modal therapy. The home environment should also be modified to allow easy access to favored places. Pet steps or ramps can provide easy access to preferred resting area for cats with DJD. Providing food, water, and litter in easily accessible areas is also critical for competition for these resources improves feline welfare.

**References**


Feline-friendly handling is essential to prevent patient stressors that lead to negative emotions such as fear, and pain, and result in behavior responses that are considered difficult. Understanding the cat is the foundation of these handling principles and techniques as cats are like no other domestic animal. A question often asked is how to handle the “difficult cat”. The better questions to ask are “Why is that cat difficult?” and “How do we prevent this?” It is the multiple stressors associated with the veterinary experience that lead to the cat’s attempts to protect itself. The stressors cause emotions such as fear and pain which subsequently lead to the behaviors that we consider difficult. Our handling goals must be to prevent feline stressors, dramatically reducing the number and severity of “difficult cats” and increasing the safety and satisfaction of the veterinary team, the client, and the cat. Feline-friendly handling also addresses cats that continue to display fear and other negative emotions despite our best efforts.

Feline-friendly handling can be incorporated successfully into any type of practice, reducing feline stressors and the associated negative emotions and behaviors that team members often consider difficult. The final outcome is more relaxed cats, happier clients, increased job satisfaction and safety.

Increasing human safety measures
To improve our safety, we must also work to make the cat feel safe, reducing their stressors and the associated behavioral response. Safe territory is critical to the cat’s survival and to protect itself from dangers. Territory provides the cat with a sense of control, security in familiarity and predictability, and enhances the cat’s ability to cope. Territory for the cat during veterinary visits means a “safe place” or a place for the cat to feel hidden, enhancing the cat’s ability to cope in the veterinary practice.

Sense of control
Cats need to have a sense of control and familiarity to feel safe in the environment. Teaching owners to train their cats to the carrier provides sense of control and positive experiences with the carrier. Ask owners to bring the cat’s familiar and favorite bedding, toys, and food to veterinary visits.

The cat feels safer with the fewest people to handle the cat and with minimal restraint. During appointments, allow the cat to choose to remain within the carrier and to remain in the position it prefers. Standing or approaching the cat from the front is a potential threat. Instead, calmly approach the cat from the side or behind. Keeping one hand in contact with their body is much less threatening than repeatedly placing your hand on and off the cat, allowing the cat to anticipate touch. Allowing the cat to remain in the position it has chosen also prevents fear and other negative emotions. Gently place fingers between legs to control them rather than tightly holding or stretching the cat.

A place to hide
Hiding is an important coping strategy for a cat that feels threatened, which is common in an unfamiliar environment. Hiding means that the cat does not see us. Allowing it to feel hidden while we can still work with it facilitates examination, diagnostics, and treatments. A preferred option is to allow the cat to remain in the bottom half of the carrier if the cat does not come out on its own. A towel may also be used to cover the carrier so that the cat does not need to see the unfamiliar. Other hiding options are a cat bed with high sides or an igloo shape, or a towel surrounding the cat, which can be loose in an attempt to hide the cat, or placed more securely to prevent struggling.

Where to touch and where not to touch
Cats prefer to be touched by humans over the facial glands, the same sites that cats allogroom (groom each other). These scent glands are peri-oral, under the chin and around the cheeks, as well as over the temporal area between the eyes and ears. Massaging, petting, or gently rubbing these sites from the side or behind is more respectful for the cat, reducing fear and keeping the patient calmer. Massaging over these areas should replace “scruffing”, as the latter does not give the cat a sense of control and can lead to patient fear and subsequent aggression.

Cats do not like to be petted or patted over the abdomen and this area should be avoided except for necessary examination. Even if cats like when an owner pats them over the rump, patting this area should also be avoided during veterinary visits.
Emotions and the emotional response to stressors

Stressors trigger emotional responses in the brain that mediate functions that contribute to the survival and well-being of the individual. Both positive and negative emotions occur in cats, although we primarily see negative emotions in practice—fear, anxiety, frustration, and pain. More than one negative emotional system can be triggered at the same time, such as pain and anxiety, with the painful cat anxious that handling will aggravate the pain. Prevent the cat’s negative emotions from facilitating successful handling.
Fear/anxiety: Fear is a normal emotional response to what an individual considers a threat and commonly occurs in the veterinary practice. Anxiety is the anticipation of a threat, such as a cat anticipating that handling will cause pain. Fear and anxiety are part of the same emotional system.  

Pain is both a sensory and an emotional response, impacting not only physical function but also the emotional welfare of the patient – the cat’s ability to perform its normal behaviors and to cope with everyday life. Anxiety or fear can exacerbate pain, further impairing an individual’s ability to cope. In addition to analgesia, it is important to minimize stressors both at home and in in the practice.

Frustration is the inability to access safety or receiving less or no reward than anticipated. Examples of frustration caused by an inability to access safety are tight restrained and removing a cat from its cage against its will. Frustration in these situations can lead to severe aggression in an attempt to protect itself.

The behavioral response – understanding feline communication to recognize their stressors

The primary goal of feline communication is to prevent physical altercations with other cats. When we can recognize feline communications, we can better develop a handling plan. Cats communicate their emotions through scent, pheromones, visual displays, vocalizations, and touch. Although humans cannot detect the cat’s chemical communication, we can learn to recognize the meaning of vocalizing, body posturing, and facial expressions. Being able to “read” the cat and knowing how to work with the cat prevents further arousal and human injury. Body postures such as hiding, crouched, or head lower than rest of body, facial expressions such as ears back or rotated to the side, dilated pupils, and whiskers splayed and inhibition of normal behaviors (inappetance, failure to groom) are communications that help us recognize emotional triggers.

Customized handling techniques for different cats

Each cat is an individual

How a cat responds is based on multiple factors, including its genetics, the sociability of its parents to humans, and its own early experiences, especially during the sensitive period of development (~2-7 weeks of age).

Previous veterinary experiences will also impact the cat’s emotions and behaviors during future visits. Cats have long-term memory and those that have had negative experiences surrounding the veterinary experience will remember and be more reactive early on, likely hissing and aggressive. It is important to recognize that how we handle a patient may have long-term ramifications during future visits, leading to behaviors to protect self that we may consider undesirable.

The fearful/anxious cat

As solitary hunters that need to protect themselves at all times, cats have developed a heightened fear response. The three major responses that may be exhibited in response to a potential threat are fleeing (avoidance), freezing (inhibition), and aggression (repulsion). Fear responses can escalate quickly based on our behavior.

Handling the cat that ‘freezes’ (inhibition): This fear response might seem easy to work with, but it is important to recognize that cats that “freeze” are fearful, and that the response to fear can quickly change to aggression based on our behavior. If we allow the cat to remain in a hiding place such as the bottom half of the carrier or high-sided cat bed, the cat will likely remain quiet and hidden throughout the exam, and is more amenable to handling. It is even more likely to relax.

Handling the cat that ‘flees’: Chasing a cat that flees will exacerbate fear and greatly increase the potential for self-protective aggression. Instead, give the cat time to calm down and the option to return to the carrier or another hiding area. Decreasing arousal can take time, and it is best to keep the cat in a quiet, dark or soft-lit room, with several hiding options. Educate owners and provide options to “drop off” or reschedule the appointment. These cats often require an anxiolytic for upcoming visits.

Handling the aggressive cat: The cat is aggressive because it is frightened and attempting to protect self. It is not a “bad” or ‘evil’ cat! Sedation or anesthesia with analgesia prevents potential injury and exacerbated fear and anxiety at future visits. Anxiolytics should be recommended for future appointments.

The painful cat

Pain often underlies fear of being handled and possible aggression. Many senior cats have degenerative joint disease and are painful although owners often do not recognize the signs of slowing down and decreased height of jumps. Analgesia should be given to painful and potentially painful cats. The AAHA/AAFP Pain Management Guidelines 2007 and 2015 provide information on unrecognized painful feline conditions, feline degenerative joint disease, and multi-modal therapy.

Handling cats with frustration

frustration and associated aggression by allowing the cat to feel safe through respectful handling, providing a place to hide within a cage, and removing the cat from the cage along with its hiding place. As frustration also occurs when a cat is not given attention or food when anticipated, consistent times for feeding, play, and human attention if desired helps prevent this negative emotion.
References
Intercat conflict is a common cause of poor welfare, negatively impacting physical and mental health. Whether cats are actively fighting or signs of conflict are subtle and passive, many cats suffer from distress in multiple cat households. A cat blocking another cat from a litter box or other such stressors can result in behaviors that owners consider undesirable, such as withdrawal, scratching furniture, house soiling and/or urine spraying. These behaviors are signs of distress. Further distress may occur with owner misunderstanding, punishment, relinquishment and perhaps euthanasia. Veterinary professionals have a tremendous opportunity and ability to prevent, detect, and even manage less complex cases of intercat conflict to enhance human-cat relationships and feline welfare.

**Why is this important in all veterinary practices?**

Cats continue to be the most popular pet in the United States and other developed countries, and most cats live in multiple cat households. Approximately one-third of cats live in homes with 2 cats, and an additional 24% lives in homes with 3 or more cats. 64% of cat owners think their cats need other cat companions, and 53% enjoy having multiple pets.1 Sadly, intercat conflict underlies 25-30% of behavior problems and 54% of newly adopted cats are relinquished within 2 weeks of adoption.2-

Intercat conflict usually results in poor welfare whether there is active fighting or the more frequent subtle and passive behaviors that occur in multiple cat households. Our veterinary and technician oaths emphasize our commitment to animal welfare. The AVMA defines welfare as “how an animal is coping with the conditions in which it lives. An animal is in a good state of welfare if (as indicated by scientific evidence) it is healthy, comfortable, well nourished, safe, able to express innate behavior, and if it is not suffering from unpleasant states such as pain, fear, and distress…. Protecting an animal’s welfare means providing for its physical and mental needs.”

Even if cats are not physically fighting, chronic distress and inability to cope can occur, with one or more cats in a household unable to perform normal feline behaviors and not feeling safe within the environment. It is now well recognized that distress can result in stress-associated medical problems. The most commonly recognized problems are feline idiopathic cystitis and suppression of the immune response. The latter is responsible for outbreaks of feline herpesvirus-1 in humane shelters; fortunately many shelters now take measures to enrich the environment, preventing severe outbreaks.

Behavior problems, as well as normal behaviors that are considered undesirable occur more commonly in multiple cat households where cats do not get along. This includes marking either as urine spraying and/or increased scratching. Both these behaviors and the owner distress associated with cats not getting along often lead to a breakdown of the human-feline bond. This further compromises feline welfare, either as punishment within the home or surrender of the cat. Surrender could include rehoming, the cat entering the stray cat population, or relinquishment to a humane shelter. Euthanasia often occurs due to associated behavior problems, shelter overcrowding, or lack of adoptability due to fearful behavior within the shelter.

**Why don’t all cats just get along?**

**Understanding normal feline social behavior**

Although cats are social animals, their social system differs from that of dogs and other more social species. Cats do not need other cats to survive, but rather safe territory and essential resources (e.g., enough food, water, and safe space). However, the average number of cats per household is two, and often there are several cats within a household.

In the wild, cats live in colonies of related queens and kittens if there are sufficient resources. The males generally leave the colony when mature and remain solitary. Colonies don’t readily accept strangers or unfamiliar cats. If the stranger continues to come around the colony on a regular basis, it may become familiar and may gradually be integrated into the colony. This gradual process of increasing familiarity should occur when we introduce a new kitten or cat into a household with already existing cat(s).

There are 4 important points that should be remembered here, and we will address how to use this information to help prevent intercat conflict later in the webinar:

- The decision to adopt a cat should not be based on getting a “friend” for another cat.
- Related cats that remain together such as sibling kittens are much more likely to bond for life.
- There must be sufficient resources for cats to live compatibly.
- Cats don’t readily accept strangers or unfamiliar cats. In many cases, intercat conflict begins early on in pet households because owners choose cats to live together that cats in the wild would not choose. Additionally, rapid introduction greatly reduces the chance of existing cat(s) bonding with a new adoption.
Cats not socialized to other cats
Cats that were not socialized to other cats early in life, most specifically during the sensitive period of development (between 2-7 weeks and often up to 9 weeks of age) are less likely to be able to interact normally with another cat. These include single kittens that have no siblings and orphan kittens that did not receive maternal care early on.

Bonded cats that develop intercat conflict
A few different situations can lead to previously bonded cats developing intercat conflict. The first is when one of the cats reaches social maturity at 2-4 years of age. At that stage, that cat is likely to become more territorial and the relationship deteriorates. A medical condition can also lead to a breakdown of the social bond. For example, when a senior cat develops degenerative joint disease or another condition that leads it to withdraw if not adequately controlled. The addition of a new pet to the household can also lead to a change in social bonds.

Redirected aggression can also occur in socially bonded cats. The most common cause is an outdoor cat on the property, and one of the household cats becomes very aroused. If another household cat approaches, the aroused cat redirects the aggression, often inflicting physical injury and fear.

Preventing intercat conflict
Prevention is always easier than treatment, and this is absolutely true with preventing intercat conflict because cats don’t “forgive and forget”, also known as reconciliation. Pre-adoption education or education at first appointments is key to address environmental needs for each cat and how to set up the home with resources in multiple locations. Helping owners with adoption decisions helps prevent intercat conflict and relinquishment. Adopting an already existing social group - siblings or queen and sibling – is ideal. However, owners often have cats at home and want to adopt one more. Counseling to make owners aware that their cat(s) does not need a friend and may not accept a new cat and how to gradually introduce cats reduces the likelihood of intercat conflict. If one cat will be added to the home, choose a kitten that has been well socialized to other cats. Client awareness of subtle signs of conflict – blocking, a cat hiding or less active – and to contact the practice with questions or concerns during the adoption process can prevent intercat conflict and behavior problems.

Introducing a new cat
How a new cat is introduced to the household, including unfamiliar cats within the home makes a tremendous difference in the stress of the household cats, and making the new cat feel comfortable.

Most owners introduce cats by putting them together right away. Although some cats adapt quite readily, the majority have a more difficult time.

There are several different suggested methods for how to introduce a cat, but the most important principles are to educate owners to make introductions very gradually, and ensuring a sense of control for each cat through the provision of multiple resources in different locations. Reward positive and calm behaviors, and isolate cats again if any fear is noted.

The speaker’s preferred method of introduction is to set up a separate room before bringing home the new cat so that it has its own safe space with all resources, including safe hiding spaces such as a cardboard box on its side or cat bed. The existing cat(s) should have their preferred area in the home, also with multiple resources in different locations. Add synthetic feline pheromones to all cat areas, including both the new cat’s space and that of the other cat(s). If cats are calm, swap scent by wiping a towel first on one cat and then the other and repeating daily. Once cats are comfortable with the scents and sounds – usually several days to weeks – start to play with cats under the separating door, and reward calm and curious behavior. If things are going well, open the door a crack so that cats can see each other safely. It is important to remind clients that patience and time are our friends with introductions. Weeks to months can make a lifetime of difference, and trying to introduce too quickly can backfire.

Recognizing intercat conflict
Aggression usually only occurs early in the relationship, most commonly if introductions occur shortly after adoption. Owners are often unaware of the subtle signs of conflict unless behavior problems occur, a thorough history and client education are important at each visit. Ask about number of cats (they may not all come to the practice), and if they demonstrate affiliative behaviors such as grooming one another or demonstrating positive touch (e.g. rubbing against one another), which indicates that they are a social group. If the cats never have positive touch, they are not socially bonded. Cats may have demonstrated affiliative behavior but do not anymore because of redirected aggression, a medical problem, or change within the household.

Also ask whether owners notice any stalking, staring, or blocking as these signs occur more routinely than physical aggression. Behavior problems and stress-associated disease may also occur. Changes in relationships can also occur when a cat develops a illness or pain.

Managing intercat conflict
Always rule out medical problems if there has been a negative change in the cats’ relationship, as painful conditions and chronic disease are not uncommon causes. Behavior consultation and conducting a stress audit will help identify the problems and work with the owner to resolve problems as much as possible. Ensure that each cat has its environmental needs met. Allowing cats to live in separate areas with their option to enter a common space will meet the environmental needs of each cat and reduce distress. Medical therapy may be needed, but should never be used as a replacement for meeting the cats environmental needs and providing a safe and secure environment for each cat. SSRIs reduce anxiety, helping cats learn new behavior, but must be used in conjunction with behavior modification. With the right solutions and owner awareness, cats can have happy homes.

References

2. United States Pet Population and Ownership Trends Report 2017 - Focus on Dogs, Cats, and Other Pets
Although the cat is fairly adaptable, cats have retained many of the behaviors of their wild ancestor, *Felis silvestris lybica.* In fact, pet cats are still more similar to their wild ancestors than to other species and require an environment that provides for their needs. Poor welfare results from unmet needs, with inability to perform normal behaviors, inactivity, stress and stress-associated disease, intercat conflict, and behavior problems. Regardless of whether the cat lives exclusively indoors or is indoor/outdoor, whether in a single or multiple cat home, or whether hospitalized, boarding, or in a humane society, each cat’s needs must be met to ensure good welfare. Veterinary team and client education to understand the cat, and its essential environmental needs prevents physical and mental health problems, and is an important component of the treatment of behavior problems and stress-associated disease.

**Understanding the cat and its needs**

As a unique species and the only solitary hunter to live amicably with people, the cat’s environmental needs are often unrecognized. Cats are territorial animals and essentially solitary survivors that need to protect themselves at all times. The lifestyle of most pet cats differs greatly from that of feral cats – and often from the environment from which they were born. One may consider this to be positive, and it can be, but only as long as the cat’s environmental needs are met. The AAFP/ISFM developed guidelines that address the 5 pillars of feline environmental needs to educate veterinary professionals. Newer information continues to reinforce these pillars, and expand our knowledge of the home and cage environments. Prevention of behavioral problems and stress-associated disease is easiest done through client education of the cat’s needs during pre-adoption and kitten appointments. However, educating about environmental needs can occur at other preventive care appointments and should be incorporated into treatment of stress-associated disease and other medical problems.

**Safe space**

Regardless of whether a cat is free-roam outdoors, indoors, or housed within a cage, cats remain territorial animals with strong protective mechanisms. Safe territory in the wild and safe space within a confined area provide a sense of control, familiarity, and predictability, increasing a cat’s ability to cope. This is especially important when a cat is placed in an environment different from what it knew during the first 2 months of life. Many behavior problems occur because of a threat to the security of safe space.

Hiding is a coping behavior that cats often display in response to changes in their environment. In the home, this could look like a cat hiding in a closet or under the bed because of acute or chronic stressors. Acute stressors may be someone visiting or the addition of a new pet or person to the household. Problems often occur with a newly adopted cat being introduced to already existing household cats without gradual introduction. Unfortunately, chronic distress is also common, and it is not unusual for cats that live in the same household even for long periods not to like each other. Even affiliate cats – cats that like each other – prefer to sleep alone and out of sight of others approximately half the time. Appropriate sleeping areas are also good hiding places, such as a box, a cat bed with high sides, or a carrier with soft bedding. Some safe places should be elevated.

In a cage environment, hiding is also an important coping strategy, whether in the carrier, in the box or other hidden area of a cage, within a tail-sided cat bed, or under a towel. Providing an option to hide helps cats cope and prevents fearful and other aggressive behaviors.

**Multiple and separated resources**

In both a single and multiple cat households, there should be multiple resources placed in multiple locations, with easy access and out of view of other resources. This includes hiding places and use of vertical space to allow cats to be apart if they so choose. Vertical space increases overall space and provides for the cat to oversee the environment. Litter boxes, food, and water stations that are placed in different locations so that individual cats don’t need to see each other reduces competition for resources, bullying, and stress. Signs of intercat conflict are often subtle, seen as blocking a cat from reaching a resource. Therefore, all resources should be placed in rooms favored by each cat, and away from hallways and stairs, and should have at least 2 entry/exits to prevent conflict with another individual. Even cats that demonstrate affiliative behavior, such as rubbing against or grooming each other, or sleeping in close physical contact prefer to rest, toilet, and eat by themselves.

**Resting areas and vertical space**

Increasing overall space by providing cat trees, perches, shelves, or other vertical space helps prevent conflict between cats. Cats can also monitor or oversee the environment from a vertical space, increasing security and predictability. Perches should be placed in multiple areas of the home. Hospitalized and boarding patients that are awake and able should also have areas to perch.
Food and water
Cats should not be fed together, and cats prefer water placed away from their food. More information about predatory and feeding behavior is found under “Play and predatory behavior.

Litter boxes
Litter boxes should be placed in multiple locations around the home, but away from food, water, and sleeping areas. It is not uncommon for cat owners to prefer to place 2 or more litter boxes in the basement next to each other. This poses multiple problems – usually there are noisy appliances and equipment in the basement, the boxes next to each other don’t provide easy access to a box if a more confident cat is blocking a timid cat, and a cat with degenerative joint disease or another condition is likely to have difficult climbing stairs. Any of these and more can lead to a cat soiling outside the litter box.

Many litter boxes are also too small for cats. Cats prefer larger boxes so that they can turn around, dig, and eliminate. Boxes should be 1.5 times the size of the cat from the tip of the nose to the base of the tail. Dog litter boxes and plastic storage containers with an opening make excellent cat boxes. Most cats prefer unscented clumping sand litter. Scooping boxes a minimum of once daily and changing boxes completely when needed (weekly for clay or non-clumping litter and once every 2 or more weeks for clumping litter) will also help to prevent house soiling problems.

Scratching posts
Scratching is a normal feline behavior that marks territory with both visual and olfactory markings. Scratching also sharpens claws, removes old sheaths, and stretches muscles. Scratching behavior is increased with distress, such as with intercat conflict, lack on adequate environmental resources or a real or perceived threat preventing the cat from reaching its resources. 12

Scratching posts should have a sturdy base and be long enough to allow the cat to have a good stretch. Most cats prefer vertical posts, and cat trees with two or more levels and simple upright scratching post are most frequently chosen by cats.13 However, cats 14 years and older prefer horizontal posts on the floor.13 Preferred textures are sisal rope and carpet. 12,13

Educate owners to place posts in rooms that cats consider safe.13 If a cat is also scratching in a different location, such as where new scents occur (e.g., the front or back door), place additional posts in that location. Declawing is opposed by most veterinary organizations in America, and the procedure is outlawed in many countries and in a number of US cities. In addition to preventing cats from performing normal scratching behavior, chronic pain and increased behavior problems have been reported in declawed cats.13

Play and predatory behavior
Cats are not pack hunters, but rather solitary hunters, eating 10-20 small meals per day, with repeated cycles of hunting to catch their small prey. Not all attempts to catch prey are successful (some suggest that up to 50% of the hunt cycles are not successful).14 Think about how much time and energy the cat utilizes just to survive! Compare that to what happens with many owned cats. People usually control the feedings, often providing 1-2 meals daily of highly palatable food. The inability to control access to food is associated with feline stress. The sedentary house cat expends very little energy and time hunting, and more time eating. In some countries, including the US, many cats are kept indoors, depriving cats of mental and physical activity, and contributing to development of obesity and its associated health problems.16

Environmental distress also predisposes to overeating.13 Since people are social eaters usually enjoying meals together, they often provide multiple cats with food either in one bowl or in bowls placed side-by-side, not recognizing that this causes competition for food resources and stress for the cat. One can understand why some cats may eat large volumes very rapidly, often overeating, and perhaps regurgitating.

Regardless of how much cats are fed, the hunting instinct still exists; cats often bring in these unwanted “presents” to their people. Cats are also crepuscular animals, hunting primarily at dawn and dusk, when their prey is usually present. This sometimes leads to waking owners during the wee hours of the morning, which can be quite annoying for humans. Often owners inadvertently reinforce this behavior in their attempt to quiet the cat so that they can go back to sleep, leading to a long-term and frustrating problem for owners. Client education can prevent this problem as long as we welcome clients to discuss their frustrations or concerns about their cats with us.

As veterinarians, we have the opportunity and responsibility to educate clients about normal feeding behavior of the cat as part of the nutritional advice we provide. This will help prevent both medical and behavioral problems, obesity, and stress in the home environment. This can be done by simulating “hunting” through the use of food toys or puzzles, tossing kibbles, or hiding them around the house. This more normal feeding behavior will increase exercise, reduce boredom, and help prevent obesity.14 Providing feeding areas in multiple locations which are out of sight of each other will prevent competition for food resources.

Play is associated with predatory behavior, and an important component of the cat’s day. Interactive and self play multiple times per day will increase the relationship with owners and prevent predatory play leading to human injury.
Positive consistent, and predictable human-cat social interaction

The kitten’s ability to adapt to people and other environments depends on genetics, socialization, experiences, and whether the queen is comfortable around people. Kittens become more social with people if the queen herself is well-socialized and calm around people, and if kittens are present with their mother during positive interactions by the queen with people. The sensitive period for socialization to humans is the time during which particular events will most likely have long term effects on development; for kittens, this is between 2 and 7 weeks of age (much earlier than it is for puppies, which is between 7-14 weeks of age). Kittens that have positive handling experiences during this period cope with stress better, display less fear, and learn tasks more quickly than kittens that don’t receive positive handling during this period.

Older kittens and adult cats can still learn and adapt to new experiences and individuals. However, cats do not learn from punishment, either physical or verbal. Learning occurs through rewarding desired behavior, and with patience and understanding for the cat’s need for a sense of control.

When it comes to interactions with cats, think of affiliative behavior between cats and their need as solitary hunters to have a sense of control. Teach clients to let the cat choose when it wants to interact, and when it wants the interaction to stop. The client should not pull the cat towards them, or make the cat stay with them. To entice the cat to interact, put a hand out or stay in one place with a toy. Massage or pet the cat around the head and neck when the cat approaches. Reward with treats or calm praise, and the amount of petting around the head and neck that the cat desires. Cats often prefer short but more frequent interactions as opposed to our intense, longer, and more infrequent interactions with others. If we pay attention to their social desires, we will be able to win over and interact with almost any cat (except for the most fearful feral).

Interactive play is important to allow the cat enrichment and normal behavior. Each cat should get the time and attention to play with a person individually. Play is often a good initiator to more affiliative interactions between people and cats.

Cats should never be punished, but rather rewarded for desired behavior and redirected from negative behaviors. Habituating cats to what they may potentially be exposed to, including home maintenance procedures, other animals and people of different ages and gender, carriers, and car rides is done through gradual and positive experiences that are rewarded.

Environment respects the cat’s scent and pheromones

Cats mark with scent and pheromones, and are most comfortable in an environment that is familiar and respects their olfactory system.

Treatment of behavior problems, stress-associated disease, and medical conditions that incorporates feline environmental needs

A stress audit with comprehensive behavioral history will identify unmet essential needs and stressors at home that exacerbate needs not being met. These include physical and social changes within the household. Although prevention is always easier, veterinary teams can enhance quality of life and resolve many behavior problems by incorporating the feline essential needs into a comprehensive treatment plan. Although more obvious when behavioral, changes that allow sick or painful cats to perform their normal behaviors is also essential to enhance quality of life.

References


Total body water (TBW) comprises 60% of body weight. The two main fluid compartments in the body are the intracellular fluid (ICF) and extracellular fluid (ECF). The ICF compartment comprises approximately 67% of TBW and the ECF compartment makes up ~26% TBW. The remaining fluid is intravascular fluid (~7% TBW) and transcellular fluid (<1% TBW). Intracellular fluid is found inside the bi-layered cell plasma membrane and is in osmotic equilibrium with the ECF. While ICF and ECF differ markedly in electrolyte composition, their osmolalities are essentially equal due to the high water permeability of most cell membranes.

The ECF is divided into three chambers: the interstitial compartment, the intravascular compartment, and the third space. The interstitial compartment is the fluid space that surrounds cells and allows movement of ions, proteins, and nutrients across cell membranes. Approximately 75% of ECF is in the interstitial compartment and is continuously turned over and recollected by the lymphatic vessels. The intravascular compartment comprises approximately 25% of the ECF, and fluids do not normally collect in the third space. Common examples of the third space, also referred to as transcellular fluid, are the peritoneal fluid, pleural fluid, cerebrospinal fluid, aqueous humor of the eye, fluid within the digestive tract, synovial fluid, and renal tubular fluid.

**Fluid types**

Fluids are classified as crystalloids or colloids.

**Crystalloids**

Crystalloids contain variable amounts of electrolytes, water and dextrose, and are characterized by tonicity and their effect on acid-base status. Crystalloids are used either to replace sodium loss or maintain the status quo. Replacement fluids contain sodium at concentrations similar to normal plasma while maintenance fluids have sodium concentrations similar to normal total body concentration. Approximately one-third of administered isotonic replacement fluid remains in the intravascular space and two-thirds enter the interstitial space.

Dogs and cats normally lose potassium through urine, and this loss is augmented during dehydration, aldosterone release and sodium conservation. Replacement fluids should be supplemented with potassium when used long-term. Normal saline is the fluid of choice for hypercalcemia and hyperkalemia given it contains no calcium or potassium. Normal saline may exacerbate volume overload, metabolic acidosis, heart disease, and hypertension.

Maintenance fluids are designed to replace daily sodium losses and are appropriate for long-term administration. Dextrose is commonly supplemented to approximate plasma tonicity and prevent hemolysis. These lower sodium fluids do not stay in the vascular space, do not meaningfully expand blood volume, and thus should never be used for volume resuscitation.

Hypertonic saline is used for rapid intravascular volume expansion. Volume expansion is short lived, as the sodium redistributes throughout the extracellular compartment quickly. Do not administer hypertonic saline faster than 1 mL/kg/min to avoid vagally-mediated bradycardia and potential cardiopulmonary arrest.

**Colloids**

Colloids are large molecules that remain in the intravascular space due to the Gibbs-Donnan equilibrium. Smaller volumes compared to cry stallloids are required to achieve intravascular expansion, and thus when used appropriately are less likely to induce hemodilution, hypoproteinemia, extracellular edema, and fluid overload.

Synthetic colloids, most notably dextrans and hydroxyethyl starches (HES), contain high molecular weight particles that allow these fluids to increase plasma osmotic pressure (COP). As albumin is the main contributor to COP, colloids are advantageous in the treatment of hypoalbuminemia due to their ability to increase COP. The HES most commonly used in veterinary medicine are hetastarch, pentastarch, and tetrastarch. The differences between hetastarch, pentastarch, and tetrastarch are the average molecular weight of the particles and the degree of substitution of glucose units on the starch particle with a hydroxyethyl group. Serum \( \alpha \) amy lase degrades HES, and elimination occurs through the kidneys. Therefore, measured serum amylase levels will increase in patients receiving artificial colloid solutions.

Several numbers are used to describe unique qualities of HES solutions, including:

1. **Concentration**: Concentration mainly influences the initial volume effect. A common concentration, 6%, is iso-oncotic in vivo, and thus 1L replaces 1L of blood loss. Concentrations range from 6-10%.
2. **Mean molecular weight (MW)**: Hetastarch has an average molecular weight (450 kDa), pentastarch (260 kDa) and tetrastarch (130 kDa). Larger molecules are degraded more slowly, and accordingly, solutions with a higher average molecular weight last longer.
3. **Molar substitution (MS)**: Molar substitution refers to the modification of the original substance by the addition of hydroxyethyl groups. The higher the degree of molar substitution, the greater the resistance to degradation;
consequently, the fluid remains in the intravascular space longer. A value of 0.7 indicates the HES preparation has an average of seven hydroxyethyl residues per 10 glucose subunits. Starches with this level of substitution are called hetastarches, and similar names are applied to describe other levels of substitution (0.4 – tetrastarch; 0.5 – pentastarch; 0.6 – hexastarch).

4. **C2:C6 ratio**: The C2:C6 ratio refers to the site where substitution occurs on the initial glucose molecule. The higher the C2:C6 ratio, the longer is the T1/2 and subsequently the longer is the persistence in the blood. Synthetic colloids have been associated with side effects, including affecting coagulation and increasing the potential for volume overload due to the efficacy of expanding intravascular volume.

Hemoglobin-based oxygen carriers (HBOC; i.e.: Oxyglobin®) are ultra-purified, polymerized, stroma-free bovine hemoglobin products that promote oxygen off-loading at the tissue level and are potent colloids. The hemoglobin is suspended in a modified LRS solution, has an osmolality of 300 mOsm/L, and has a 3-year shelf life. Administration of HBOCs does not require blood typing and/or cross matching. The potential beneficial effects of HBOCs stem from their COP and vasoconstrictive properties; they are efficient scavengers of nitric oxide (NO) and thus can help combat severe vasodilation commonly observed in patients with systemic inflammatory response syndrome (SIRS), severe sepsis and septic shock. 9,10 Given these fluids are bovine products, one-time use is recommended because of the potential for antibody formation and subsequent immunologic reactions. HBOCs do cause a patient’s mucous membranes, sclera and urine to turn red or yellow, and will affect colorimetric diagnostic blood and urine tests. The availability of HBOCs is currently extremely limited due to decreased commercial production.

Natural colloids are also available for infusion. Fresh frozen plasma (FFP) is collected and spun within 6 hours and frozen ideally at -70°C for up to one year. This fluid contains stable clotting factors (II, VII, IX, X), labile clotting factors (V, VIII), von Willebrand’s factor, fibrinogen and albumin; it does not contain red blood cells and platelets. Indications for FFP administration are replacement of all clotting factors, anticoagulant rodenticide intoxication, von Willebrand’s disease and hemophilia (A & B). A common dose for replacement of clotting factors is 10-20 mL/kg. Frozen plasma (FP) is collected similarly to FFP but is stored for longer than one year. It contains stable clotting factors, fibrinogen and albumin, but does not contain red blood cells, platelets and labile clotting factors. Fresh plasma may be used for hemophilia B, and is administered at similar doses as for FFP. Use of FFP and FP to hypoalbuminemia is not practical or safe excepting in very small patients (cats, toy breed dogs) due to the potential for hypervolemia.

With progressive hypoalbuminemia, the COP similarly decreases, contributing to a fluid shift from the intravascular space to the interstitial space to potentially cause edema if tissue safety factors are overwhelmed. Serum albumin (SA) has been used in critically ill patients to help support blood pressure and to aid in the treatment of significant hypoalbuminemia. Currently two types of serum albumin are available for administration: human serum albumin (HSA) and canine serum albumin (CSA). Human serum albumin (HSA) has been used successfully in both dogs and cats, but both acute and delayed immunologic reactions have been documented. 11,12 This product is available as a solution, and may be infused in 4-hour aliquots over 4-72 hours. Canine serum albumin (CSA) is available as a lyophilized powder for reconstitution with sterile saline. Concentrations currently range from 4-25% and do not contain any preservatives and thus must be administered within six hours. Infused albumin remains in the intravascular space for 24 hours, and therefore close monitoring for fluid overload is required. The reported doses for both HSA and CSA range from 100 mg/kg to 6.3 g/kg.

**Classical starling model**

In 1896, Dr. Ernest Starling said:

- “…there must be a balance between the hydrostatic pressure of the blood in capillaries and the osmotic attraction of the blood for the surrounding fluids.”
- “and whereas capillary pressure determines transudation, the osmotic pressure of the proteins of the serum determines absorption.”

Thus, Starling’s hypothesis stated fluid movement due to filtration across the wall of a capillary is dependent on the balance between the hydrostatic pressure gradient and the oncotic pressure gradient across the capillary. The relationship between hydrostatic pressure and oncotic pressure to promote fluid movement across capillary walls can be described by the following equation:

\[ J_v = K_f \left( \left[ P_c - P_i \right] - \sigma_c(p_c - p_i) \right) \]

Where:

- \( J_v \) is the net fluid movement between compartments
- \( P_c \) is capillary hydrostatic pressure
- \( P_i \) is interstitial hydrostatic pressure
- \( p_c \) is capillary oncotic pressure
• \( \pi \) is interstitial oncotic pressure
• \( \sigma \) is the reflection coefficient
• \( K_f \) is the filtration coefficient

Based on this hypothesis, the net driving pressure is outward at the arteriolar end and inward at the venous end of the capillary. This change in net driving pressure is due to the decrease in the capillary hydrostatic pressure along the length of the capillary.

The endothelial glycocalyx

We now know Dr. Starling’s classical hypothesis was inaccurate. It was wrong because it failed to account for an important structure called the endothelial glycocalyx (EG). The luminal surface of endothelial cells is lined with the ED, thin gel-like matrix of sulfated proteoglycans, hyaluronan, glycoproteins, and plasma proteins.

The principle role of the EG is to maintain vascular permeability. It also shields the vascular walls from direct exposure to blood flow, mediating shear-stress-dependent nitric oxide production. It helps retain vascular protective enzymes (e.g.: superoxide dismutase) and coagulation inhibition factors (e.g.: antithrombin, the protein C system, tissue factor pathway inhibitor). It also helps modulate the inflammatory response by preventing leukocyte adhesion and binding of chemokines, cytokines, and growth factors.

Given these unique characteristics of the EG, Dr. Starling’s hypothesis has now been modified. Important key points:

• The EG is the vital semi-permeable membrane for microvascular fluid exchange, not the vascular endothelia.
• The oncotic pressure gradient is between the plasma compartment and the sub-endothelial glycocalyx space; it is not between the plasma and the interstitial space.
• The interstitial oncotic pressure exerts minimal effect on microvascular fluid exchange. Net outward movement is opposed but not reversed by the oncotic gradient. Simply stated, there is no reabsorption in venules.

Hypovolemia vs. dehydration

Hydration status is a measure of interstitial fluid, and is determined by evaluating skin turgor, moisture of the mucous membranes and possibly enophthalmos. Volume status is a measure of tissue perfusion, and is initially evaluated by checking heart rate, capillary refill time, mucous membrane color and blood pressure. Indiscriminate use of the terms dehydration and hypovolemia risks confusion and therapeutic errors. Hypovolemic cats commonly have prolonged capillary refill times, tend to have pale mucous membranes and are often (but not always) hypotensive. While dogs may present with tachycardia, most cats either have normal heart rates or bradycardia.

If hypovolemia is severe, one may see obtundation, weak peripheral pulses, and lack of venous distension when the veins occluded.

Treatment of hypovolemia should typically be finished within 1–2 hours of presenting to the hospital. This type of resuscitation routinely requires rapid administration of large volumes of intravenous replacement crystalloids known as “shock boluses” until endpoints of resuscitation (EOR) are reached.

Route of administration

Common routes of fluid administration in cats include intravenous (peripheral, central or PICC line), subcutaneous, enteral, intraosseous, and intraperitoneal. Hypovolemic patients should have at least one short large bore peripheral intravenous catheter placed. If venous access is not immediately possible, the intraosseous route may be used until vascular access is achieved. The subcutaneous route is not appropriate for hypovolemic patients as peripheral vasoconstriction severely limits absorption. With mild
dehydration, the subcutaneous route may be adequate. Dextrose should not be delivered subcutaneously, and potassium delivered via this route may induce patient discomfort. Subcutaneously fluids are commonly administered at 10-20 mL/kg per site. Enteral water supplementation may be used to help prevent villous atrophy, and may be combined with other forms of enteral nutritional support.

Volume & rate of administration

When determining the most appropriate fluid volume and rate of administration, one should consider the three major components of fluid administration:

1. Resuscitation
2. Replacement
3. Daily physiologic requirements (“maintenance”)

<table>
<thead>
<tr>
<th>Component</th>
<th>Questions to Ask</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resuscitation</td>
<td>Is the patient hypovolemic?</td>
</tr>
<tr>
<td>Replacement</td>
<td>Is the patient dehydrated?</td>
</tr>
<tr>
<td></td>
<td>Are there any ongoing losses?</td>
</tr>
<tr>
<td>Maintenance</td>
<td>What are the patient’s daily physiologic requirements?</td>
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</tbody>
</table>

During fluid resuscitation, intravascular volume is restored with intravenous fluids. Hypovolemic patients require fluid resuscitation, and the volume infused depends on the stage of shock. Stabilizing interventions for patients with shock should target EORs. With hypoproteinemic hypovolemia, administration of a synthetic colloid may be appropriate. Reassess EOR after each bolus.

Common EORs in veterinary medicine are listed in the table below:

<table>
<thead>
<tr>
<th>Veterinary Endpoints of Resuscitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restoration of normal vital signs</td>
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<tr>
<td>Normalization of abnormal mentation</td>
</tr>
<tr>
<td>Restoration of normal blood pressure (systolic &gt;80-90 mmHg)</td>
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<tr>
<td>Normal serum lactate (&lt;2.0 mmol/L)</td>
</tr>
<tr>
<td>$S_tO_2 &gt; 70%$</td>
</tr>
<tr>
<td>PCV &gt; 25%</td>
</tr>
<tr>
<td>UOP &gt; 1 mL/kg/hr</td>
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<tr>
<td>$S_tO_2 &gt; 93%$ @ $F_iO_2 = 21%$</td>
</tr>
<tr>
<td>CVP = 5-10 cmH₂O</td>
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</tbody>
</table>

After addressing hypovolemia, an appropriate fluid therapy plan must address dehydration, daily physiologic requirements, and ongoing losses. Isotonic crystalloids should be used for fluid replacement (correcting dehydration and replenishing ongoing losses). The volume required to correct dehydration is the product of the estimated % dehydration and body weight in kilograms, and should be delivered over ~6-24 hours. After correcting dehydration, a patient’s fluid therapy plan should be reevaluated. Ongoing losses may be estimated by weighing diarrhea and vomitus, and frequent weight monitoring is recommended to help gauge a patient’s fluid status. Daily physiologic requirements, often termed maintenance requirements, may be calculated either with $[(30 \times BW \text{ in kilograms}) + 70]$ or $(80 \times BW^{0.75})$. Both isotonic and hypotonic crystalloids may be appropriate to meet daily physiologic fluid requirements.

Fluid therapy is commonly employed during the peri-anesthetic period. Potential benefits of providing fluids to healthy patients during the peri-anesthetic period include cardiovascular support, countering potential anesthesia-induced adverse reactions, and correction of normal ongoing losses. The American Animal Hospital Association and American Association of Feline Practitioners recently released fluid therapy guidelines for cats, recommending a peri-anesthetic initial starting rate of 3 mL/kg/hr.

Monitoring

With the provision of fluid therapy comes the requirement to monitor a patient’s response to that intervention. Multiple parameters may be readily evaluated to assess a patient’s response to prescribed fluid therapy. Common physical variables are:

- Body weight: The change in a patient’s body weight is a non-invasive method for evaluating fluid gain or loss on a day-to-day basis. As several variables (feces in colon, degree of urinary bladder filling, insensible losses, etc.) predispose this measurement to errors, one is encouraged to measure a patient’s body weight at the same time of day using the same scale to minimize variability.
- Mucous membranes: Dehydrated patients frequently have dry mucous membranes. Thus, a change from tacky mucous membranes to moist ones commonly indicates a positive response (i.e.: improved hydration) to fluid therapy.
- Capillary refill time (CRT): Patients with poor perfusion typically have prolonged capillary refill times (>2 seconds), and poor perfusion frequently arises from hypovolemia. An improving CRT suggests improved perfusion.
- Skin turgor: Evaluation of a patient’s skin turgor or degree of “skin tenting” is been historically used to assess various degrees of dehydration. Overly hydrated patients are commonly described as having a gelatinous feeling to their skin and may readily develop gravity-dependent edema. Geriatric patients and underweight patients have reduced turgor
normally and overweight/obese patients commonly have increased skin turgor; thus, evaluation of skin turgor in these patients is an unreliable indicator of hydration status and response to prescribed fluid therapy.

- Heart rate: Compensatory and early decompensatory hypovolemic shock in dogs and occasionally cats is associated with a reflex tachycardia. Cats do not consistently develop this compensatory response due to concurrent sympathetic and vagal stimulation. Providing appropriate bolus fluid therapy to hypovolemic dogs and some cats will resolve the compensatory tachycardia, indicating a positive response to prescribed fluid therapy.

In addition to readily measured physical variables, trained medical personnel may easily measure other values with minimal patient discomfort, particularly:

- Blood pressure (BP): Marked hypovolemia can overwhelm a patient’s homeostatic responses to result in hypotension (mean arterial pressure <60 mmHg). Hypertension is exceedingly rare in patients with fluid overload due to the large capacitance of the venous circulation, and patients may manifest hypotension without hypovolemia due to either reduced cardiac contractility and/or reduced systemic vascular resistance (SVR).

- Urine output (UOP): A normal UOP is 1-2 mL/kg/hr, and values of 0.5-1 mL/kg/hr and less than 0.3 mL/kg/hr indicate oliguria and anuria, respectively.

- Central venous pressure (CVP): Central venous pressure is the pressure within the lumen of cranial vena cava and is believed to be equivalent to right atrial pressure. A patient’s CVP is influenced by cardiac output (CO), venous return and venous tone, and thus this variable estimates the relationship between blood volume and capacity. Excluding myocardial dysfunction and/or increased pulmonary resistance, CVP is a reliable indicator of an effective circulating blood volume. In dogs and cats, a normal CVP range is 0-10 cmH₂O, and measurements should always be compared with previous data; trends in CVP are uniquely more useful than singular measurements for assessing a patient’s response to fluid therapy.

- Lactate: Hypoperfusion secondary to a reduction in effective circulating volume readily results in tissue hypoxia that induces Type A lactic acidosis. Improving tissue perfusion via fluid resuscitation may resolve the Type A hyperlactatemia. However, tissue hypoxia may occur without volume depletion (i.e.: reduced cardiac output [CO], decreased SVR, decreased arterial oxygen content [P₅O₂]) and Type B lactic acidosis may occur without hypoxia, and thus lactate values must be interpreted carefully.

- Cage-side sonography: Changes in the diameter of the abdominal aorta in longitudinal section during the respiratory cycle may provide meaningful information about a patient’s volume status. The aorta is viewed at the level of the 11th or 12th intercostal space. A greater than 33% change in diameter suggests hypovolemia while a change of less than 10% suggests hypervolemia.

Measuring the amount of fluid provided to a patient, as well as all the eliminations of a patient, are essential for providing the best fluid therapy. Serially and accurately monitoring a patient’s “ins and outs” are invaluable and potentially challenging, but the astute clinician must be keenly aware of all possible sources of fluid loss from a patient, particularly losses via urine, feces, vomitus, blood, and cavitary effusions. A patient’s treatment sheet should accurately document both the volume of fluid administered to and the total amount of eliminations from a patient in a given time period.

Complications & controversies

Fluid overload

While healthy animals are generally quite tolerant of excessive fluid administration, debilitated patients are less able to endure excessive volume. Signs of fluid overload and overhydration include weight gain, restlessness, tachypnea, dyspnea, serosus nasal discharge, chemosis (cats), adventitious lung sounds (i.e.: crackles), tachycardia, gallop rhythm (cat) coughing and jugular venous distension.

Synthetic colloids

Hydroxyethyl starches have been recommended for resuscitation in patients with hypoalbuminemia and/or sepsis given their ability to induce a more rapid and lasting circulatory stabilization than crystalloids. The use of HES has been called into question and, indeed, advised against in some human patients given various adverse reactions reported in randomized clinical trials, most notably coagulation alterations, immunologic reactions, increased incidence of acute kidney injury (AKI) and need for renal replacement therapy (RRT). Yozova et al evaluated 201 dogs in an ICU who received an infusion of 6% tetrastarch, and they screened for changes in serum concentrations; infusion of this specific HES was not associated with increased creatinine concentrations. Hayes et al showed an increased incidence of adverse outcomes, including AKI and death, in dogs in the intensive care unit (ICU) who received 10% HES when compared to a general ICU population. Sigrist et al showed an increase in AKI grade within 10-day post-HES infusion was significantly associated with an increasing number of days that dogs received the synthetic colloid. Randomized controlled clinical trials regarding the safety of HES in both dogs and cats is needed. Minimally clinicians should limit the use of HES in patients with pre-existing renal injury and/or at risk for renal tubular injury.
Hydroxyethyl starches have been implicated in inducing several coagulation abnormalities in various patient populations, including dogs and cats. Certainly, the potential to induce a coagulopathy must be considered when contemplating the use of HES in septic patients. Both *in vitro* and *in vivo* studies in dogs and cats have documented adverse effects on platelet function and coagulation. However, the clinical relevance of these studies is not yet truly known, and clinical prospective investigation is warranted. Now, use of HES in patients with pre-existing coagulation abnormalities is cautioned.

Although immediate and delayed immunologic reactions to HES have been rarely documented in humans, only anecdotal reports of such reactions exist in veterinary medicine to the author’s knowledge.

**Albumin**

Although human and canine albumins have significant amino acid homology, some important differences exist between the two molecules, thus raising concerns over antigenicity. A large retrospective study of 588 critically ill dogs and cats by Vigano *et al* showed administration of 5% HSA was noted associated with any severe hypersensitivity reactions. The current recommendation is one should not administer HSA after more than one week from the initial dosing due of the increased risk of foreign antigenicity. Both HSA and canine albumin are hyperoncotic solutions, and thus fluid overload and overhydration are possible.

**References**

Available upon request
Beyond the Bloodwork: Diagnostics for Liver Disease
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MedVet Indianapolis
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The liver is a vital organ necessary for many functions in the body, including nutrient metabolism and detoxification of various substances. As a result, liver dysfunction due to many different etiologies can be potentially life threatening. The powerful regenerative capability of the liver, however, makes early disease detection critical when considering a favorable long term prognosis.

Liver diseases can be divided into two broad categories: hepatocellular injury and hepatocellular dysfunction/failure. Clinical signs may overlap between these two groupings, and in general will typically be non-specific with liver disease. These categories can frequently be differentiated based on initial laboratory work, including a serum chemistry panel, complete blood count, and urinalysis. More often, however, baseline blood work will be suggestive of generalized liver disease but more information will be required to make informed decisions on treatment and prognosis.

Serum biochemistry profile
ALT, AST, ALP, and GGT are all liver enzymes that can be elevated with liver injury or dysfunction. The pattern of elevation can be helpful in determining the source of the injury. For example, if ALT and AST are substantially higher than ALP then damage to hepatobiliary cells should be suspected. If, however, GGT and ALP are more elevated compared to ALT then biliary obstruction or cell membrane damage should be suspected. Elevated total bilirubin would also be expected with the latter (Center SA 2007). With severe liver disease (i.e. chronic active hepatitis, fibrosis, cirrhosis, toxicity-induced failure, etc.) all four of the previously mentioned enzymes may be markedly elevated due to multifactorial cellular injury. Caution should be taken when evaluating a case where ALT and AST are the only elevated liver enzymes, as these enzymes are found in myocytes and muscle injury will cause their release.

In addition to liver injury and membrane damage, late-stage hepatocellular dysfunction may also be recognized on a biochemistry profile, characterized by a deficiency in products made by the liver. This may include hypoglycemia, hypocholesterolemia, and hypoaalbuminemia. These results remain non-specific, as various gastrointestinal and endocrine diseases will have similar results. Normal concentrations of liver-specific enzymes cannot be used to rule out liver disease in these cases either, as with severe liver failure there may be a deficiency of hepatocytes to even synthesize and release the enzymes. An elevation in serum total bilirubin can also be non-specific, as this can be seen with hepatocellular dysfunction (intra-hepatic cholestasis), extrahepatic biliary obstruction, or even hemolysis unrelated to liver disease.

Complete blood count
Red blood cell microcytosis can be seen in some dogs with liver disease, most frequently being seen with a portosystemic shunt. With acute cholangiohepatitis, neutrophilia with or without a left shift may be present. Thrombocytopenia has been reported with late stage liver failure due to a diminished concentration of thrombopoietin (Webster CRL and Cooper JC 2014).

Urinalysis
Isosthenuria may be present due to medullary washout with liver failure secondary to decreased urea nitrogen production. Overflow of bilirubin will also be reflected in the urine, characterized by bilirubinuria. This can be a normal finding in low concentrations, especially in male dogs.

If initial blood work is suggestive of liver disease, additional diagnostics should be pursued in a logical and step-wise fashion.

Blood tests for liver function
When liver function is greatly diminished, many toxins that are ordinarily filtered remain in circulation. The most well documented and easily measured is ammonia. A portosystemic shunt is the most common cause of hyperammonemia, however this can be seen with other diseases that lead to diminished liver function including cirrhosis and fibrosis as well. Measurement of ammonia has historically been challenging due to instability in serum over time, however recent advancements in laboratory technology now allow in-house testing. This now makes serum ammonia concentration testing a reasonable non-invasive first step in the evaluation of liver function. Ammonia testing should be considered especially when the patient has clinical signs suggestive of hepatic encephalopathy.

When diagnosing a portosystemic shunt, fasting serum ammonia concentration has a sensitivity of 85% in dogs. Serum bile acid testing has even higher sensitivity at 93% (Ruland K et al 2010). These results from 2010 contrast a 2006 study where fasting ammonia concentration was found to be both more sensitive (100% vs. 92%) and specific (89% vs. 68%) than serum bile acids for detecting a portovascular anomaly (Gerritzen-Bruning MJ et al 2006). There are many cases where liver function is compromised but not to the extent of hepatic encephalopathy and resulting hyperammonemia, such as with mid-stage chronic active hepatitis. In these
Other laboratory tests
Most coagulation factors are synthesized in the liver, making the measurement of prothrombin time and partial thromboplastin time useful cage-side tests to evaluate liver function. Since clotting times are frequently elevated in dogs with liver dysfunction, this is useful information prior to obtaining liver aspirates or biopsies as well.

Plasma protein C can be used to help differentiate portosystemic shunts from microvascular dysplasia, when these are the top differentials for liver dysfunction (protein C was <70% in 88% of dogs with a portosystemic shunt) (Toulza O et al 2006).

Diagnostic imaging
Abdominal radiographs can be a sensitive test for evaluating liver size in dogs, with microhepatica being associated with a negative gastric axis on the right lateral view while hepatomegaly presents with a near horizontal gastric axis on the same view.

Abdominal ultrasonography by an experienced ultrasonographer is a useful test for evaluating liver shape and size and investigating echotexture. Size and number of visible intrahepatic vessels, intra- and extra-hepatic bile duct size and shape, presence of single or multifocal nodules or masses, etc. are just a few of the potential abnormalities that can be identified using ultrasound. Surgical planning can be accomplished using ultrasound, especially when finding a single mass versus identifying diffuse infiltrative disease. Mass location, however, can be challenging to definitively identify. A recent study showed only 52% success in correctly locating the lobe affected by a single hepatic mass (Wormser C et al 2016). This should be taken in to account when planning a surgery for a liver lobectomy. Ultrasound is the test of choice for some clinicians for identifying a portosystemic shunt, however sensitivity can be as low as 75% (Berent A and Weiss C 2010). If an anomalous vessel is not directly identified on ultrasound, ancillary findings including bilateral renomegaly and urolithiasis as well as microhepatica and inadequate intrahepatic vasculature can help increase the degree of suspicion for a shunt (d’Anjou MA et al 2004).

There are some liver diseases that have characteristic findings on ultrasound, including a confirmed anomalous vessel. Many diseases, however, have non-specific and sometimes even minimal to no abnormalities seen on ultrasound. A recent study found that 64% of livers that had no ultrasonographic abnormalities had some degree of pathology when biopsies were taken, including moderate to severe fibrosis (Kemp SD et al 2013). Nuclear scintigraphy can be used as a highly sensitive test for portovascular anomalies, however availability is limited for most practitioners. Computed tomography (CT) is becoming more prevalent and is available in most specialty practices. This test is sensitive for even small liver masses (ie. primary or metastatic) and with angiography can be diagnostic for portosystemic shunts.

Liver sampling
A fine needle aspiration is the least invasive method of sampling the liver. This can frequently be done with an awake or lightly sedated patient. Use of a 22-gauge needle with a 6cc syringe is the author’s preference. Ultrasound-guidance can help target specific lesions and avoid important vasculature. Cytology results should be interpreted carefully, however, as diagnostic accuracy is marginal. Correlation between cytology and histopathology of the liver is reported to occur only 30% of the time (17/56 dogs) (Wang KY et al 2004). Vacuolar hepatopathy was the diagnosis with the highest degree of accuracy, however this was also the most commonly misdiagnosed disease with cytology. Hepatocellular inflammation was incorrectly identified 75% of the time.

While comparisons can be made, as above, to cytology and histopathology, not all biopsy samples are equal either. There are many ways to obtain a liver biopsy, including some of the following: ultrasound-guided tru-cut biopsy, laparoscopic biopsy, punch biopsy, guillotine method, liver lobectomy, etc. Clinician preference and confidence in various techniques plays a significant role in which method is used, as well as the suspected underlying disease. When a single mass lesion is detected a liver lobectomy accomplishing an excisional biopsy may be recommended, whereas a collection of laparoscopic or surgical punch biopsies may be indicated with diffuse infiltrative disease. Equipment availability will also play a role, as laparoscopic capabilities are not available in all practices.

When an abdominal ultrasound identifies diffuse liver disease it is preferable to biopsy multiple liver lobes if possible. Even though the external appearance of the liver may be similar diffusely the histopathology may vary between lobes; odds of obtaining the correct diagnosis increases with each additional liver lobe that is sampled (Kemp SD et al 2015). Method of biopsy has not correlated well with an increased odds of diagnosing the disease, provided at least 3 portal triads are sampled from each lobe (Kemp SD et al 2015).

References
Webster CRL, Cooper JC. Diagnostic approach to hepatobiliary disease. *Kirk’s Current Veterinary Therapy XV* 2014;139:569-575.


Pancreatitis is a commonly diagnosed condition that affects dogs of all ages. Clinical signs can vary greatly depending on both the chronicity and severity of disease. While in some cases pancreatitis is a straightforward diagnosis, the presenting complaints are often vague or non-specific, diagnostic tests may be misleading, and concurrent illnesses may complicate the clinical picture. This is made even more difficult by the lack of a single gold standard test.

**Diagnosing pancreatitis**

- **Clinical signs**
  - Mild pancreatitis: Decreased appetite, lethargy, loose stools, etc.
  - Severe pancreatitis: Vomiting, diarrhea, abdominal pain, lethargy, fever, hypovolemic shock
- The initial diagnostic testing should help to begin ruling out other illnesses with similar presenting complaints, including gastroenteritis, acute renal failure, gastrointestinal obstruction, cholangiohepatitis, etc.
  - A thorough medical history may be the most important step in making a diagnosis. Questions should focus on whether there have been any changes in diet, has the dog eaten anything unusual lately, is he taking any medications, are there any concurrent illnesses, etc.
  - Physical examination: Is the patient clinically dehydrated, is there abdominal pain (focal vs. non-specific), is nausea present, how do stools look on rectal examination.
    - Will guide the clinician towards a working diagnosis as well as help to start formulating a treatment plan.
  - Baseline blood work: When a dog is presented for evaluation of vague, non-specific clinical signs initial lab work should include a minimum of a serum chemistry panel, complete blood count, and urinalysis.
    - Abnormalities that may be seen directly related to or secondary to pancreatitis may include:
      - Inflammatory leukogram (mild to marked)
      - Non-regenerative anemia
      - Thrombocytopenia (with severe necrotizing pancreatitis, leading to DIC)
      - Azotemia (pre-renal or renal)
      - Cholestasis (secondary to post-hepatic biliary obstruction from inflamed pancreas)
      - Hypoalbuminemia (negative acute phase protein)
      - Elevated amylase and lipase (variable)
      - Metabolic acidosis secondary to azotemia, poor perfusion, etc.
      - Isosthenuria
  - If vomiting and abdominal pain are presenting complaints, then 2-view abdominal radiographs should be included in the initial diagnostic testing. While radiographic changes are often non-specific for pancreatitis this can be a helpful measure to rule out other differentials including intestinal obstruction.

If the above test results remain suggestive of pancreatitis, then more specific testing should be pursued. Since amylase and lipase have poor sensitivity for canine pancreatitis (32-73% and 42-69%, respectively) (Hess RS et al 1998) as well as low specificity (~50%) (Steiner J 2008), additional testing is often necessary to confirm or to rule out the diagnosis. The SPEC cPL (pancreatic lipase assay) is the most accurate confirmatory test for pancreatitis, with a sensitivity of 87-94% and specificity of 81-88% (McCord K et al 2012). A newer test (Precision PSL) has similar accuracy to the SPEC cPL (Kook PH et al 2014). The SNAP cPL has similar sensitivity and specificity to the SPEC cPL and Precision PSL, but has the advantage of being cage-side. Specificity of the SPEC cPL will increase to as high as 88% when a more stringent cut-off of 400ug/L is used, which makes it a preferred test when clinical signs are suggestive of pancreatitis (McCord K et al 2012). When used in union, the SNAP cPL can be an effective and rapid screening tool, however for a more definitive diagnosis (and to obtain a quantitative result) the SPEC cPL should be the follow-up test.

Abdominal ultrasonography is a useful diagnostic test for pancreatitis in the hands of an experienced ultrasonographer. Possibly more so than with any other test for pancreatitis, this is a great deal of user variability with ultrason which makes results difficult to interpret. Challenges in imaging the pancreas are multifactorial, including:

- Some animals can be challenging to image due to body shape (deep-chested dogs), making even identification of the pancreas.
- Patients with severe pancreatitis will frequently have severe abdominal pain which makes accurate imaging difficult without adequate pain management and/or sedation.
Ultrasound machines vary greatly in quality. Older machines will deliver grainy images and make detailed imaging nearly impossible, especially when trying to evaluate the pancreas.

User inexperience is the biggest road block to obtaining an image of the pancreas and being able to interpret the finding.

Even in the hands of an experienced radiologist making a diagnosis of pancreatitis can be demanding. In some cases ultrasonographic changes lag behind clinical signs, and vice versa. Sensitivity of making a diagnosis with ultrasound has at best been shown to be 70% (Steiner J 2010).

The gold standard diagnostic test to confirm pancreatitis remains histopathology, but this is widely considered an unnecessary test that can lead to increased morbidity and mortality. Placing a hemodynamically compromised patient under general anesthesia and manipulating the pancreas may be indicated if there is acute bile duct obstruction or neoplasia is suspected, but a presumptive diagnosis can often be made prior to going to surgery and rarely does a patient benefit clinically from this procedure.

**Treatment of pancreatitis**

Fluid therapy is the most important management strategy in dogs with pancreatitis. Early and aggressive use of IV fluids can be the difference between a patient surviving or not, however caution should be taken to avoid over-use of crystalloids leading to fluid overload. While dogs with mild pancreatitis may thrive on crystalloid therapy alone, patients with more severe pancreatitis often require a more diverse plan. Hypoalbuminemia, vasculitis, severe pain, and hypotension can all be components of pancreatitis requiring a tailored fluid plan including the following:

- Colloid therapy (ie. Vetstarch) in the form of boluses initially to raise BP as well as a continuous infusion.
- Vasopressor therapy such as a dopamine CRI, to help raise blood pressure (once rehydration has been completed).
- Continuous infusion of pain management (ie. fentanyl CRI) either administered separately through a syringe pump or combined in a bag of crystalloids.
- Ongoing anti-emetic therapy in the form of a CRI (ie. metoclopramide).
- Other targeted colloids, including human albumin and fresh frozen plasma. The success / failure of these products with acute pancreatitis has not been confirmed, and there are risks especially with albumin, but severely critical cases may require this level of aggressive management.

Additional medical therapy is dictated by the patient’s ongoing clinical signs and severity of clinical illness, including the use of other antiemetics, intravenous antacids, alternative pain control, supplemental oxygen therapy, etc.

Management of chronic pancreatitis can be frustrating, especially if the only abnormality is in the blood work. In asymptomatic dogs with persistently elevated cPL, for example, a low fat diet may be all that is indicated. If the dog is symptomatic (including inappetance, mild chronic abdominal pain, intermittent vomiting, etc.) then supportive care including antiemetics, appetite stimulants, antacid therapy, etc. may be necessary during supposed flare-ups. If these therapies are not effective, an alternative diagnosis should be suspected and more testing may be indicated (ie. intestinal or liver biopsies, gall bladder culture, etc.).

Pancreatitis can be a challenging condition to both diagnose and manage, especially when 24 hour care is not available. Learning how to interpret the available diagnostic tests (including having a solid understanding of their pitfalls and inaccuracies) and implementing early and, if necessary, aggressive therapy will help to improve the outcome of your patients with pancreatitis.

**References**


Diarrhea is one of the most common reasons for presentation of a cat to their veterinarian. There are many underlying causes of diarrhea in cats, including both acute and chronic disease. Acute gastroenteritis characterized by diarrhea seems to occur less frequently in cats compared to dogs, possibly because cats are less likely to experience dietary indiscretion after getting into the trash, eating human food, etc. Chronic diarrhea is a more common occurrence, however this can be difficult to detect for some cat owners as certain types of cat litter can help the stool clump and appear more solid than it actually is. Additionally, many pet owners have more than one cat and defecation is rarely observed so it may take longer to make the diagnosis.

Characterize the diarrhea

There are some distinct differences between large and small bowel diarrhea that must be determined prior to pursuing appropriate diagnostic tests. Classic signs of large bowel diarrhea include tenesmus, production of excessive mucus, frequent defecation (up to 5-6 times per day), and frank blood in the stools. In cats exclusively large bowel diarrhea is rare and usually accompanies an infectious disease such as Tritrichomonas or Giardia. Small bowel diarrhea includes weight loss, normal frequency of defecation, large voluminous stool, and normal urgency. In many cases there is some degree of overlap between these two types of diarrhea, however certain diseases are more likely to be associated with either large or small bowel diarrhea so localization can be helpful especially if considering histopathology.

Determining the cause

Most cases of feline diarrhea can be characterized as either infectious or non-infectious. Infectious diarrhea is more common in younger cats, especially cats who have originated from a cattery or a shelter environment. Differentials for infectious diarrhea include feline panleukopenia, Giardia, Tritrichomonas, Campylobacter, multiple intestinal parasites, Histoplasmosis, and Salmonella. Non-infectious causes of diarrhea include food allergy, antibiotic-responsive diarrhea, inflammatory bowel disease (IBD), hyperthyroidism, intestinal neoplasia, pancreatitis, and idiopathic gastroenteritis.

Clinical history is crucial to helping to differentiate infectious versus non-infectious causes of diarrhea. In many cases a detailed medical history will help to prioritize the differential diagnosis list which will help guide further diagnostic tests. Important questions to ask include the following:

- Has the cat spent any time recently in a cattery or cat shelter, or been exposed to other cats that have?
- Does the cat spend any time outside, or indoors only?
- Has there been any change in diet recently?
- Is the diarrhea acute and progressive, or chronic?
- If chronic, has the cat been losing weight?
- Are there any concurrent clinical signs, such as vomiting, loss of appetite, lethargy, etc.?

Knowing the answers to these few questions will go a long way towards determining the first tests that need to be performed. A thorough physical examination should also be performed and can be helpful for similar reasons. While most physical examination findings are unlikely to be pathognomonic for any one disease, there are some classic findings that will help shape your diagnostic plan. Diffusely thickened, or “ropey” intestines is more likely to be associated with chronic infiltrative disease. This finding in addition to enlarged mesenteric lymph nodes is more suggestive of GI lymphoma (although there are other causes including severe IBD and Histoplasmosis). Muffled lung sounds and labored breathing accompanying diarrhea is suggestive of either a protein losing enteropathy (less common in cats compared to dogs) or a diffuse systemic disease such as lymphoma.

Initial diagnostic testing should be prioritized based on the history and physical examination. In all cases of feline diarrhea a routine fecal ova and parasite test should be completed, as even older cats with chronic diarrhea may have a compromised GI immune system making them more at risk for intestinal parasites. I will routinely add Giardia testing on to that as well. If the cat is young and very ill with acute and severe diarrhea, a canine Parvovirus SNAP test can be performed to quickly rule out Feline Panleukopenia. If the cat is young and otherwise healthy with a negative ova and parasite test, and if infectious diarrhea is still considered likely due to the history, then a fecal PCR panel should be considered. This test is available through outside reference laboratories and will test for the following infectious causes of feline diarrhea:

- Campylobacter
- Clostridium
- Salmonella
- Giardia
- Cryptosporidium
- Coronavirus
- Feline panleukopenia virus
- Toxoplasmosis
- Tritrichomonas

Results from this test should be regarded with some skepticism depending on the patient’s clinical signs. For example, many cats may be Clostridium perfringens positive but have diarrhea due to another cause that has compromised the normal flora of the GI tract. In this case using targeted therapy against the Clostridium may help in the short term but would be unlikely to cure the patient’s diarrhea entirely, and it may come back just as bad once treatment is completed. Regardless of the results of fecal testing, there are some intestinal parasites that are not shed regularly and thus may not be seen on routine testing, so a trial treatment with a broad spectrum de-wormer such as fenbendazole should be considered.

Routine chemistry, complete blood count, and urinalysis should be tested primarily to rule out non-intestinal causes of diarrhea. In older cats a T4 should also be checked, especially if weight loss is also occurring. Causes of diarrhea that may be found on routine lab work include kidney disease (acute vs. chronic), pancreatitis (may be suspicious for based on these tests but serum amylose and lipase are highly inaccurate for diagnosis pancreatitis in cats), liver disease (bacterial cholangiohepatitis, liver failure, etc.), and hyperthyroidism.

**Advanced testing**

If the above diagnostics do not provide a diagnosis for the patient’s diarrhea, there are many options on how to proceed. If the diarrhea is chronic, non-life threatening, and no other clinical signs are present, then a diet trial is suggested. For cats my first option is a hydrolyzed diet such as Hill’s z/d, followed by a novel protein diet. The new food should be transitioned slowly over the course of a few days, and then fed exclusively for a minimum of 3 weeks.

If the food trial is unsuccessful but the patient remains stable with only diarrhea (and/or mild vomiting) then an antibiotic trial may next be pursued to rule out antibiotic-responsive diarrhea, such as metronidazole or tylosin. If the patient’s clinical signs are too severe to warrant 4-5 weeks of therapeutic trials, or these both fail, then an abdominal ultrasound should be considered. This test is unlikely to provide a definitive diagnosis for the diarrhea, but can provide valuable information to help determine additional testing. The most common abnormalities seen when investigating chronic diarrhea include overall intestinal wall thickness, loss of normal wall layering, and thickening of the muscularis layer of the small intestine. Additional findings may include enlarged and irregular mesenteric lymph nodes, and enlarged or irregular pancreas, dilated common bile duct, etc. Thickening of the muscularis layer, when coupled with enlarged mesenteric lymph nodes and clinical signs consistent with GI disease, is highly suggestive (although not pathognomonic for) GI small cell lymphoma. Inflammatory bowel disease (most commonly associated with lymphoplasmacytic enteritis / colitis) with no documented underlying etiology has no consistent, reliable changes seen on ultrasound.

Measurement of serum cobalamin and folate should be performed especially in cases of weight loss accompanying the diarrhea. These B vitamins help determine the presence of malabsorption with chronic intestinal disease. The value of this test is low in acute cases and should only be considered if a chronic enteropathy is suspected. Serum TLI should be tested especially if weight loss is present along with the diarrhea, as this is the most common presenting complaint in cats with exocrine pancreatic insufficiency.

If the diagnosis remains elusive with all of the above testing completed, intestinal biopsies should be taken. With presumed diffuse infiltrative disease, gastrointestinal endoscopy is an appropriate option to obtain biopsies. Evidence has shown a difference in histopathologic diagnosis (especially with severity of inflammation) in different segments of the bowel so when possible the stomach, duodenum, colon, and ileum should be sampled. Alternatively, full-thickness surgical biopsies can be taken of these same areas (although full thickness colon biopsies are not typically recommended unless a mass must be removed).

If results of the biopsies do not correspond with the clinical picture, or if neoplasia is suspected but not confirmed, additional testing may be performed. Immunohistochemistry and PARR are two different tests that can be performed on formalin-fixed tissue that will help to confirm or deny monoclonal lymphocyte populations, suggestive of lymphoma.

Treatment of feline diarrhea is entirely dependent on the underlying cause. Some pharmaceuticals such as metronidazole or even prednisolone will be effective for multiple underlying etiologies but will be simply masking the disease and not curing it. Alternative therapies including probiotics may be helpful in conjunction with more targeted therapy.

**References available upon request**
The pancreas consists of both an exocrine and an endocrine component, each with its own very different purpose in normal daily homeostasis. The endocrine pancreas consists of alpha, beta, and delta cells that produce glucagon, insulin, and somatostatin, respectively. The most common disease involving the endocrine pancreas is under-production of insulin by the Beta cells, leading to diabetes mellitus. The much larger (anatomically) portion of the pancreas, the exocrine pancreas, is responsible for producing and secreting digestive zymogens that, when mixed with proteases in the intestinal lumen, become digestive enzymes such as amylase and lipase. Pancreatitis is the most commonly diagnosed disease affecting the exocrine pancreas; however other diseases including exocrine pancreatic insufficiency and pancreatic neoplasia are also reported.

Unlike in dogs, most cats that develop diabetes mellitus retain some normal functioning ability of the Beta cells in the pancreas. This equivalent to “Type 2” diabetes in humans more often occurs due to insulin resistance systemically (due to obesity, systemic inflammation, etc.) and less often due primary pancreatic dysfunction. Since the remaining Beta cells retain their ability to produce and secrete insulin, early management should be focused on trying to diagnose and eliminate the underlying cause of insulin resistance, and an attempt should be made to achieve diabetic remission. In some studies the ability to achieve remission is as high as 40-50%.

A connection between exocrine and endocrine pancreatic disease has been suspected, with the belief that cats with pancreatitis are at higher risk for diabetes mellitus. A recent study, however, has shown that there was no significant difference in pancreatic histopathology between cats with diabetes mellitus and healthy control cats. Additionally, the presence of ketoacidosis did not increase the risk of inflammation. In a separate study in dogs, up to 40% of diabetic cases were associated with pancreatitis.

The primary focus of this talk will be on diseases of the exocrine pancreas. Pancreatitis is by far the most common of these diseases, with neoplasia and EPI being seen much less frequently. Literature has shown that pancreatitis is a highly under-diagnosed disease, as necropsy studies have shown up to 67% of cats have evidence of pancreatic inflammation, including 45% of cats with no clinical signs of GI disease. Relevance of this finding, however, should be questioned, as many of these cats had no prior clinical signs associated with pancreatic disease. While histopathology is considered the gold standard for diagnosis, this is also an impractical test for most cats. This lack of a highly available gold standard test makes the definitive diagnosis even more challenging, and makes a thorough physical examination and history even more important when determining the cause of illness and how to develop a treatment plan.

There is a wide range of disease severity when discussing pancreatitis in cats, from very mild to life-threatening. There are also many suspected triggers for this disease, although in most cases the underlying cause goes unidentified. Recent dietary fat intake and obesity / hypertriglyceridemia is not a common cause of pancreatitis in cats, unlike what is seen in dogs. In up to 2/3 of cases of cats with pancreatitis, however, a concurrent disease is present including inflammatory bowel disease, cholangiohepatitis, diabetes mellitus, etc. Ultimately the most important reason to identify this trigger is to be able to avoid it in the future, as case management will be similar regardless of cause. The most common clinical signs in cats with pancreatitis are lethargy and anorexia, with vomiting only seen in ~40% of cats and diarrhea even less commonly occurring at 11-38%. Other physical examination findings are often vague and non-specific, including depression, dehydration, and cranial abdominal pain.

When a cat is presented with clinical signs consistent with gastrointestinal disease, baseline blood work including chemistry, complete blood count, and urinalysis should be performed. Unfortunately the standard lab variables on these tests (specifically amylase and lipase) are highly insensitive and non-specific for pancreatitis. As a result, interpretation of these two tests should only be done as one component of all other lab and clinical abnormalities. There are, however, many other diseases that will present with similar clinical signs and history that will have detectable abnormalities on these tests. These might include acute cholangiohepatitis, acute renal failure, pyelonephritis, etc. Cats are even less likely to have changes seen with the leukogram compared to dogs.

When pancreatitis is suspected based on clinical history, physical examination, and baseline blood work, additional testing should involve testing pancreatic lipase (SNAP test or SPEC iPL) and an abdominal ultrasound. While pancreatic lipase testing is more sensitive and results are more accurate compared to serum lipase, there are still great deficiencies with these tests. The cage-side SNAP iPL is designed to be highly sensitive and acts as a screening test for pancreatitis; sensitivity ranges up to 92% depending on the severity of the pancreatitis. The SPEC iPL lacks the sensitivity of the SNAP test (54% for mild pancreatitis), but is a more specific test (82%) for pancreatitis. The interpretation of ultrasonographic changes is dependent upon skill and experience of the ultrasonographer. There are no specific guidelines for the diagnosis of pancreatitis, but typical changes seen with the disease include

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The Common and Not So Common

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pancreatic enlargement, peri-pancreatic inflammation or edema, cystic changes, hyperechoic echotexture, and a dilated pancreatic duct.

Ultimately the diagnosis of pancreatitis in cats is made based on a combination of clinical history, physical examination findings, laboratory abnormalities, and ultrasound results. The gold-standard diagnosis is made via histopathology, although this is rarely necessary to make the diagnosis and this is not without a high risk of morbidity.

Treatment of pancreatitis should be dictated based on the severity of the patient’s clinical presentation. Regardless of severity, fluid therapy is the most important component of treatment. Rehydration with crystalloid fluids followed by correction of ongoing losses (if vomiting and diarrhea is present) will help maintain adequate blood flow to the pancreas and aid in healing. Further supportive care is dictated by the patient’s clinical signs (anti-emetics, anti-diarrheal medication, etc.). Pain management can be a very important aspect of therapy as well. Overt abdominal pain is not always clearly evident, but is frequently present. As such, pain management should always be considered in the treatment plan. My top recommendations for the abdominal pain associated with pancreatitis include intermittent buprenorphine injections or, preferably, a CRI of fentanyl. Fentanyl is very fast acting and has a short half-life, which means the dose can easily be manipulated to meet the patient’s needs. Abdominal pain associated with pancreatitis can often be deceiving and lead to lethargy, depression, inappetance, and vomiting, even in cats that don’t outwardly object to abdominal palpation. In these cats a trial of pain management should be instituted to see if clinical signs begin to resolve.

Exocrine pancreatic insufficiency (EPI) is an uncommon pancreatic disease in cats. The presumed cause of this condition is severe end-stage chronic pancreatitis, resulting in loss of the acinar cells; however there are many cats that have no clinical history of chronic pancreatitis that are diagnosed with this disease. Regardless of the cause, clinical disease is typically not detectable until ~95% of pancreatic function is lost. Unlike in dogs with EPI who are frequently presented with chronic history of a ravenous appetite and voluminous soft stools, the most common presenting complaint in cats is weight loss. Diagnosis of EPI in cats is made by confirming a low serum TLI (trypsin like immunoreactivity). Management of this disease is simple in most cats, as they will frequently respond quickly to supplemental pancreatic enzymes added to the diet. When adding the enzyme, it is suggested to moisten the food (if feeding kibble), sprinkle the powder over the food, and then allow it to sit for 10-15 minutes prior to feeding.

Hypocobalaminemia is a common concurrent finding in cats with EPI, so serum cobalamin should be measured with subsequent supplementation if necessary. If weight gain and clinical improvement are not noticed within a few weeks of starting the pancreatic enzyme then evaluation for other underlying diseases such as inflammatory bowel disease should be considered.

Pancreatic adenocarcinoma is the most common neoplasia found in the cat pancreas. In the largest case series available, 34 cats were reviewed. Of these 34 cats, 11 had evidence of metastatic disease at the time of presentation. Prognosis is guarded with pancreatic adenocarcinoma, especially if the tumor has already metastasized at the time of diagnosis. 10% of cats survived over 1 year, but overall median survival was just 97 days. Median survival was slightly longer if surgery or chemotherapy was pursued (165 days).

There are other less common pancreatic diseases in cats, including pancreatic abscesses (which can be an adverse effect of severe pancreatitis), pancreatic cysts, and parasitic pancreatic disease.

References
Vomiting is a common yet non-specific presenting complaint in dogs, which can be defined as forceful, active expulsion of gastric contents from the body (Twedt D 2010). In some cases, vomiting is a necessary response to expel toxic contents from the body. In the majority of cases, however, a non-gastric disorder will stimulate the emesis center leading to the act of vomiting.

There are two central locations that respond to hormonal influence to inducing vomiting: the emetic center and the chemoreceptor trigger zone. Various factors are capable of stimulating these areas, which makes vomiting such a non-specific clinical finding. These include gastric over-distention, pancreatic inflammation, pain, intestinal stretch receptors, uremic toxins, vestibular imbalance, and other factors. Hormone receptors that are capable of inducing vomiting include serotonin (5-HT3), alpha adrenergic receptors, and neurokinergic (NK-1). This information is clinically important when considering anti-emetic therapy.

The single most important first step when evaluating a patient for vomiting is a detailed medical history. Differentiating vomiting from regurgitation is a vital first step, since the diagnostic approach for each is very different. Identification of the three stages of vomiting (nausea, retching, and actual emesis) is crucial to differentiating it from the more reflexive act of regurgitation. Once vomiting has been established, the rest of the patient’s history will help dictate what additional testing may be indicated to most quickly determine an underlying cause. The following are some pertinent questions to be asked during the history:

- What is the duration and frequency of vomiting
- Is the patient on any current medications
- Has there been a recent diet change or unusual food eaten / table scraps, etc.
- Have any remedies been tried and failed
- What other clinical signs are the patient exhibiting (diarrhea, inappetance, chronic weight loss, etc.)
- Are there any known concurrent diseases
- Has there been recent travel or exposure to infectious diseases

Once the medical history is complete, a thorough physical examination is performed. Baseline lab work can then be completed (chemistry, CBC, urinalysis). Many underlying metabolic diseases that cause acute or chronic vomiting can be identified by reviewing these basic tests, including acute or chronic renal failure, pancreatitis, liver failure, chronic hepatitis, pyelonephritis, and others. In the absence of abnormalities on the initial lab work, further investigation will be needed:

- Abdominal radiographs
  - Evaluate the stomach for over-distention, foreign material, and marked thickness.
  - Inspect the small intestines for two separate populations (one normal, one markedly distended) indicating an obstruction.
  - Evaluate overall serosal detail for suggestion of peritoneal effusion.
  - If there is still suspicion of regurgitation, remember to take a right lateral thoracic radiograph to look for megaesophagus.

- Barium study
  - With increased availability of ultrasound, this is becoming a less desirable test. Barium in the GI tract prevents the ability to perform endoscopy until it has all passed, and if a bowel perforation has occurred barium peritonitis can aggravate the septic process further (Ko JJ 2014).
  - Interpretation can be difficult, especially when trying to determine gastric outflow and speed of transit through the gastrointestinal tract. Gastric and intestinal mobility may be delayed due to an underlying metabolic disease, hypoperfusion, or medications the patient is receiving, leading to a possible false positive for intestinal obstruction.

- Abdominal ultrasound
  - This can be a useful test to determine a cause of vomiting, however be cautious to avoid over-interpreting results. Severe intestinal ileus from pancreatitis, for example, can lead to dilated, fluid-filled loops of intestine. This finding can also be suggestive of an obstruction.
  - While abdominal ultrasound can be a sensitive and specific test when performed by an experienced ultrasonographer evaluating a case of acute vomiting for bowel obstruction, it has a lower utility for cases of chronic vomiting. A recent study showed that abdominal ultrasonography helped establish a diagnosis in only 23% of cases of chronic vomiting(Leib MS 2010). In the majority of cases the results did not change the clinical course.
• Additional lab work
  o Resting cortisol
    ▪ Atypical Addison’s disease is an uncommon cause of chronic vomiting, but should not be overlooked (Sadek D 1996). Failure to recognize this disease prior to anesthetizing a patient for more invasive diagnostics can lead to a possible crisis with increased risk of morbidity. A resting serum cortisol >2.0ug/dL should be sufficient to rule out this condition. Perform a full ACTH stimulation test if the resting cortisol is <2.0.
  o Bile acids
    ▪ Decreased liver function can be present in the absence of marked elevations in liver enzymes; conversely primary gastrointestinal disease can contribute to elevated liver enzymes. Bile acid testing will help to differentiate these disorders.
  o Leptospirosis antibody titer
  o GI Panel
    ▪ Cobalamin/folate
    ▪ SPEC cPL
    ▪ TLI

When a definitive diagnosis cannot be obtained using the above diagnostics, consider the clinical history to help dictate the course of action. When the vomiting is acute and gastritis / acute gastroenteritis are suspected, symptomatic therapy including supportive care and anti-emetics should be pursued. If abdominal pain is initially present and persists, or if it develops after a therapeutic trial has begun, repeat abdominal radiographs may be indicated to recheck intestinal gas distention.

Anti-emetic therapy:
  • Serotonin antagonists (5-HT3 receptor inhibitor) (Plumb D 2015)
    o The 5-HT3 receptors are found both centrally and peripherally.
    o Receptors are stimulated by serotonin when intestinal mucosa is disturbed
    o Ondansetron
  • Substituted benzamides (Plumb D 2015)
    o Dopamine antagonist (and 5-HT3 receptor blocker at higher doses)
    o 5-HT4 agonists
    o Also include prokinetic properties (caution if obstruction is suspected)
    o Metoclopramide, cisapride
  • Neurokinin-1 antagonist (Benchaaoui HA 2007)
    o Acts as a ligand for Substance P in the brain (Substance P-Neurokinin receptor complex is thought to be the final pathway in the vomiting reflex).
    o Effective with both central and peripheral causes of vomiting.
    o Maropitant

Prior to more invasive diagnostics, and in a patient with chronic vomiting, consider prescribing a hypoallergenic diet as an elimination diet trial. If the patient continues to vomit after 3 weeks on an exclusion diet, a food allergy can be ruled out. If a novel protein diet is chosen instead of a hydrolyzed diet, two or three diet trials may be indicated if the patient’s complete diet history is not well known.

If vomiting continues in the face of symptomatic therapy, and a definitive diagnosis has yet to be reached, consider obtaining gastrointestinal biopsies. Gastroduodenoscopy is the least invasive method of obtaining samples for histopathology, with the ability to reach the stomach, duodenum, colon, and possibly ileum. Disadvantages of this technique include availability and experience of the endoscopist, ability to only take mucosal biopsies, and inability to visualize the entire gastrointestinal tract. Benefits include it being an out-patient procedure with minimal complications. Alternatively, a laparotomy with full thickness intestinal biopsies can be pursued. This approach allows for full evaluation of the gastrointestinal tract. If no foreign bodies or masses are identified, multiple full-thickness biopsies can be obtained representing various segments, including stomach, duodenum, jejunum, and ileum. A negative exploratory should not be looked upon as a waste of time or an inappropriate test, but an opportunity to obtain biopsies.

Once histopathology results have been evaluated, any further therapy that may be indicated should be started. If full thickness biopsies are taken surgically and corticosteroid therapy is warranted, I recommend waiting at least 5 days after surgery before starting, to allow adequate healing time.
References
Plumb D. Veterinary Drug Handbook 8th Ed. 2015
Rumen fluid analysis

Evidence of abnormal abdominal shape, increased or decreased rumen motility that is not explained by systemic illness, reduced or abnormal fecal output, changes in rumen texture, or suspicion of rumen acidosis are all indications for rumen fluid analysis. A sample of rumen fluid can be obtained via orogastric intubation or rumenocentesis. A larger volume of fluid can be obtained orally if a weighted tube is used. This approach does have the disadvantage of potential saliva contamination that may hinder accurate pH measurement. Rumenocentesis yields a smaller volume of fluid, but enough for most analyses. This sample will not be contaminated with saliva, but can lead to abscessation at the collection site. To obtain this sample, a five inch needle is inserted in the lower left flank cranial to the stifle and directed towards the opposite shoulder. A small amount of air is introduced to clear the needle, and then 3-5 ml of fluid can be obtained.

Analysis should include visual inspection, measurement of pH, and assessment of bacterial and protozoal activity. The fluid should be olive to brownish-green in animals with a hay diet, deep green in those on grass, and yellowish-brown in those with a diet of silage and grain. A milky gray color is consistent with rumen acidosis, and greenish-black is suggestive of rumen stasis. The fluid should be slightly viscous, which will increase with saliva contamination and decrease with decreased microbial activity. The smell of rumen fluid should be aromatic if normal, and will become more acrid in cases of acidosis and putrid with prolonged rumen stasis. The normal pH should be between 5.5 and 7 with animals on high grain diets tending to be in the lower end of the normal range. Either pH paper or a portable pH meter is an acceptable method to assess rumen pH. A pH less than 5.5 is almost always due to rapid fermentation of grain, while an increased pH can be due to anoxemia or saliva contamination of the sample.

Bacterial activity can be assessed by sedimentation and methylene blue reduction. Normal fluid should separate with small particles settling out and large particles floating within 4-8 minutes. A decreased time is consistent with decreased microbial activity, and an increased time is typically due to increased surface tension that is associated with frothy bloat. Methylene blue reduction is measured by mixing 1ml of 0.03% methylene blue with 10 ml of rumen fluid. It should return to its previous color in less than 10 minutes as the bacteria reduce the dye. Increased time indicates a decrease in bacterial activity.

Protozoal activity can be easily assessed by placing a single drop of fresh fluid on a slide and examining it at low power. There should be numerous large, medium, and small protozoa actively swimming in the fluid. The larger protozoa are the least hardy, so they are the most sensitive to changes in the rumen environment.

Additional analyses can be done in certain cases. Chloride measurement can be useful in determining the type of obstruction in cases of Type II or III vagal indigestion. Normal fluid should be less than 30 mEq/L, and increases are typically due to reflux of abomasal contents due to a pyloric outflow obstruction. This measurement can be made using traditional chemistry analyzers, though acetate may falsely elevate the value. In this case, it must be interpreted in light of serum chloride and bicarbonate.

Transthecal wash

Most routine cases of respiratory disease can be treated based clinical signs without further diagnostics. In outbreaks, it is necessary to obtain a diagnosis of a causative agent as this will impact control recommendations. Necropsy samples may be biased by chronicity and previous treatment; therefore, obtaining a bacterial culture from an early case is ideal. Transthecal washes allow examination of the leukocyte population and provide samples for bacterial and Mycoplasma culture.

The calf or cow is sedated with 0.05-0.1mg/kg of lidocaine. A site for the tracheal puncture is selected in the middle of the neck, ventral to the larynx, and this area is clipped, scrubbed, and infiltrated with lidocaine. A stab incision is made though the skin to allow for easier placement of the catheter. The supplies for this procedure can be purchased as a kit, or repurposed, sterile needles and urinary catheters can be used. A 12 gauge, 3 inch needle is placed between two tracheal rings and directed ventrally into the trachea. This is used as a cannula to pass a stiff catheter (approximately 50 cm, 5 French). The distance that the catheter is passed is dependent on the size of the animal as the goal is to reach the thoracic inlet and the horizontal portion of the trachea. Twenty milliliters of sterile saline is infused, and immediately aspirated. Typical yield is only a few milliliters. If no sample is obtained, the catheter may need to be repositioned cranially or caudally. This injection and aspiration can be repeated several times to obtain adequate samples for culture and cytology. The cannula should be removed first, and then the catheter withdrawn to prevent shearing of the catheter.

Thoracocentesis

Pleuropneumonia can be a rare sequela to respiratory disease in cattle. Other causes of pleural effusion include neoplasia, trauma, traumatic reticuloperitonitis and pleuritis. A cow in respiratory distress with decreased to absent lung and heart sounds on one side of
her thorax likely has a significant fluid accumulation. These animals may also develop right heart failure due to compression of the right atrium. Thoracic ultrasound is invaluable in identifying the location, quantity, and type of fluid present. If ultrasound is not available, a sample can be taken blindly from just caudal to the elbow. Clip and prep the chosen sampling site, and infuse lidocaine into the skin and intercostal muscles. A small stab incision is made in the skin and external surface of the muscles. A 3 in, sterile teat cannula is bluntly placed into the fluid. This will take significant force and care should be taken not to damage the underlying lung or heart. A syringe should be attached to the cannula prior to placement to prevent creation of a pneumothorax if the fluid pocket is missed. A fluid sample is obtained for cytology and culture. If a large amount of fluid is present, the cannula can be removed, and a larger thoracic catheter can be placed in a similar manner to drain the fluid.

**Abdominocentesis**

Collection of abdominal fluid is useful in diagnostic evaluations of cows with vague signs of weight loss and anorexia. It is also useful in cases of suspected lymphosarcoma, peritonitis, and uroabdomen. As rapid fibrin deposition commonly prevents diffuse peritonitis in cattle, the location of abdominocentesis can have diagnostic value. The ventral abdomen just cranialateral to the udder, behind the fold of the flank on the right side is a commonly productive site for initial sampling. Diffuse peritonitis, localized peritonitis due to reproductive trauma, or lymphosarcoma can be diagnosed in this area. Sampling on the left in the same area is generally interpreted in a similar manner, but obtaining a sample is more difficult due to the presence of the rumen. Abdominocentesis in the cranial left abdomen just caudal to the sternum is helpful in diagnosing traumatic reticuloperitonitis. On the cranial right side, lymphosarcoma and peritonitis due to abomasal leakage or liver abscesses can be found. The selected site should be clipped and prepared steriley. The sample can be obtained with an 18 gauge, 1.5 inch needle or a teat cannula. If a teat cannula is used, the site should be infused with lidocaine, and a small stab incision should be made through the skin. The needles should be slowly advanced to check for fluid and prevent enterocentesis. It is not uncommon to not obtain a sample; therefore, the needle (or cannula) is left in place and another location near the previous site is chosen for additional sampling. This can be repeated multiple times until an adequate sample is obtained. Cytology is typically diagnostic, though culture can at times be of value to guide treatment.

**Arthrocentesis**

Analysis of joint fluid can be important in differentiating degenerative joint disease and septic arthritis. Selection of the site for sampling is based on ease of collection (i.e. choose the pouch of the joint that appears the most distended). The site is prepared steriley, and an 18 gauge, 1.5 inch needle is inserted into the joint. The fluid is commonly under great pressure, and will easily be obtained. If the needle is clearly in the pocket of fluid, but no sample is obtained, the joint fluid may be too caseous to pass through the small needle. In this case, the joint can be resampled with a 16 or 14 gauge needle. Cytology and bacterial and Mycoplasm cultures are commonly useful in these cases.

**Cerebrospinal fluid collection**

Most common neurologic diseases of cattle do not produce pathognomonic changes to the CSF, so CSF analysis is only indicated in certain cases to rule out specific diagnoses. Meningitis (most commonly in calves), salt toxicity, and cerebrospinal nematodiasis can all be diagnosed and may be ruled out on CSF analysis. Collection is almost exclusively done at the lumbosacral space. If the animal is recumbent, it should be placed in sternal recumbency with each hindleg out to either side or complete lateral recumbency. In either position or in the standing animal, the lumbosacral space can be identified as a depression that is approximately between the tuber coxae. In most cattle, a 3.5 in spinal needle is sufficient, but in larger cows and bulls, a 5 in needle is needed. In calves, a 1.5 in needle is sufficient. The skin is clipped and prepped, and the skin is blocked with subcutaneous lidocaine. The needle is introduced into the center of the depression, perpendicular to the spine, and exactly on midline. The needle is advanced until a distinct pop is felt as the needle penetrates the ligamentum flavum. Often the animal will twitch or wag its tail at this time. The stylet is then removed and fluid should flow from the needle. Fluid is collected for cytology or measurement of sodium concentration in cases of suspected salt toxicity.
Administration of drugs to food animals presents unique challenges due to veterinarians’ responsibility to ensure that no drug contaminates an edible product. This challenge is compounded by changing regulations associated with treatment of food animals, the relative lack of drug options for many species, and varying considerations for meat-, milk-, and egg-producing animals. Nonetheless, it is imperative that practitioners are cognizant of the legal framework for treatment of food animals including extralabel drug use, understand the importance of withdrawal times, and can apply these concepts to a wide variety of patients that present to mixed animal practices.

The first aspect that is crucial to understand is that food-producing animals are defined by their species and class (i.e. lactating dairy goat), not by the owner’s intended use (i.e. companion). Therefore, any goat, chicken, pig, etc. are all legally classified as food-producing animals and should be treated as such no matter the owner’s intention of ever consuming any edible product from the animal. There are essentially two main designations within food-producing animals—major species: cattle, pigs, chickens, turkeys; and minor species—sheep, goats, rabbits, other poultry, cervids, and aquaculture. Most legal restrictions apply equally to both classifications, though some restrictions are relaxed for minor species which are detailed below.

In order to minimize the risk of drug contamination of the food supply, practitioners are legally obligated to use drugs as labeled in these species. Yet, due to the limited number of FDA-approved drugs for many of these species and some common conditions, extralabel drug use is often necessary. This is allowable in food-producing animals if the health, welfare, or life of the animal is at risk which implies that using drugs in an extralabel manner to improve production is not allowed. Extralabel drug use constitutes a use of a medication in any way that varies from its FDA-approved label. This includes altering the dose or route or frequency of administration, administering the drug for a non-labeled indication (i.e. for treatment of septicemia when labeled for respiratory disease), or giving the drug to a non-labeled species or class of animals (i.e. treating a lactating dairy cow when labeled for beef cattle).

The FDA has designated some drugs and drug classes as prohibited from extralabel use in food producing animals. These restrictions have historically been focused on concerns for toxicity in humans due to residues in edible products, but more recent additions have often been due to an effort to restrict uses of antimicrobials considered critical to human health. The intent is to reduce total usage in order to minimize transfer of antimicrobial resistant bacteria through the food chain. Note that some of these drugs have no approved uses in food animals, and therefore, all uses would be illegal (i.e. chloramphenicol), others are only restricted in certain classes (i.e. phenylbutazone in lactating dairy cattle), some have labeled uses and can be used legally in those narrow parameters (i.e. fluoroquinolones), and some restrictions only apply to major species (i.e. cephalosporins).

Extralabel use is prohibited for the following drugs:

1. Chloramphenicol
2. Clenbuterol
3. Diethylstilbestrol (DES)
4. Dimetridazole
5. Ipronidazole
6. Other nitroimidazoles (including metronidazole)
7. Furazolidone
8. Nitrofurazonc (including topical administration)
9. Sulfonamide drugs in lactating dairy cattle (except approved use of sulfadimethoxine, sulfabromomethazine, and sulfaethoxypyridazine)
10. Fluoroquinolones
11. Glycopeptides
12. Phenylbutazone in female dairy cattle 20 months of age or older
13. Cephalosporins (not including cephalxin) in cattle, swine, chickens, or turkeys for disease prevention purposes; at unapproved doses, frequencies, durations, or routes of administration; or if the drug is not approved for that species and production class.

The following drugs, or classes of drugs, that are approved for treating or preventing influenza A, are prohibited from extralabel use in chickens, turkeys, and ducks:

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1. Adamantanes
2. Neuraminidase inhibitors

In order for a veterinarian to use a drug in an extralabel manner in a food animal, specific criteria must be met. First, it must be done on order of a veterinarian within a valid veterinary-client-patient relationship. This means that any extralabel use of an over-the-counter drug in a food animal must be under the supervision of a veterinarian. Extralabel use of drugs in the feed is prohibited, and the extralabel use cannot cause a violative residue. The drugs used in an extralabel manner must be labeled appropriately with the veterinarian’s name and address, drug name, animals to be treated, dosing regimen, and withdrawal time. Again, this means that drugs purchased over-the-counter and used in an extralabel manner must be labeled appropriately by the veterinarian recommending the treatment.

Additionally, the veterinarian must determine that there is not a labeled drug with the appropriate active ingredient that would be effective. If there is not an appropriate labeled drug (or it is deemed not clinically effective), the practitioner should preferentially use a drug labeled for food animals in an extralabel manner over a drug labeled for companion animals or humans. The veterinarian must make a “careful diagnosis” and ensure that a scientifically sound withdrawal time can be given and adhered to by the owner.

Determining a scientifically valid withdrawal interval for extralabel drug use can be challenging. An appropriate withdrawal interval is influenced by the drug dosing regimen, species being treated, disease condition, and FDA mandated tolerance for residues in specific products. The simplest example is increasing the drug dose. When doubling the dose, the withdrawal interval typically only needs to be increased by one half-life of the drug, which is approximately 10-20% of the labeled withdrawal time. When treating a different disease indication, the impact on the withdrawal time is more difficult to predict. In cases of significant hepatic or renal disease, the withdrawal interval may need to be doubled due to the delayed clearance. Extrapolation from a larger species to a related smaller species (i.e. cattle to goats) is fairly straightforward in that the small species generally metabolizes drugs faster than the larger species. This suggests that the labeled withdrawal interval for cattle would generally be adequate for small ruminants. Nonetheless, it is prudent to extend this withdrawal time somewhat due to differences in residue tolerance. In the labeled species (cattle in this example), there will be an established tolerance for the approved drug in the edible product, but in the off-label species (goat), there is no tolerance established, and therefore it is zero. Therefore, to allow for drug clearance to drop below the limit of detection, extending the cattle withdrawal time is prudent. Many situations arise in which these simplistic approaches are inadequate. In these scenarios, we recommend contacting the Food Animal Residue Avoidance Databank (farad.org) in order to determine the most appropriate withdrawal interval.

Treating laying hens presents unique challenges for veterinarians, particularly in cases of individual hens needing treatment. Few drugs are labeled for use in laying hens, and almost all of these are designed for mass medication through the feed or water. Therefore, many backyard hens are being treated in an extralabel fashion. The physiology of egg production allows for drugs to be incorporated into the yolk up to 6 weeks prior to being laid. Further, once a drug enters the yolk it does not equilibrate with the plasma, so it will persist well beyond the time the drug is cleared from the bloodstream. Therefore, egg withdrawal intervals for extralabel drug use are often quite long to allow for 6 weeks after the drug is cleared from the plasma.
**Fluid Therapy in Calves**

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**Oral fluid therapy**

Oral fluid therapy is the mainstay in treatment of dehydration in neonatal calves. Oral rehydration solutions can be used as a stand-alone treatment in mild to moderate dehydration or combined with IV fluid therapy in cases of more severe dehydration. Goals of oral fluid therapy include correction of dehydration, acidosis, and electrolyte abnormalities and provision of energy. There has long been a theoretical admonition to avoid feeding milk with oral fluids as inhibition of the abomasal milk clot could be detrimental. Several studies have now demonstrated that this risk is overstated, and that calves can continue to gain weight in spite of diarrhea if fed milk. Ideally, milk feedings are continued as usual with administration of oral fluids spaced equally between the feedings.

Oral rehydration solutions should contain at least 90 mmol/L of sodium to provide adequate osmolarity to adequately rehydrate the calf. In order to maximize sodium absorption and subsequent water absorption, it should also contain an amino acid (glycine is most common), volatile fatty acid (acetate or propionate), and glucose (ratio of 1:1-3:1 with sodium). Each of these molecules have co-transporters with sodium so inclusion of them in oral fluids will increase sodium and water absorption. Sodium concentration should not exceed 130 mmol/L and glucose should not exceed a 3:1 ratio with sodium to maintain an appropriate osmolarity of the solution.

Other electrolytes, including potassium and sodium should also be included. Chloride concentration should be less than that of sodium so that the strong ion difference is increased to provide greater alkalining ability. Potassium should also be included as it is commonly lost in diarrheic calves. The optimal level of both of these electrolytes is unclear at this point.

An alkalinizing agent should be included in all oral solutions for calves. Historically, sodium bicarbonate was included but it has significant drawbacks when compared to acetate and propionate. Acetate and propionate do not alkalinize the abomasum, increase absorption of sodium and water, and will provide additional energy when metabolized. Further, they have been shown to be as effective as bicarbonate.

**IV fluid therapy**

Intravenous fluid therapy is often critical to the success in treating calves with diarrhea. Any calf that is greater than 8% dehydrated, cannot stand, or has lost its suckle reflex should receive IV fluid therapy. Goals of fluid therapy in diarrheic calves include correction of hypovolemia, acidosis, hypoglycemia, and electrolyte abnormalities.

Isotonic fluids that are commonly used in calves include 1.3% sodium bicarbonate, lactated Ringer’s solution, and other alkalinizing fluids including Normosol and Plasmalyte. Calves with a severe acidosis (pH<7.2) commonly have a D-lactic acidosis which is very slowly eliminated by the calf. As lactated Ringer’s solution contains a racemic mixture of L and D-lactate, it should be avoided in these calves, and administration of 1.3% sodium bicarbonate is recommended. Adding 150ml of 8.4% sodium bicarbonate or 13g of sodium bicarbonate to 1L of sterile water will create isotonic sodium bicarbonate. Typically 1-4L of this solution is required to correct the acidosis and dehydration. This can be administered over several hours through a temporary jugular catheter. Balanced electrolyte solutions more slowly correct acidemia and should only be used in mild cases or after initial correction of a severe acidosis. Many diarrheic calves will be hyperkalemic, but have a total body deficit of potassium. Therefore, addition of potassium chloride to IV fluids is not recommended without measuring serum potassium levels first. If this is not possible, IV potassium should not be given, and this deficit can be corrected using oral fluids. In an anorexic calf, dextrose can be added to lactated Ringer’s solution to create a 2.5% solution by adding 50ml of 50% dextrose to a 1L bag of LRS. This can be administered at a rate of 2-4ml/kg/hr.

To avoid the use of a short term IV catheter, hypertonic solutions can be used in combination with oral fluids to provide sustained fluid therapy. Hypertonic saline (7.2%) at 4-5ml/kg IV over 5 minutes can be administered, and, when followed by oral fluids, provides equal benefit to continuous IV fluids. This will correct dehydration and decrease serum potassium. Hypertonic saline will also partially correct acidosis as improving renal perfusion will allow for increased excretion of D-lactate. Yet it is unlikely to correct a severe acidosis as which is commonly found in diarrheic calves. To overcome this problem, recent work has focused on the use of hypertonic sodium bicarbonate solutions (5% or 8.4%). Initially discouraged due to concerns over paradoxical CNS acidosis and severe respiratory acidosis, significant side effects of use of hypertonic sodium bicarbonate have not been seen even in cases of hypoventilation. Doses used range from 5 to 10 ml/kg and are typically infused over 5-10 minutes. When followed by oral electrolyte solution, hypertonic sodium bicarbonate is more effective than hypertonic saline in correcting acidosis and as effective in correcting dehydration. It appears to be safe, and can provide a convenient and effective method of correcting dehydration and acidosis in neonatal calves.
Rumen Distension and Dysmotility: Causes, Diagnostics, and Treatment Options
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Rumen distension and dysmotility are not uncommon presentations in both cattle and small ruminants. These clinical signs often are linked as dysmotility can lead to rumen distension and distension can lead to dysmotility. Identifying the underlying cause of the distension and dysmotility and determining if it is truly gastrointestinal of origin is critical to appropriate treatment. Generally, a thorough physical exam combined with some routine diagnostics can accurately identify the reason for rumen dysfunction, and guide appropriate treatment and prognosis.

Normal rumen contour and motility
Examination of rumen shape, fill and motility should be a part of the physical exam on all ruminants. Assessment of abdominal shape and rumen fill provides crucial information on feed intake and potential causes of distension. Decreased rumen motility can be a sensitive indicator of disease, though not specific as many inflammatory processes and increased sympathetic tone will decrease normal rumen motility.¹

Abdominal and rumen contour
Assessment of abdominal shape is preferably done early in a physical exam while observing a cow from a distance. While standing directly behind the cow, determine if the cow’s abdomen appears gaunt, normal or distended.² Abdominal shape is not entirely dictated by rumen shape, but rumen size is the most common reason for abnormal distension.³ Abnormalities identified at this time can be useful in guiding a more thorough examination of the forestomach during the remainder of the physical exam. Nonetheless, practitioners must remember that other conditions including intestinal distension, peritoneal effusion, pathologic accumulation of uterine fluid, and rupture of the prepubic tendon can affect abdominal shape and must be considered.

In a normal cow or small ruminant, the abdomen should be slightly wider than the stifles bilaterally. Typically, it will be somewhat symmetrical, though slight differences from right to left are not uncommon. The most prominent distension on left in a normal cow is typically around the level of the stifle in the mid abdomen due to the fiber accumulation in the rumen. On the right, the normal shape is a slight enlargement below the stifle due to the small intestine.

The rumen should be palpated in the left paralumbar fossa and rectally. The normal rumen stratification can be identified on physical exam. There should be a gas cap in the caudodorsal rumen, a fiber mat throughout most of the rumen, and fluid ventrally. The gas cap, found dorsally, is softer and will immediately return to its previous shape when compressed. The doughy fiber mat is the most easily distinguished layer on palpation as one can press into the rumen wall and leave an indentation when it is palpated rectally. On palpation through the flank, the fiber mat simply feels firm. The fluid layer is found in the ventral left flank. This area is softer than the fiber mat, but ballottement of this area is difficult due to the weight of the rumen contents.

Normal rumen motility
Rumen motility should similarly be evaluated as a part of the physical exam of all ruminants. Simultaneous auscultation and palpation in the left paralumbar fossa will allow the examiner to assess the frequency and strength of rumen contractions while also hearing any abnormal sounds associated with the contraction. The normal rate is 1-3 contractions per 2 minutes. Each contraction should be strong enough to lift the examiner’s hand on the paralumbar fossa. The sound should grow louder and then softer as the fiber mat turns inside the rumen and brushes along the rumen wall. There should not be any splashes or bubbling sounds associated with the contraction.² This assessment of rumen motility measures the contraction rate of the dorsal rumen sac, and does not differentiate primary versus secondary contraction as the dorsal sac will contract with both patterns. In most cases, simply determining the overall rumen contraction rate is adequate.

Abnormal abdominal and rumen shape
Finding that the cow’s abdomen is narrower than her stifles suggests prolonged anorexia as completely emptying the rumen can take several days. While specific in identifying a significant and prolonged decrease in feed intake, a gaunt abdomen provides little guidance as to the underlying problem.²

If cow is found to have a distended abdomen, first characterize the location of the distension, the organ leading to abdominal distension, and determine if the distension is due to the accumulation of gas, fluid or feed material. The distension is most commonly found in the mid abdomen and dorsally on the left, ventrally on the right, dorsally on the left and ventrally on the right, or ventrally bilaterally. Other locations (i.e. just ventrally on the left or just dorsally on the right) are less common due to the abdominal anatomy of ruminants.³
Once the distension is localized, ballottement of the abdomen and rectal palpation can be used to determine the organ or organs leading the change in abdominal shape and whether the abnormal distension is due to gas, fluid or feed material. Based on the location and type of distension, the veterinarian can then develop a relatively short differential diagnosis list (Table 1).

### Table 1

<table>
<thead>
<tr>
<th>Distension Type</th>
<th>Location</th>
<th>Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gas Distension</td>
<td>Dorsal Left</td>
<td>Type 1 Vagal indigestion</td>
</tr>
<tr>
<td></td>
<td>Ventral Right</td>
<td>Uncommon</td>
</tr>
<tr>
<td></td>
<td>Dorsal Left and Ventral Right</td>
<td>Uncommon</td>
</tr>
<tr>
<td></td>
<td>Ventral Bilaterally</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Fluid Distension</td>
<td>Dorsal Left</td>
<td>Rumen acidosis</td>
</tr>
<tr>
<td></td>
<td>Ventral Right</td>
<td>Type 3 or 4 Vagal indigestion</td>
</tr>
<tr>
<td></td>
<td>Dorsal Left and Ventral Right</td>
<td>Type 2 or 3 Vagal indigestion</td>
</tr>
<tr>
<td></td>
<td>Ventral Bilaterally</td>
<td>Peritoneal effusion, hydrops conditions</td>
</tr>
<tr>
<td>Feed material</td>
<td>Unilateral distension on the left side</td>
<td>Rumen impaction</td>
</tr>
<tr>
<td></td>
<td>Unilateral distension on the right side</td>
<td>Abomasal impaction</td>
</tr>
</tbody>
</table>

Unilateral distension on the left side is almost always due to enlargement of the rumen. Palpation of the rumen at paralumbar fossa and rectally will allow practitioners to determine the reason for the distension. Excessive gas will accumulate dorsally, and will feel like a large balloon. This is consistent with a type 1 vagal indigestion (failure of eructation). Excessive fluid distension of the left side is consistent with a rumen acidosis and the subsequent fluid shifts that occur due to osmosis. Early type 2 vagal indigestion (failure of rumen outflow) cases may only have fluid distension on the left, but most commonly they are distended bilaterally. An enlarged, dothy rumen is consistent with a feed impaction due to poor quality feedstuffs or inactivity of the rumen microorganisms. Additional information on these disorders are provided later in this chapter and elsewhere in this volume.

Distension ventrally on the right side is most common either fluid or feed. If the distension is due to fluid, the most likely reasons are type 3 (failure of abomasal outflow) or 4 (failure of pyloric outflow) vagal indigestion or small intestinal distension. With type 3 or 4 vagal indigestion, the abomasum initially becomes distended and then ultimately, the rumen becomes distended as well. Therefore, most of the animals present with bilateral distension. Cattle with small intestinal distension, on the other hand, may not have rumen distension, as they often present with signs of abdominal pain due to the stretch of the intestinal wall earlier in the disease process. Feed distension on the lower right side is consistent with an abomasal impaction.

Bilateral distension most commonly occurs due to fluid accumulation in the rumen or rumen and abomasum. As fluid is trapped in the rumen, it initially distends on the left in the midflank. Overtime, the ventral sac of the rumen expands greatly towards the right such that there is now distension of both sides. If there is a type 3 vagal indigestion, distension of the abomasum will contribute to the ventral, right-sided distension, and eventually fluid will back up into the rumen and cause the left sided distension. In either case, the distension on the left is more diffuse and located in the middle to dorsal region of the flank (“apple” shaped), while the distension on the right is in the ventral flank (“pear” shaped). This combination leads to the description of these cows as “papple” shaped due to their asymmetric bilateral distension.

Bilateral ventral distension is generally due to fluid accumulation in the abdomen or uterus, and therefore, rarely GI in origin (Figure 3). Differentials for these animals include pathologic accumulations of fluid in the uterus due to placental or fetal abnormalities, peritoneal effusion, or uroabdomen. Appropriate history, rectal palpation, and abdominocentesis can be used to differentiate these, but this is beyond the scope of this chapter.

### Abnormalities of rumen motility

Hypermotility of the rumen is a relatively uncommon finding, though in actuality it likely occurs quite frequently. In cases of early rumen distension, hypermotility may be noted as the moderate stretch receptors in the rumen wall are stimulated. The rumen continually senses this distension as a recent meal, and increases the rate of primary contractions in response to this distension. Therefore, in most cases of pathologic rumen distension, there is an early phase associated with rumen hypermotility.4 Due to the early nature of the disease course and mild distension, it is unusual for an owner to present an animal for examination at this stage, and the hypermotility is missed. As the distension increases, the severe stretch then stops rumen contractions, and it is at this stage at which animals are typically examined.

Hypomotility is a much more common finding in clinically ill ruminants. As mentioned above, systemic inflammation or increased sympathetic tone from a variety of causes will decrease rumen motility. Hence, most cases of rumen hypomotility are from causes outside the rumen. A thorough physical exam is necessary to rule out other causes of decreased rumen contractions. Hypomotility due to rumen diseases are most commonly associated with rumen distension or rumen acidosis. When the rumen is severely distended, rumen contraction rate will slow down and ultimately stop. Some disorders, traumatic reticuloperitonitis for example, may first disrupt normal motility, leading to rumen distension, which then further slows the contraction rate. Other disorders, such a physical
obstruction of the omasal canal, lead to a primary rumen distension, and the distension ultimately slows and stops rumen contractions. This distinction is important prognostically, as cases with primary motility disorders are less likely to return to productivity after relieving the distension and underlying problem, while those with hypomotility due to distension are more likely to return to normal function after relieving the distension.

**Disorders associated with rumen distension and dysmotility**

Ruminants with both rumen distension and dysmotility typically are diagnosed with vagal indigestion, though rumen acidosis and rumen impactions should also be considered depending on the animal’s abdominal shape, rumen fill, and dietary history. In spite of the name, clinical cases of vagal indigestion have been repeatedly shown to not involve the vagus nerve in most cases. Further, Hoflund’s original description of the disease based on experimental transection of the vagus nerve does little to guide diagnostic and therapeutic decisions. The classification scheme of 4 types of vagal indigestion by Ferrante and Whitlock provide a more clinically useful approach to understanding these diseases and will be used here. No matter the underlying cause, the disease typically progresses from mild rumen distension leading to hypermotility, then progressive distension causes rumen hypomotility. At this point, the animal usually presents with severe rumen distension, decreased rumen contraction rate, and anorexia. Type 1 vagal indigestion is associated with a failure of eructation. These animals present with gas distension of the dorsal left flank, and rumen hypomotility. This can occur due to a failure of secondary contractions, an inability to clear the cardia of fluid, failure of the cardia to open, or esophageal obstruction. A loss of secondary contractions appears to be relatively rare though this may play a role in the bloat that can be seen in some calves with chronic respiratory disease. It is hypothesized that the vagus nerve can become inflamed in the thorax secondary to the respiratory disease. Bloat that is seen with in laterally recumbent ruminants is due to fluid flooding the cardia in spite of normal rumen motility. Similarly, the froth that can be created from consumption of legumes is sensed as fluid at the cardia, and prevents eructation. Damage to the rumen epithelium in the area of the cardia from rumenitis can damage the receptors responsible for sensing the presence of gas at the cardia allowing it to open for eructation. Obstruction of the esophagus can occur from an intraluminal obstruction (swallowing an apple), an extraluminal mass (tracheobronchial lymphadenopathy in cases of respiratory disease), or a mass at the cardia (papilloma). Note that in all of these cases the distension arises from a failure to eructate, not from an increased rate of gas production. Even with significant gas production from fermentation, the normal ruminant can increase eructation adequately to eliminate the gas.

Animals with type 2 vagal indigestion present with bilateral distension of the abdomen due to fluid accumulation in the rumen. The abdomen is distended at the midflank and dorsally on the left and ventrally on the right. On rectal exam, the classic finding of an “L” shaped rumen is felt due to the significant expansion of the ventral sac towards the right flank. The fluid accumulation arises from a failure of rumen outflow with continued food and water intake and saliva production. The obstruction of the omasal orifice can be either functional or mechanical. Functional failures are most commonly due to traumatic reticuloperitonitis leading to inflammation and adhesions around the reticulum. Without normal reticular contractions, primary contractions are disrupted, and fluid is not aspirated into the omasal canal. Other causes of peritonitis in the cranial abdomen including liver abscesses may present similarly. Mechanical obstructions can occur secondary to consumption of a foreign body including rope, hay netting, or placenta. Masses including fibropapillomas and other neoplasias can also obstruct outflow. In these cases, primary contractions are not disrupted initially, and they serve to maintain the foreign body lodged in the omasal orifice. Once the rumen becomes overly distended, then the rumen contractions stop.

Type 3 vagal indigestion presents similarly to type 2 in that the animal has the classic “papple” shape and fluid distension of the rumen. The difference is that the distension is due to a failure of abomasal motility and outflow. Reflux of abomasal fluid leads to the rumen distension, and the abomasum and rumen both contribute to the abdominal distension that is seen externally. The combination of abomasal and rumen distension leads to rumen hypomotility. Like type 2 vagal indigestion, type 3 also can be due to a functional or mechanical failure of abomasal motility. Functional causes include abomasal lymphosarcoma, traumatic reticuloperitonitis and abomasal damage after an abomasal volvulus. Roughly 15% of cattle with an abomasal volvulus will go on to develop abomasal motility disorders. This appears to be due to ischemic damage to the abomasal wall, peritonitis, and/or damage to the vagus nerve. Mechanical obstructions here are less common, though lymphosarcoma and feed or sand impactions can also physically disrupt pyloric outflow. Iatrogenic causes should be considered including inappropriately placed pyloropexy or incorrect placement of a toggle suture.

Type 4 vagal indigestion is a less well defined syndrome of partial pyloric obstruction or generalized ileus. These animals have less abdominal distension compared to those with type 2 or 3 vagal indigestion. A common reason for this presentation is late term pregnancy, as the fetus may physically impede pyloric outflow or proximal intestinal motility. Other causes are related to severe systemic disease including hypocalcemia, peritonitis, septicemia, and enteritis leading to reduced intestinal motility.

Rumen acidosis is another cause of rumen distension and hypomotility. Due to the rapid production of volatile fatty acids from grain fermentation that exceeds the absorptive capacity of the rumen, water is pulled by osmosis into the rumen. This accumulation of
fluid in the rumen causes a left-sided abdominal distension that may initially appear similar to a type 2 or 3 vagal indigestion. The abnormally low pH of the rumen fluid stops rumen contractions as the rumen attempts to slow fermentation. These animals with rumen acidosis will typically be more depressed and dehydrated than those with vagal indigestion, and examination of the rumen pH allows for easy differentiation of these diseases.

Animals with a rumen impaction will present with a firm, left-sided abdominal distension due to feed accumulation in the rumen. Rumen contraction rate will be variable depending on the degree of distension, and could range from increased to absent. The underlying pathogenesis of this disease could be either a lack of appropriate rumen microbial populations or feeding a low quality, largely indigestible forage. The former can be seen in young animals who begin consuming large amounts of forage prior to developing a functional rumen or in an adult animal who has lost the normal rumen bacterial population after acidosis, anorexia, or antimicrobial administration. When fed indigestible forage, the rumen bacteria cannot adequately breakdown the plant material or the fermentation is excessively slow. This leads to an accumulation of fiber within the rumen as the animal continues to consume a large volume of feed material, yet cannot meet its nutritional needs. Hence, in chronic cases, animals will present with severe rumen distension but extremely poor body condition. The severe weight loss may be overlooked by owners due to the animal’s large abdomen.3

Diagnostic approach to animals with rumen distension and dysmotility

History and physical exam

When examining an animal with rumen distension and dysmotility, a complete physical exam will generally provide practitioners with a reasonably short list of differentials that can be further assessed with minimal diagnostic testing. Prior to examining the animal, it is useful to gather an appropriate nutritional and housing history. How much grain is fed? What is the quality of forage that is provided? Any exposure to legumes? Recent construction or building of fences? Evidence of trash or other potential foreign bodies in the pasture or animal’s enclosure? Has the animal had a recent abomasal volvulus, pyloropexy, or toggle procedure? Then the animal is observed prior to restraint to properly assess abdominal contour as described above.

Rumen contraction rate and strength should be assessed by auscultation of the left paralumbar fossa. Most of these animals will have few or no rumen contractions. If the animal does have some contractions, simultaneous auscultation the reticulum with palpation of the rumen will determine if the contractions are primary or secondary contractions. During the exam, particular attention should be paid to those potential diseases that can lead to vagal indigestion. A withers pinch should be performed. A lack of response could be due to any cause of cranial abdominal pain, though traumatic reticuloperitonitis is the classic disease associated with this finding. Other considerations include a ruptured liver abscess or a perforating abomasal ulcer. Practitioners may get some indication of the underlying problem if the cow responds more severely to sternal pressure on the right or left as traumatic reticuloperitonitis will typically cause more pain on the left, while other causes are more likely located on the right. On auscultation of the thorax, is there evidence of respiratory disease or muffling of the heart associated with traumatic reticulopericarditis? Is there any lymphadenopathy that might be suggestive of lymphosarcoma? On rectal exam, the rumen size and texture is assessed to determine if there is fluid distension of the ventral sac. Also, the pregnancy status of the animal is determined, internal lymph nodes are palpated, and the viscera are palpated for evidence of peritonitis and adhesions.

Ancillary diagnostic testing

Rumen fluid analysis

After completing the physical exam, passing a stomach tube is valuable diagnostically and therapeutically. In many cases of type 1 vagal indigestion, gas will be released when the tube is passed. With type 2 or 3 vagal indigestion, fluid may spontaneously reflux from the tube. If not, fluid should be siphoned off the rumen to reduce the distension and provide a sample for diagnostic evaluation. Upon collection of the fluid, the pH should be evaluated to rule out rumen acidosis. In cases of vagal indigestion, the pH will be normal (5.5-7) or slightly alkaline due to anorexia. The reflux of abomasal fluid with type 3 vagal indigestion is not sufficient to reduce rumen pH out of the normal range. When collecting rumen fluid orally, it is critical to collect several hundred milliliters of fluid to minimize the impact of saliva contamination on the pH. Excessive saliva contamination in a small volume sample will artificially elevate the pH due to the buffering capacity of ruminant saliva. A drop of the fluid should be placed on a microscope slide and evaluated at low magnification to assess protozoal activity. There should be numerous protozoa of varying sizes rapidly moving across the field. This can be used as a proxy measure of general microbial activity as the protozoa appear to be more susceptible to changes in the rumen environment. In particular, the larger Holotrich protozoa appear to be especially sensitive to changes in the rumen environment.22 Acidosis or prolonged anorexia in vagal indigestion are the most common causes of decreased protozoal numbers. This assessment needs to be done relatively rapidly as these protozoa can be quite susceptible to changes in temperature and exposure to oxygen. Bacterial populations can be further investigated by Gram staining a sample of fluid, and measuring the methylene blue reduction time.
A sample of rumen fluid should also be strained for measurement of chloride content. In normal rumen fluid, the chloride content should be less than 30 mEq/L. Abomasal outflow obstructions (type 3 vagal indigestion) cause an increase in rumen chloride as the chloride secreted into the abomasum refluxes back into the rumen. It remains sequestered there due to the rumen epithelium’s relatively poor ability to absorb electrolytes. This finding is quite useful in differentiating type 2 and type 3 vagal indigestion as they often present similarly. It has been demonstrated that acetate in the rumen fluid can falsely elevate chloride measurement when assessed using routine potentiometric blood chemistry analysis. This interference is of less concern in animals with anorexia as the acetate levels will be lower. Further, a chloride level less than 30 mEq/L can be reliably interpreted as normal, while an elevated rumen chloride concentration could be due to abomasal reflux or increased acetate levels. Therefore it is critical to interpret rumen chloride concentrations in concert with blood chemistry analysis.

Table 2

<table>
<thead>
<tr>
<th>Color</th>
<th>Yellow-green to olive green depending on diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>5.5-7</td>
</tr>
<tr>
<td>Protozoal Activity</td>
<td>Abundant protozoa of different sizes</td>
</tr>
<tr>
<td>Methylene Blue Reduction</td>
<td>Less than 5 min</td>
</tr>
<tr>
<td>Chloride</td>
<td>Less than 30 mEq/L</td>
</tr>
</tbody>
</table>

**Blood chemistry analysis**
Assessment of serum chloride and bicarbonate can be useful in distinguishing between type 2 and 3 vagal indigestion for similar reasons as rumen chloride. Reflux of the chloride and subsequent sequestration in the rumen leads to a severe hypochloremia as the chloride is normally reabsorbed in the duodenum. Similarly, the hydrogen ions secreted into the abomasum to acidify the contents are associated with bicarbonate moving into the bloodstream. Normally, the bicarbonate from the bloodstream is then taken by the duodenum to neutralize the abomasal pH when ingesta enters the proximal small intestine. When this flow is disrupted, a severe metabolic alkalosis occurs as the bicarbonate remains in the circulation. Hence, animals with a type 3 vagal indigestion will have a severe hypochloremia and metabolic alkalosis. Those with other rumen motility disorders may have similar electrolyte and acid-base derangements, but not to the same degree. The hypochloremic, metabolic alkalosis in these cases is associated with reduced abomasal motility due to anorexia and systemic disease. Other findings on the blood chemistry analysis can also be instructive as an increased globulins would suggest a chronic inflammatory process such as traumatic reticuloperitonitis.

**Ultrasound of the reticulum**
To definitively identify reticular contractions, it is helpful to use ultrasound to visualize the reticulum as auscultation can be difficult, and does not let one evaluate the strength of the reticular contraction. The reticulum can be identified to the left of midline, just caudal to the xiphoid. It will appear as a U-shaped structure, and only the wall can be seen due to the gas mixed into the ingesta. The cranial sac of the rumen will appear just caudal to the reticulum. The reticulum will have a biphasic contraction in which the first contraction is smaller, and the second completely collapses the reticular lumen as it moves dorsally. Identification of normal reticular contractions in cases of rumen distension suggest that the problem is less likely a functional motility disorder of the stomach. A lack of reticular contractions, on the other hand, may suggest either a primary motility disorder or hypomotility due to rumen distension. Interestingly, many animals with rumen hypomotility will have reticular hypermotility, and this was particularly pronounced in cases of type 2 vagal indigestion. Further, imaging this area can identify abscesses, adhesions, or fluid accumulation associated with traumatic reticuloperitonitis.

**Rumenotomy/Abdominal exploratory**
Abdominal surgery may ultimately be necessary to accurately diagnose the underlying disease in animals with rumen distension and dysmotility. This has the advantage of being both diagnostic and therapeutic. Prior to surgery though, one must determine if the animal most likely has a type 1 or 2 vagal indigestion versus a type 3 or 4. This distinction is important as surgical diagnosis and correction of type 1 and 2 vagal indigestion is best accomplished through a left flank celiotomy and rumenotomy, while type 3 and 4 problems are best addressed from a right flank celiotomy and exploratory.
Table 3

<table>
<thead>
<tr>
<th>Location of Abdominal Distension</th>
<th>Rumen Contents</th>
<th>Rumen Chloride</th>
<th>Serum Chloride</th>
<th>Serum Bicarbonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1--Failure of eructation</td>
<td>Dorsal left</td>
<td>Gas</td>
<td>Normal</td>
<td>Normal to mildly decreased</td>
</tr>
<tr>
<td>Type 2--Failure of rumen outflow</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Normal</td>
<td>Normal to mildly decreased</td>
</tr>
<tr>
<td>Type 3--Failure of abomasal outflow</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Increased</td>
<td>Moderate to severely decreased</td>
</tr>
<tr>
<td>Type 4--Partial failure of pyloric outflow/proximal intestinal obstruction</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Normal to increased</td>
<td>Mild to moderately decreased</td>
</tr>
</tbody>
</table>

Treatment options
Treatment of the most these disorders associated with rumen distension and dysmotility will commonly require a rumenotomy or abdominal exploratory as discussed above. A few principles of therapy applicable to any of the above disorder are discussed below.

**Emergency treatment**
Emergency treatment of severe rumen distension may be necessary even prior to complete evaluation. As the rumen becomes distended, the animal’s ability to breathe is reduced as the rumen impedes normal movement of the diaphragm. Passage of a large diameter orogastric tube should always be one’s initial consideration as this will allow passage of accumulated gas or fluid without the risk of peritonitis associated with rumen trocarization. A surfactant, such as poloxalene, can be administered at this time if there is any suspicion of a frothy bloat. Trocarization of the rumen can be performed in cases of extreme respiratory distress or if passage of an orogastric tube is not possible. A self-retaining, screw-in trocar is best, but no matter what type is used, owners should be made aware of the significant risk of peritonitis.

**Transfaunation**
For any of these diseases, there is likely to be an associated disruption of the rumen microbial populations. This disruption could be due to pH changes following rumen acidosis or due to prolonged anorexia in cases of vagal indigestion. Correction of the underlying cause of the motility disorder is key, but transfaunation with normal rumen fluid can speed the animal’s return to normal productivity by replenishing the microbial populations. Drenching via ororuminal tube an adult bovine (or adding directly to the rumen during a rumenotomy) with 10-16 L of fresh rumen fluid appears to be clinically effective. Similarly, transfaunation of 1-4 L of fresh rumen fluid in sheep and goats can re-establish normal microbial populations.28

**Conclusions**
Rumen distension and dysmotility (most commonly hypomotility) are often found together in clinical cases. A thorough physical exam to determine the location of the rumen distension, assess the rumen contents, and careful auscultation of rumen contraction patterns will commonly provide the examiner with a relatively short differential diagnosis list. From here, rumen fluid analysis, ultrasound of the reticulum, and blood chemistry analysis can further guide surgical planning. Based on these findings, the practitioner can then make an informed decision concerning the surgical approach—left flank rumenotomy for rumen acidosis, type 1, or type 2 vagal indigestion or a right flank exploratory for type 3 or 4 vagal indigestion.

**References**
Severe head trauma is associated with high mortality in human beings and animals. Although there is no standard of care for head trauma in human medicine, a series of guidelines have been developed centered around maintaining adequate cerebral perfusion. The appropriate therapy for head trauma patients remains controversial in veterinary medicine due to a lack of objective information on the treatment of dogs and cats with head injuries. Treatment of affected animals must be immediate if the animal is to recover to a level that is both functional and acceptable to the owner. Many dogs and cats can recover from severe brain injuries if systemic and neurological abnormalities that can be treated are identified early enough.

**Patient assessment**

As with all types of acute injury, the “ABCs” (airway, breathing, cardiovascular status) of emergency care are extremely important. Initial physical assessment of the severely brain-injured patient focuses on imminently life threatening abnormalities. It is important not to focus initially on the patient’s neurological status as many patients will be in a state of hypovolemic shock following a head injury, which can exacerbate a depressed mentation. Hypovolemia and hypoxemia need to be recognized and addressed immediately. In addition, a minimum essential data-base includes a PCV, total protein level, a blood urea level, and electrolyte levels as well as a urine specific gravity. Specific attention should also be paid to the serum glucose levels as hyperglycemia has been demonstrated to be related to head trauma severity, although unlike in humans, a specific association with outcome has not yet been demonstrated. Respiratory system dysfunction can be common after head injury. The most dramatic respiratory abnormality seen following head injury can be neurogenic pulmonary edema. Neurogenic pulmonary edema is usually self-limiting if the patient survives, and will resolve in a matter of hours to days, but can cause severe dyspnea, tachypnea and hypoxemia. Hypoxemia exacerbates the development of secondary tissue damage.

**Neurological assessment**

Neurological assessment should be repeated every 30 to 60 minutes in severely head injured patients to assess the patient for deterioration or to monitor the efficacy of any therapies administered. This requires an objective mechanism to ‘score’ the patient so that treatment decisions could be made logically.

**Medical therapy**

1. **Minimizing increases in ICP**
   
   Simple precautions can be taken in positioning the animal with its head elevated at a 30° angle from the horizontal to maximize arterial supply to and venous drainage from the brain. It is also important to ensure that there is no constrictive collar obstructing the jugular veins as this immediately elevates ICP.

2. **Fluid therapy**
   
   The basic goal of fluid management of head trauma cases is to maintain a normovolemic to slightly hypervolemic state. There is no support for attempting to dehydrate the patient in an attempt to reduce cerebral edema and this is now recognized to be deleterious to cerebral metabolism. In contrast immediate restoration of blood volume is imperative to ensure normotension and adequate CPP. Initial resuscitation usually involves intravenous administration of hypertonic saline and or synthetic colloids. Use of these solutions allows rapid restoration of blood volume and pressure while limiting volume of fluid administered. In contrast, crystalloids will extravasate into the interstitium within an hour of administration and thus larger volumes are required for restoration of blood volume. As a result this could lead to exacerbation of edema in head trauma patient. Hypertonic saline administration (4-5 ml/kg over 3-5 minutes) draws fluid from the interstitial and intracellular spaces into the intravascular space which improves blood pressure and cerebral blood pressure and flow, with a subsequent decrease in intracranial pressure. However, this should be avoided in presence of systemic dehydration or hypovolemia and it should be noted that the effects of this fluid only last up to an hour. Colloid solutions, such as Dextran-70 or Hetastarch should be administered after hypertonic saline is used, to maintain the intravascular volume. Hypertonic solutions act to dehydrate the tissues, thus it is essential that cry stalloid solutions are also administered after administration of HSS to ensure dehydration does not occur. The sole use of colloids will not prevent dehydration; in addition, the co-administration of hypertonic solutions and colloids are more effective at restoring blood volume than either alone.

3. **Osmotic diuretics**
   
   Osmotic diuretics such as mannitol are very useful in the treatment of intracranial hypertension. Mannitol has an immediate plasma expanding effect that reduces blood viscosity, and increases cerebral blood flow and oxygen delivery. This results in vasoconstriction within a few minutes causing an almost immediate decrease in ICP. The better known osmotic effect of mannitol reverses the blood-brain osmotic gradient, thereby reducing extracellular fluid volume in both normal and damaged brain. Mannitol should be
administered as a bolus over a 15-minute period, rather than as an infusion in order to obtain the plasma expanding effect; its effect on decreasing brain edema takes approximately 15-30 minutes to establish and lasts between 2 and 5 hours. Administering doses of 0.25 g/kg appear equally effective in lowering ICP as doses as large as 1.0 g/kg, but may last a shorter time. Repeated administration of mannitol can cause an accompanying diuresis, which may result in volume contraction, intracellular dehydration and the concomitant risk of hypotension and ischemia. It is therefore recommended that mannitol use is reserved for the critical patient (Glasgow coma score of < 8) or the deteriorating patient. There has been no clinical evidence to prove the theory that mannitol is contraindicated in the presence of intracranial hemorrhage. There is contradictory evidence that the combination of mannitol with furosemide (0.7 mg/kg) may lower ICP in a synergistic fashion, especially if furosemide is given first.

4. Oxygenation and ventilation

Hyperoxygenation is recommended for most acutely brain-injured animals. Partial pressure of oxygen in the arterial blood (PaO₂) should be maintained as close to normal as possible (at or above 80 mm Hg). Supplemental oxygen should be administered initially via face-mask as oxygen cages are usually ineffective as constant monitoring of the patient does not allow for a closed system. As soon as possible, nasal oxygen catheters or transtracheal oxygen catheters should be used to supply a 40% inspired oxygen concentration with flow rates of 100 ml / kg / min and 50 ml / kg / min respectively. If the patient is in a coma, immediate intubation and ventilation may be needed if blood gas evaluations indicate. A tracheostomy tube may be warranted in some patients for assisted ventilation. Hyperventilation has traditionally been known as a means of lowering abnormally high ICP through a hypopcapnic cerebral vasoconstrictive effect. However, hyperventilation is a double-edged sword. Besides reducing the ICP, it induces potentially detrimental reductions in the cerebral circulations if the pCO₂ level is less than 30-35 mm Hg. The major difficulty with hyperventilation is our present inability to monitor the presence and effects of ischaemia on the brain. It is important that animals do not hypoventilate, and such animals should be ventilated to maintain a PaCO₂ of 30-40 mmHg. Aggressive hyperventilation can be used for short periods in deteriorating or critical animals.

5. Seizure prophylaxis

Although the role of prophylactic anticonvulsants in preventing post-traumatic epileptic disorders remains unclear, seizure activity greatly exacerbates intracranial hypertension in the head injury patient. For this reason, it is recommended to treat all seizure activity in these patients aggressively but not prophylactically. As most cases need to be treated parenterally, phenobarbital (2 mg/kg IM q 6-8hrs) is recommended. This can be continued for 3-6 months after the trauma and can then be slowly tapered off if there have been no further seizures. Phenobarbital will have the additional benefit of reducing cerebral metabolic demands and therefore acts as a cerebral protectant.

6. Corticosteroids

Corticosteroids, known to be beneficial in brain edema attributed to a tumor, have been studied extensively in head injury. Clinical trials in people have shown a beneficial effect of corticosteroids, including methylprednisolone sodium succinate, in the treatment of head injury. In fact, they are now contraindicated based on an increased incidence of mortality following their use. In addition, they have been associated with increased risks of infection, are immunosuppressive, cause hyperglycemia and other significant effects on metabolism.

Surgical therapy

A description of the surgical techniques for intracranial surgery can be found elsewhere. Although it is rare that surgery is indicated in head injury cases, there are several specific abnormalities that can be associated with an episode of head trauma that may warrant the consideration of surgical treatment:

- **Acute extra-axial hematomas**

  Generous craniotomies are generally indicated once these abnormalities have been diagnosed with imaging. If the hematoma is due to a fracture across a venous sinus, there may be profuse bleeding associated with surgical intervention. The need for blood transfusions should be expected. Hematoma removal also risks the chance of bleeding from previously compressed vessels.

- **Calvarial fractures**

  A skull fracture per se may or may not have significant implications for patient management. Skull fractures are typically differentiated based upon pattern (depressed, comminuted, linear); location; and, type (open, closed). A fracture is generally classed as depressed if the inner table of the bone is driven in, to a depth equivalent to the width of the skull. All but the most comminuted, depressed fractures can be managed without operative intervention.

- **Acute intraparenchymal hematomas**

  In contrast to acute extra-axial hematomas, acute intraparenchymal clots may be conservatively managed, unless subacute enlargement of initially small intraparenchymal clots is identified with repeat MR scanning.
Hemorrhagic parenchymal contusions
Most hemorrhagic contusions do not require surgical management. The main indication for surgery with these types of lesions is limited to cerebellar contusions with compression of the 4th ventricle and brain stem; surgery aims to reduce the potential for further compression and herniation, which can develop over the initial 24-48 hours.

Intracranial hypertension (ICH)
Benefit can be found when decompressive procedures are carried out before irreversible bilateral papillary dilation has developed. Conversely, “prophylactic” decompressive surgery seems inappropriate before non-surgical management of elevated ICH has been carefully maximized.
A Video Tour Through the Neurological Examination
Simon Platt, BVM&S, MRCVS, DACVIM, DECVIM
University of Georgia
Athens, GA

The nervous system plays a role in nearly all body processes. Disease syndromes may affect the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system, which includes cranial nerves, spinal cord nerve roots, spinal nerves, peripheral nerve branches, and the neuromuscular junction.

Suspicion of neurological dysfunction arises from the history and physical examination. The signalment, presenting chief complaint, time course of clinical signs, and history may suggest the type of disease process or species-specific disorder. A complete neurologic examination is necessary to localize the anatomic distribution, to determine the severity of the disease process, and to assess the prognosis for patient recovery.

A neurological examination is easily integrated into a routine physical examination. The objectives of the neurological examination are to confirm if there is a neurological abnormality and to specifically localize the abnormality within the nervous system. In conjunction with the history, signalment, presenting complaint and the physical examination, the neurological lesion localization is a piece of a jigsaw essential to creating a list of differential diagnoses for the disease. However, caution must be used as some manipulations necessary for the neurological examination could exacerbate problems such as spinal cord disease.

Observation
Observation of the dog or cat is essential as it allows evaluation of the mentation, posture, attitude, and gait. Changes in mentation (level and content of consciousness) are revealed by a history of personality change, change in awareness of surroundings, and inappropriate behavioural responses. Consciousness is a function of the brainstem (responsible for arousal) and the cerebral cortex (responsible for content and regulation). The evaluation of the state of consciousness can classify the patient as depressed, demented or obtunded, delirious, stuporous and comatose.

Palpation
The musculoskeletal system should be palpated for asymmetry, masses, tenderness and tone. A mass, tenderness, or contour change requires further investigation. The vertebral column should be palpated for deviations and pain being cautious not to apply too much pressure if suspicious of an instability. Unilateral muscle mass loss or atrophy may indicate disuse if it is chronic, or a neurogenic loss if it is acute (within 7-10 days).

Cranial nerves
Cranial nerves have specific functions and evaluation of these functions can help to precisely locate a neurological lesion due to their well-documented anatomy. The general functions and specific tests are summarized in Table 1.

Simplistically, cranial nerve dysfunction may indicate a central nervous system (CNS) lesion (brainstem disease) or a peripheral lesion (affecting the cranial nerves after they have exited the brainstem and course through the skull). Evaluation of the cranial nerves should follow observation and palpation, with particular attention paid to normal functions of eye movement, head movement, blinking, jaw and tongue movement and general symmetry of the head.

Initially an ophthalmic exam should be performed, which will assist with the evaluation of the optic (CN II), oculomotor (CN III), trochlear (CN IV), and abducens (CN VI) nerves.

The following tests are essential to the evaluation of cranial nerve function:

The menace response
- **How to perform** – obscure the vision in one eye and make slow threatening hand gesture toward the other eye.
- **How to interpret** - this is a learned response, not a reflex, to a perceived threat, which evaluates CNs II and VII (responsible for innervation of the orbicularis oculi muscle which closes the eyelids), as well as the central visual pathways and the cerebellum. Normal function is demonstrated by a blink or retraction of the globe in response to the threat. To localize the lesion, other cranial nerve tests would be required.

The pupillary light reflex
- **How to perform** – shine a bright light in each eye to evaluate the response of the pupil.
- **How to interpret** – this is a reflex. Light is sensed by CN II; parasympathetic fibers of CN III cause contraction of the iris muscle with direct and indirect simulation. The pupil is also innervated by sympathetic fibres responsible for dilation, which have their origin in the thalamus and send fibres down the cervical spinal cord to the T1-T3 spinal nerve roots, before they ascend up the neck and through the middle ear. A resting inequality in pupil size is termed anioscoria; to determine which pupil is abnormal, the animal should be evaluated in the light and dark. In the dark, a sympathetic
lesion will mean the affected pupil will not be able to fully dilate. In the light, a parasympathetic lesion will mean the affected pupil will not be able to fully constrict. Animals with sympathetic lesions will often demonstrate miosis in accompaniment to third eyelid protrusion and enophthalmus; a condition called Horner’s syndrome.

**Evaluation of strabismus**
- **How to perform** – observe the animal’s head in a normal position for a deviation of one or both globes in the orbit(s).
- **How to interpret** – cranial nerves III, IV and VI aid vision by maintaining the globe in a central position. Deviation of the globe from its central axis indicates dysfunction in one or more of these nerves: ventrolateral – CN III, dorsolateral – CN IV, and medial – CN VI.

**The palpebral reflex**
- **How to perform** – touch the medial canthus of the normal eyelid and watch response.
- **How to interpret** – the normal eyelid should close. Cranial nerve V (trigeminal nerve) is responsible for facial sensation, whereas the motor response to facial sensory stimulation is generally provided by the facial nerve (CN VII). Facial paresis presents as a drooping of the facial muscles, most notably the lips and the eyelids. It may also be detected as a reduction or absence in the blink response.

**Evaluation of jaw tone**
- **How to perform** – observe patient for a dropped lower jaw and / or an inability to eat. Assess the strength of the jaw safely by manually opening the mouth and evaluating the resistance to opening.
- **How to interpret** – the mandibular branch of CN V provides motor function to the jaw. A dropped lower jaw or the inability to chew can indicate damage to CN V.

**The oculocephalic reflex/physiological nystagmus**
- **How to perform** – move the head from side to side in a horizontal plane and observe the resulting movement of the eyes.
- **How to interpret** – in normal animals, a physiological nystagmus will be induced, with the fast phase in the direction of head movement. This reflex tests the integrity of CN VIII (vestibulocochlear nerve), which is the sensory arm of this reflex, and CNs III, IV and VI, which are responsible for the motor movement of the eyes. Clinical signs of peripheral vestibular disease are manifest after damage to the inner ear or vestibular branch of CN VIII, which effectively gives unbalanced input to the intact central vestibular system. In the absence of head motion, spontaneous horizontal nystagmus is consistent with CN VIII damage, with the fast component away from the side of the lesion. Unilateral peripheral disease may cause a head tilt and circling to the side of the lesion.

**Postural reactions**
The postural reactions are complex, requiring intact sensory and motor pathways throughout the nervous system as well as unimpaired processing and integration in the brain. The complexity of the postural reactions allows detection of minor deficits in any key component of the pathway. Postural deficits are seen caudal to or at the level of the lesion. Additional testing must be performed to use the postural deficit to help localize the lesion within the pathway of the deficit.

- **How to perform** – a leg is placed in an abnormal position and a correcting response by the animal is observed. Knuckling the toes over whilst supporting the body can be done to evaluate how long it takes for the animal to correct. Alternatively, a piece of paper may be placed under each foot and slowly moved sideways, to see if the animal returns its foot to the standing position. Other postural reactions include wheelbarrowi ng, hopping, hemistanding and extensor postural thrust.
- **How to interpret** – conscious proprioception is the patient’s awareness of limb position and movement without visual information. When the knuckling test is performed, an abnormality is indicated by a delay or absence of the response. The sensory branch of proprioception is carried from the skin, muscle and joints of the leg through the spinal cord and brainstem to the sensory motor cortex, where the brain responds by sending messages back to the lower motor neuron for motor function, resulting in a rapid correcting foot placement. Ascending sensory pathways are located in the outermost regions of the spinal cord and are very sensitive to compression. With minor spinal cord injury, proprioceptive deficits may be present because of disrupted sensory pathways, while motor function persists because the deeper motor tracts are unaffected. Both visual and tactile placing reactions require an intact motor cortex and intact motor pathways to the involved limb. A cortical lesion may produce deficits in the contralateral limb, whereas lower lesion produces deficits in the ipsilateral limb.

**Spinal reflexes**
It is rare to have any reflex abnormalities if the animal has no evidence of gait abnormality, muscle mass loss or conscious proprioceptive deficits. In these cases, a complete reflex examination is unlikely to be helpful. Completion of a reflex requires an intact sensory nerve that provides transmission to the spinal cord and an intact motor nerve that elicits function from the innervated
muscle. The reflex arc itself does not involve the brain or the remainder of the spinal cord. Lesions in the motor arm of the reflex arc, termed lower motor neuron (LMN), may cause a decreased or absent reflex (hyporeflexia or areflexia). An exaggerated response (hyperreflexia) results from an interruption in proximal motor pathways that modulate the reflex, termed upper motor neuron (UMN); however, stress or anxiety may cause an apparent increased reflex response, so it should not be considered too important without other evidence of neurological disease. Lower motor neuron signs indicate damage to one or more components of the reflex arc. Upper motor neuron signs indicate damage anywhere between the reflex arc and the brain (Table 2). The most reliable reflex is the flexor withdrawal in the thoracic and pelvic limbs. The other reflexes can appear to be present in small dogs just because the limbs will move when struck with a reflex hammer irrespective of reflex function.

The anal sphincter reflex
- How to perform – pinch the anal sphincter with haemostats and wait for a wink-like contraction of the external sphincter muscles and tail flexion.
- How to interpret – this reflex reveals information regarding the pudendal nerve and caudal segments of the spinal cord. A flaccid unresponsive anus indicates LMN damage to the pudendal nerve or its spinal roots. A hypertonic, hyperreflexive anal sphincter indicates UMN damage at any point cranial to the pudendal nerve.

The pedal flexor reflex
- How to perform – apply a pinch stimulus to each foot and evaluate the response of the ipsilateral and contralateral limb.
- How to interpret – this is a withdrawal reflex in which stimulation of sensory receptors in the toes elicits contraction of flexor muscle groups in the leg. Presence of a withdrawal reflex requires an intact sciatic nerve (sensory and motor) and an intact spinal segment at the lumbosacral plexus, but does not require transmission along the spinal cord to the brain. Absence of the withdrawal reflex in the pelvic limb denotes extensive lower motor neuron damage involving the lumbosacral spinal cord segments (L6-S2) as well as the nerve roots and the lumbosacral plexus; in the thoracic limb it denotes damage to the cervical spinal cord segments (C6-T2), the spinal nerve roots and the brachial plexus.

The patella reflex
- How to perform – a tap stimulus should be applied to the straight patella tendon and the response of the limb should be evaluated. Reflex hammer size must be adapted to patient size for improved accuracy.
- How to interpret – this is a myotactic (stretch) reflex that effectively stretches the quadriceps muscle. This stretch stimulates the femoral nerve (L4-L5), which generates muscular contraction to extend the stifle. Upper motor neuron lesions cause hyperreflexia and should be accompanied by weakness and poor weight bearing. Disease in the L4-L5 spinal cord segments or nerves causes hyporeflexia.

Cutaneous sensation and pain
Cutaneous sensation testing provides information regarding the location and severity of a spinal cord or peripheral nerve lesion. Evaluation of nociception (deep pain perception) is reserved for those animals showing evidence of spinal cord disease based on abnormalities in gait, proprioception and spinal reflexes. Lack of deep pain sensation is a poor prognostic factor as it indicates severe nervous system damage. Nociception requires cerebral perception of painful or injurious stimuli. It is important to remember that a withdrawal reflex is not an indicator of pain perception and may be elicited in an animal whose spinal cord has been transected cranial to the segment responsible for that reflex arc.

Hyperpathia is the sensation of pain produced by an innocuous stimulus such as palpating the vertebrae. All of the cervical and thoracolumbar vertebrae should all be palpated to detect focal points of hyperpathia which may help localize the neurological lesion and will help with the differential diagnosis.

<table>
<thead>
<tr>
<th>Cranial Nerve</th>
<th>Nerve Function</th>
<th>Applicable Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Olfactory</td>
<td>Smell</td>
<td>Blindfold the animal and monitor behavioural response to food placed near nose; loss of smell usually due to nasal disease rather than neurological disease.</td>
</tr>
</tbody>
</table>
| II Optic      | Vision         | i. Menace response  
                       ii. Pupillary light reflex  
                       iii. Obstacle course  
                       iv. Dropping cotton wool balls in front of each eye |
| III Oculomotor| Extrinsic and intrinsic ocular muscles / upper eyelid muscle | i. Eyeball position  
                       ii. Pupil size (mydriatic in disease) |
<table>
<thead>
<tr>
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<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV Trochlear</td>
<td>Extrinsic ocular muscles</td>
<td>iii. Physiological nystagmus</td>
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<tr>
<td></td>
<td></td>
<td>iv. Pupillary light reflex</td>
</tr>
<tr>
<td>V Trigeminal</td>
<td>Facial sensation / jaw movement</td>
<td>i. Eyeball position</td>
</tr>
<tr>
<td>VI Abducens</td>
<td>Extrinsic ocular muscles</td>
<td>i. Eyeball position</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ii. Physiological nystagmus</td>
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<tr>
<td>VII Facial</td>
<td>Muscles of facial expression /</td>
<td>i. Palpebral response</td>
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<td></td>
<td>parasympathetic supply to</td>
<td>ii. Evaluation of facial</td>
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<td>lacrimal glands</td>
<td>symmetry</td>
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<td>iii. Shirmer tear test</td>
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<td>VIII Vestibulocochlear</td>
<td>Hearing and balance</td>
<td>i. Oculocephalic reflexes</td>
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<td></td>
<td></td>
<td>ii. Assessment for head tilt</td>
</tr>
<tr>
<td>IX Glossohpharyngeal</td>
<td>Muscles of pharynx &amp; larynx</td>
<td>i. Gag reflex</td>
</tr>
<tr>
<td>X Vagus</td>
<td>Muscles of larynx, &amp; pharynx</td>
<td>i. Gag reflex</td>
</tr>
<tr>
<td>XI Accessory</td>
<td>Superficial neck muscles</td>
<td>None applicable</td>
</tr>
<tr>
<td>XII Hypoglossal</td>
<td>Muscles of tongue</td>
<td>i. Tongue grab / inspection</td>
</tr>
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Thoracolumbar (type I) disc disease

Clinical signs
Onset of neurological signs may be peracute (<1 hour), acute (<24 hours) or gradual (>24 hours). Dogs presented with peracute or acute thoracolumbar disc extrusions may manifest clinical signs of spinal shock or Schiff-Sherrington postures. These indicate acute and severe spinal cord injury but do not determine prognosis. The degree of neurological dysfunction is variable and affects prognosis. Clinical signs vary from spinal hyperaesthesia only to paraplegia with or without pain perception. Dogs with back pain only are usually reluctant to walk and may show kyphosis. Dogs with back pain alone and no neurological deficits often have myelographic evidence of substantial spinal cord compression. Neuroanatomic localization for thoracolumbar lesions is determined by intact (T3–L3) or hyporeflexive (L4–S3) spinal reflexes and by site of paraspinal hyperaesthesia. Asymmetric neurological deficits may be less reliable for determining the side of disc extrusion.

Diagnosis
The initial diagnosis of thoracolumbar IVDD is obtained from the signalment, history and neurological examination. Differential diagnoses to be considered include trauma, FCE, discospondylitis, neoplasia and (meningo)-myelitis. Diagnosis of thoracolumbar disc extrusion and/or protrusion is confirmed by radiography and surgery. Survey spinal radiography can help to determine the diagnosis and site of a thoracolumbar disc extrusion if Roentgenic signs are well defined and consistent with neuroanatomical localization. Studies of dogs with surgically confirmed thoracolumbar IVDD showed that when identifying the site of disc extrusion survey radiography had an accuracy of 68–72%; but the percentage accuracy was higher with myelography. Normal variants for the thoracolumbar spinal region include narrowing of the antclinal disc space at T10–T11 and of the L4–L6 interspaces. As survey radiographs only identify the correct site of disc extrusion in about 70% of cases, further imaging, such as myelography, is strongly recommended by most neurosurgeons prior to surgery. Myelographic contrast injection at the caudal lumbar region is preferred over the cerebellomedullary cistern for demonstrating thoracolumbar disc extrusion. Longitudinal lesion localization by myelography for thoracolumbar IVDD varies in accuracy from 40% to 97%, but is usually close to 90%. CT or MRI are used alone or as an adjunct to myelography to more completely delineate localization of extruded disc material. CT has been shown to be more accurate than myelography at identifying the major site of disc herniation and has the advantage of being a more rapid test with fewer side-effects than myelography. MRI can provide multiplanar views of the cord compression allowing an accurate surgical approach and can help to identify associated vertebral canal haemorrhage and determining the extent of surgical decompression required. MRI can also identify parenchymal lesions, such as oedema or infarction that may affect the prognosis.

Treatment
Conservative management – Indications for non-surgical treatment of thoracolumbar IVDD include a first time incident of spinal pain only, mild to moderate paraparesis and the financial constraints of the client. The latter is the only reason for non-surgical treatment of a recumbent patient, which should always be considered a surgical candidate if possible. Dogs can be managed with strict cage rest for 4–6 weeks combined with pain relief using anti-inflammatory drugs, opioids and muscle relaxants. Gastrointestinal protectants also may be necessary with use of anti-inflammatory therapies. Dogs should be monitored closely for deterioration of neurological status. If pain persists or the neurological status worsens, surgical management is recommended. Success rates for conservative management of ambulatory dogs with pain only or mild paresis ranges from 82% to 100%. Studies have shown that recovery rates in non-ambulatory dogs are lower and recurrence rates are higher following conservative treatment.

Surgery – Indications for surgical management of thoracolumbar IVDD include spinal pain or paresis unresponsive to medical therapy, recurrence or progression of clinical signs, paraplegia with intact deep pain perception and paraplegia without deep pain perception for <24–48 hours. Prolonged loss of deep pain perception (>48 hours) carries a poor prognosis and owners should be made aware of this prior to surgery. However, it is often difficult to know when deep pain perception was lost and recovery have been observed in dogs that had surgery more than 5 days after the onset of paraplegia. Surgery includes spinal cord decompression by removal of extruded disc material. Chronicity of disc extrusion at the time of surgery may influence the ease with which extruded disc material can be removed. Decompressive procedures for thoracolumbar IVDD are dorsal laminectomy, hemilaminectomy and pediculectomy (also termed mini-hemilaminectomy). There are advantages and disadvantages of each decompressive technique. Hemilaminectomy significantly improves retrieval of extruded disc material with minimal spinal cord manipulation; a clear advantage over pediculectomy and dorsal laminectomy. Pediculectomy is the least invasive and destabilizing technique but these advantages may not be clinically significant except in cases that require a bilateral approach to the vertebral canal. Unilateral facetectomy and
fenestration do not significantly destabilize the spine in lateral bending, which suggests that the articular facets of the thoracolumbar spine are more important to stiffness in axial rotation and extension.

The type of decompressive procedure may not affect outcome; however, the ability to retrieve disc material depends on the decompressive procedure. The primary purpose of decompressive surgery is to provide adequate exposure to allow removal of disc material while minimizing spinal cord manipulation.

Hemilaminectomy provides the same degree of decompression as dorsal laminectomy and is less frequently associated with a post-surgical restrictive laminctomy membrane.

**Prognosis**

Overall success rates after decompressive surgery range from 58.8% to 95%. However, the success of a surgical approach may depend on what criteria are used to define it, how long after the surgery the patient is assessed, as well as the outcome which the owners are willing to accept. Surgical success may be improvement of the patient’s pre-surgery neurological grade but may not mean that the patient is functionally normal and residual signs, e.g. incontinence, can be unacceptable to many owners.

Differences in recovery rates of non-ambulatory dogs vary according to the severity of neurological dysfunction (neurological grade), time interval from initial clinical signs to surgery and speed of onset of signs.

**Neurological grade** – Deep pain perception is considered the most important prognostic indicator for a functional recovery. In general the majority of dogs with intact deep pain perception, whether paraplegic or simply paraparetic, have an excellent prognosis particularly if treated surgically. Dogs with loss of deep pain perception for more than 24–48 hours prior to surgery have a poorer prognosis for return of function. Without surgery, or with delayed surgery, dogs with absence of deep pain perception have an extremely guarded prognosis, although duration of absence of deep pain perception prior to surgery as a prognostic indicator is controversial. Recovery rates for dogs with thoracolumbar IVDD and absent deep pain perception range from 0–76%. A recent study of 87 dogs with loss of deep pain perception reported 58% of the animals regained deep pain perception and the ability to walk. In summary, dogs with absence of deep pain perception that have surgery within 12–36 hours have a better chance of more rapid and complete recovery than those with delayed surgery. Dogs with more severe neurological dysfunction have a longer period of recovery. The mean time from post-surgery to walking varied from 10 days for pain only or paraparetic dogs to 51.5 days for paraplegic dogs.

More recent long-term studies reported recovery times of 2–14 days for dogs that were either ambulatory or non-ambulatory with voluntary motor movement, and up to 4 weeks for paraplegic dogs.

Onset and duration of clinical signs – There are many contradictory studies about the effect of (a) the speed of clinical sign onset and (b) the duration of the clinical signs prior to surgery, on the time taken for recovery and the final outcome. In general it is agreed that rapid removal of extruded disc material facilitates a more complete and rapid recovery. Dogs with a shorter duration of clinical signs prior to surgery and a gradual onset of neurological dysfunction (<48 hours) have a quicker recovery. However, a recent study of 71 paraplegic dogs with intact deep pain sensation demonstrated that although a shorter duration of signs was indeed associated with a shorter recovery time, the rate of onset of clinical signs did not influence the recovery time. However, the rate of clinical sign onset did influence the final outcome. Similarly, a peracute onset of signs indicated a poorer prognosis for dogs with no deep pain perception in one study. The outcome of dogs after hemilaminectomy with the duration of clinical signs has been evaluated and it was shown that delay before surgery does not influence outcome in dogs with mild neurological dysfunction but does affect functional recovery in paraplegic dogs. When performed within 12 hours of clinical sign onset, hemilaminectomy in paraplegic dogs had a higher success rate.

**Cervical disc disease**

**Presentation and pathogenesis**

Cervical disc disease is a common problem in chondrodystrophic breeds of dog such as Dachshunds, Shih Tzus, and Pekingese. It also occurs frequently in Beagles and Cocker spaniels and can occur sporadically in almost any breed. Although thoracolumbar disc herniations have been reported in cats, cervical disc herniations are extremely rare. The intervertebral disc is composed of an outer fibrous portion (the anulus fibrosus) and a gelatinous center (the nucleus pulposus). With normal ageing the nucleus is slowly replaced by fibrocartilage, but in chondrodystrophic breeds the nucleus ages prematurely and the nucleus matrix degenerates and mineralizes.

As a result of these degenerative changes, affected dogs are prone to extrusion of the mineralized nucleus pulposus into the spinal canal, (Hansen type 1 disc herniations) causing spinal cord concussion and compression. The C2/3 disc is most commonly affected, with incidence decreasing further caudally in the cervical spine.

Onset of signs can occur from eighteen months of age, with a peak incidence between three and seven years of age. It is very unusual for a disc herniation to occur in dogs less than two years of age as the predisposing degenerative changes have not occurred. The most common presenting sign is severe neck pain as there is enough space within the cervical vertebral canal for herniation of disc material without compression of the spinal cord. The dog may adopt a stance with the head held down, neck rigid and back arched as the weight is shifted to the pelvic limbs. Entrapment of nerve roots can cause a nerve root signature (holding up a thoracic limb and lameness). The neck pain is so severe that dogs avoid moving their head, and spasm and rigidity of the cervical musculature
Neurological deficits are less common but can occur when the spinal cord is sufficiently compressed and include tetraparesis or -plegia, ataxia, and conscious proprioceptive and postural reaction deficits.

**Diagnosis**
Survey radiographs should be taken to identify degenerative changes typical of a disc herniation and to rule out other causes of the signs. Changes indicative of a disc herniation include narrowing of the intervertebral disc space, narrowing of the intervertebral foramen and the presence of mineralized material within the vertebral canal and disc space. However, a definitive diagnosis cannot be reached with survey radiographs alone with adequate accuracy for surgery to be undertaken and either MRI, computed tomography or myelography are used to identify the site of spinal cord compression. CSF analysis is performed concurrently to rule out an inflammatory disorder.

**Treatment**

**Conservative**
Dogs can be managed conservatively with strict cage rest for four weeks combined with pain relief using anti-inflammatory drugs, opiates and/or muscle relaxants. Judicious use of anti-inflammatory doses of corticosteroids combined with appropriate cage confinement can be attempted if the pain is not responsive to non-steroidal anti-inflammatory drugs. Muscle spasm can also be responsive to gentle massage and hot packing of the neck. Administration of an H2 blocker such as famotidine may help to prevent the development of gastric ulceration. The aim of cage rest is to allow defects in the annulus fibrosus to heal, and resolution of pain does not mean that confinement should be discontinued. If this approach is successful, gradual reintroduction to controlled exercise can be attempted and the owners should be cautioned to prevent their pet from activities that involve jumping in the long term. Dogs should be monitored weekly and if the pain is unresponsive to conservative therapy, recurs, or neurological deficits develop, surgery should be recommended.

**Surgery**
Indications for surgery include unremitting or severe pain, recurrent pain, or neurological deficits. Once the site of disc herniation has been confirmed, a ventral slot is performed to remove the herniated disc material. Adjacent discs are fenestrated to prevent recurrence of the problem. Post operatively dogs are provided with pain relief and confined for four weeks (two weeks of strict confinement and then if doing well, two weeks of increasing controlled exercise). Dogs are then gradually re-introduced to normal activity. If the dog has neurological deficits, postoperative care includes performing passive range of motion exercises, massage, hydrotherapy and controlled exercise.

**Prognosis**
Prognosis for dogs treated conservatively is unknown. Prognosis for dogs treated surgically is excellent unless neurological deficits are severe.
Five Seizure Imposters: The Most Common Events to Consider Which Mimic Seizure Events
Simon Platt, BVM&S, MRCVS, DACVIM, DECVIM
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Athens, GA

There are several broad categories of disease or abnormality that should be considered when determining whether a paroxysmal event is a manifestation of a seizure disorder or not. These include neuromuscular disorders leading to collapse (e.g., Myasthenia gravis), cardiovascular disease causing syncope, sleep related events such as R.E.M. sleep disorder and narcolepsy/cataplexy as well as a new defined group of disorders of involuntary movement that are predominantly breed related. Obsessive compulsive disorders will also be mentioned based on their stereotypical presentations and similarities to the focal sensory seizures described in veterinary medicine. These disorders will be discussed in terms of the classical presenting signs and how they may be considered differently from epileptic disorders. Absolute confirmation of the epileptic nature can only be obtained by observing simultaneously the characteristic EEG changes and physical manifestation of the seizures.

(A) Neuromuscular collapse
Activity associated weakness is the most typical clinical sign of neuromuscular disease. The interpretation of the neurological examination may be challenging in these patients. At the time of examination, they may appear normal or only mildly affected; additionally if weakness is exhibited, it is rarely specifically indicative of nerve, neuromuscular junction or muscle disease.

In a patient with a neuromuscular disorder, observation and gait analysis may detect ventroflexion of neck, short-strided gait with overflexion of joints (often more evident in the pelvic limbs), a plantigrade and / or palmigrade stance at rest, and generalised decreased muscle tone.

(B) Involuntary movement abnormalities
Paroxysmal events are characterized by the sudden and reversible onset of neurological dysfunction in an otherwise normal animal. Some movement disorders can be paroxysmal. The animals do not lose consciousness and rarely have a structural lesion identifiable within the CNS. The underlying cause of many of these events may be a functional abnormality related to neurotransmitter imbalances or receptor abnormalities and dysfunction. Several stereotypical events have been described in specific breeds and are discussed below. Confirmation of the specific syndrome is difficult or impossible in the clinical setting but depends heavily on the exclusion of structural CNS abnormalities such as neoplasia, inflammation and cerebrovascular disease.

Dyskinesia - is defined as impairment of the power of voluntary movements resulting in fragmented or incomplete movements. Dogs reported with these abnormalities may exhibit abnormal postures such as holding up a limb in an attempt to move or adopting a kyphotic posture of the spine without being able to initiate movement. The pathophysiologic mechanisms underlying these movements are poorly understood, but may represent a central neurotransmitter or pathway abnormality, or possibly a local muscular abnormality. The impaired movement can appear and have been termed muscle ‘cramps’ which are defined as paroxysmal, prolonged and severe contraction of muscles that may be painful and can be either focal or generalized. Examples of diseases associated with cramps which may be dyskinesias include Scotty Cramp, Episodic Falling of Cavalier King Charles spaniels ‘Epileptoid cramping’ of Border terriers, and extreme generalized muscular stiffness in Male Labrador retrievers. Muscle cramps have also been described secondary to systemic diseases such as hypoadrenocorticism.

Dyskinesias are movement disorders that occur spontaneously during activity or at rest causing involuntary contractions of groups of muscles in a conscious animal. The descriptions of these conditions indicate that the most common clinical sign is that of dystonia causing increased muscle tone in one or several limbs, possibly leading to collapse. The movements can be triggered by excitement or exercise. The localization of the purported functional neurotransmitter based abnormalities responsible for these disorders may be central or peripheral nervous system. In general, movement disorders may have origins in the cerebrocortical neurons, basal nuclei or peripheral nervous system.

Idiopathic head tremor/head bob
This head tremor syndrome appears to occur without definable cause in some breeds such as the Doberman pinschers (especially dogs less than 1 year of age), Boxers and Bulldogs, however, a variety of breeds can be affected. These dogs have no other clinical abnormalities and are usually young. Head tremors may be either in an up down or in a side-to-side plane. Sometimes this is referred to as a head bob. Head tremors are usually more prominent when the dog is less active. Also, dogs seem to be able to stop this movement if they desire, are conscious, can walk and can respond to verbal commands. This is almost the opposite of an intention tremor, as the tremor can be stopped when the dog is focused on a goal-orientated task such as eating. The pathogenesis of this disease is not known. In human beings, a nodding of the head can occur with lesions of the thalamus and one of the authors has seen this in a dog with a thalamic lesion. A ‘yes’ head tremor also may accompany midline cerebellar lesions. Full diagnostic workup (blood work,
CSF analysis and imaging of the brain) is normal with the idiopathic condition. There is little information on the most appropriate treatment; although there may be a partial response to antiepileptic drugs, usually they are ineffective. Fortunately, these tremors rarely impact the animal’s quality of life.

Paroxysmal dyskinesias
Paroxysmal dyskinesias are episodes of abnormal involuntary hyperkinetic movement or muscle tone. These events are distinguished from seizures by the presence of a normal consciousness, although an EEG would be necessary to definitively determine this. A movement disorder has been described in young Bichon Frise dogs with an extreme variability of frequency and random occurrence. A rapid muscular contraction causes hyperflexion and or extension of an individual limb. The thoracolumbar spinal column can be affected by altered muscle tone during the event causing a kyphotic posture. A similar condition has also been described in young Boxer pups provoked by excitement causing abnormal facial, truncal and limb movements with sustained hyperflexion.

No successful treatment regimens have been described. It remains to be seen whether a genetic disorder confirms these as truly breed related disorders as documented below. Several drugs have been reported to cause similar dyskinesia and include phenobarbitone and propofol in dogs. These disorders are usually reversible with drug tapering or withdrawal.

(C) Syncope
The term syncope, from the Greek for “cutting short,” refers to an abrupt and transient loss of consciousness accompanied by loss of muscular tone. It is usually caused by a sudden, global reduction in cerebral perfusion, and clinical recovery occurs with restoration of normal cerebral blood flow. The very transience of this syndrome and the variety of medical disorders that can cause or mimic it are at the core of the diagnostic problems that the neurologist faces. The term fainting is often used synonymously with syncope and captures the essential criteria of the collapse - loss of consciousness and muscle tone.

During a syncopal event, the animal usually collapses into lateral recumbency. Stiffening of the limbs, opisthotonic posture, micturition, and vocalization are common, but facial ‘spasms’, persistent tonic/clonic motion, defaecation, a prodromal aura, (postictal) dementia, and neurologic deficits are not usually associated with cardiovascular syncope; however, profound hypotension or asystole can cause hypoxic ‘convulsive syncope’, with seizure-like activity or twitching. Convulsive syncopal episodes are preceded by loss of muscle tone; however, seizure activity caused by underlying neurologic disease is usually preceded by atypical limb or facial movement or staring spells before the loss of postural tone. ‘Presyncope’, where reduced brain perfusion, or substrate delivery, is not severe enough to cause unconsciousness, may appear as transient ‘wobbliness’ or weakness, especially affecting the pelvic limbs.

(D) Narcolepsy / Cataplexy and sleep disorders
Narcolepsy is a disorder of sleep/wake control characterized by a tendency to fall asleep during the day, disturbed night-time sleep patterns and cataplexy. Cataplexy refers to sudden loss of motor tone ranging in severity from a dropped jaw to complete collapse without loss of consciousness and it represents a disorder of rapid eye movement (REM) sleep. Narcolepsy has been reported in many canine breeds, including Doberman Pinscher, Labrador Retriever, Miniature Poodle, Beagle and Dachshund.

The predominant sign in dogs and cats is cataplexy but excessive daytime sleepiness and fragmented sleep patterns have also been reported. Cataplexy is characterized by paroxysmal attacks of flaccid paralysis without loss of consciousness and may last up to 20 minutes, with a sudden return to normality. The event is not accompanied by faecal or urinary incontinence, salivation or rigidity of muscle groups. The episodes, which may occur multiple times a day, are frequently induced by excitement such as eating or playing and they can be reversed by verbal or tactile stimuli. Cataplexy has been recorded in puppies and adult dogs but usually begins in the first 6 months of life with the establishment of REM sleep.

R.E.M. sleep disorders
Normal sleep is divided into 2 stages called non-rapid eye movement, the first stage of sleep lasting 20 minutes and rapid eye movement (REM) sleep. During non REM sleep there is a decrease in body temperature, heart rate and respiratory rate and the animals are immobile but retain muscle tone. REM sleep lasts for about 15 minutes during which animals have an increase in body temperature, heart rate and respiratory rate coincident with the eye movements and atonia of the postural muscles. Normal movements seen during this phase can include twitching of the eyelids, face, larynx and paws with occasional rhythmic paddling of all four limbs and yelping.

(E) Compulsive behavioural disorders
In dogs and cats, behaviors such as ‘fly biting’ and tail chasing have commonly been considered symptomatic for seizure disorders, although treatment with anti-epileptic medications may not be successful. These abnormal behaviors in companion animals have also been considered homologous to the stereotypic behavior of livestock and zoo animals. Such behaviors share similarities with human obsessive compulsive disorder (OCD), and have been referred to as OCD or compulsive disorders (CD). Obsessive compulsive behaviors in people include repetitive behaviors, such as hand washing, rituals, checking, arranging / ordering, counting and hoarding.
and are accompanied by intrusive thoughts, such as concern of contamination; concern for symmetry; fear of harm; aggressive, religious, or sexual thoughts; or pathologic doubt. Interestingly, the intrusive thoughts (obsessions) and the associated behaviors (compulsions) do not necessarily correspond.

The extent of the similarities between the human and canine conditions is not yet known. One similarity is that, overall, the behaviors in companion animals are amenable to the same pharmacologic treatment as are obsessions and compulsions in people. However, there are differences between the human and canine conditions.
Seizure
The term seizure refers to a sudden attack or recurrence of disease and often implies a dramatic or catastrophic event. The term is therefore non-specific although is often used to describe an epileptic seizure. The term is often used interchangeably with convulsion.

Epileptic seizure
An epileptic seizure is the physical manifestation of paroxysmal transient disturbance of central nervous system function resulting from excessive and/or hypersynchronous abnormal neuronal activity within the cerebral cortex.

Epilepsy
Epilepsy is not a specific disease but a chronic condition characterised by recurrent epileptic seizures. A patient having a single epileptic seizure does not have epilepsy, as the seizures are not recurrent.

It is important to recognise that an epileptic seizure is not a disease entity in itself but a clinical sign generally indicative of a forebrain disorder.

Classification of epileptic seizures
Epileptic seizure types can be classified into two major categories: partial and generalised.

Generalised seizures
In dogs, generalised seizures are the most common type. Generalised seizures have no localised signs and indicate involvement of both cerebral hemispheres. Consciousness is impaired and motor manifestations are bilateral.

Partial seizures
Compared to dogs, cats commonly exhibit partial seizures. This type of seizure indicates abnormal neuronal activity in a localised region of the cerebral hemisphere. Any portion of the body can be involved during a focal seizure depending on the region of the brain affected. The focal nature of this seizure type is associated with a higher incidence of focal intracranial pathologic change in cats. The various forms of partial seizures include:

- Focal (partial motor) seizures: unaltered consciousness with asymmetric localised motor signs such as eyelid or facial twitching, clonus of muscle groups of one limb.
- Psychomotor (complex partial) seizures: behavioural seizures pattern involving the limbic system which may present as rage, aggression without provocation, fly-catching, running in circles, floor licking, vocalisation, tail chasing, etc.

A seizure may start in a focal region of the brain only to spread throughout both cerebral hemispheres, resulting in a focal seizure with secondary generalisation.

Confirmation of the epileptic nature of a paroxysmal event
A paroxysm is defined as a sudden recurrence or worsening of the clinical signs - a spasm or a seizure.

The recognition of an epileptic seizure is essentially based on the owner’s description of the event. Although generalised tonic-clonic seizure have a fairly unequivocal description, the recognition of a partial or psychomotor seizure can pose a real challenge for the clinician. For that reason video footage obtained by the owner of the paroxysmal event can be of tremendous help.

An epileptic seizure can be suspected based on:

- The peracute and unexpected (except cases of “reflex seizures”) onset and offset.
- Stereotypical pattern.
- Presence of involuntary motor activity and/or abnormal mentation and behaviour and/or autonomic signs (salivation, urination and/or defecation).
- Elimination of other paroxysmal events (syncope, acute vestibular attack, myasthenia gravis).

Absolute confirmation of the epileptic nature can only be obtained by observing simultaneously the characteristic EEG changes and physical manifestation of the seizures.
**Paroxysmal events that could mimic an epileptic seizure**

<table>
<thead>
<tr>
<th>Non-epileptic paroxysmal events</th>
<th>Criteria for differentiation</th>
</tr>
</thead>
</table>
| Narcolepsy/Cataplexy            | Loss of consciousness and transitory flaccid paralysis  
Can be precipitated by excitement or feeding  
No post-ictal phase |
| Syncope                         | Often associated with exercise or excitement  
Partial or complete loss of consciousness  
No motor activity  
No post-ictal phase  
Associated respiratory or cardiovascular pathology (and eventually clinical signs) |
| Vestibular attack               | Associated signs of vestibular involvement (head tilt, nystagmus, asymmetric ataxia and/or circling)  
No loss of consciousness |
| Myasthenia gravis               | Exercise-induced weakness/stiffness  
Normal mental status  
No post-ictal phase |
| Painful focus                   | Physical examination |

**Classification of epileptic seizures**
The presence of epileptic seizures implies a forebrain disorder. Their causes may originate outside (extra-cranial) or inside (intra-cranial) to the brain. Intra-cranial causes may be further subdivided into functional disorders (where no gross structural changes are evident in the brain) and structural disorders (where there is a gross structural cause within the brain, e.g. a brain tumour or hydrocephalus).

- **Reactive Seizures**: This term is often used to describe seizures resulting from an extra-cranial cause.
- **Primary Seizures**: This term is often used to describe seizures resulting from a functional intra-cranial cause (e.g. Idiopathic Epilepsy).
- **Secondary Seizures**: This term is often used to describe seizures resulting from a structural extra-cranial cause (e.g. brain tumor).

**Overview of medications used for canine and feline seizure control**

**Phenobarbital (PB)**

Phenobarbital is the drug used most commonly by veterinarians, as the drug of first choice for seizure control in dogs due to its low cost and approximately 80% success rate in controlling seizures in epileptic dogs. This drug has been well documented to occasionally have fatal hepatotoxic effects in dogs as well as cause neutropenia. A good slow induction dosage of PB is 2-4 mg/kg/day divided BID or TID. If indicated, the dosage may be slowly increased to as much as 18-20 mg/kg divided BID or TID. Serum PB concentrations should be monitored to assess therapy. A PB serum concentration of 15-45 ug/ml should be achieved immediately prior to each subsequent dosage of medication. It will take 7 to 18 days to achieve a steady state serum concentration with sustained maintenance doses. If dosages of 4 mg/kg/day or higher are used to initiate PB therapy, some dogs will appear depressed, drowsy or ataxic for about one month. This effect then generally resolves, and much higher doses can be given without sedation occurring. Some dogs will be polyuric, polydipsic and polyphagic while receiving PB, especially at higher doses. The serum alkaline phosphatase (AP) and the serum alanine transaminase (ALT) will increase in many dogs maintained on the drug. At least once/year, a PB serum concentration, serum chemistry profile, and haematology should be done on any animal receiving PB maintenance therapy. Any dramatic change in results from one year to the next may signal potential toxicity. This is the drug of choice in cats with multiple seizure episodes. The dose advised is 1.5 to 2.5 mg/kg PO every 12 hours. Due to the formulation of this drug, it is often best to start with 7.5 mg twice daily, which can be increased in 7.5 mg increments as necessary. Polyphagia with weight gain is documented as a frequent side-effect of PB administration in cats. Hepatotoxicity has not been documented in cats on this drug, but cutaneous hypersensitivities and bone marrow suppression have.
Potassium bromide (KBr)
Potassium bromide is becoming the drug of first choice for the management of epilepsy in dogs since it is the only anticonvulsant known that has no hepatic toxicity and all the adverse effects of KBr are completely reversible once the drug is discontinued. KBr controls approximately 80% of the epileptic dogs it is used to treat and is often effective in dogs that fail PB therapy. When high dose KBr and low dose PB are used together, approximately 95% of epileptic dogs can be controlled.

The maintenance dosage of is 20-100 mg/kg/day (which can be divided BID to avoid GI upsets) to achieve serum concentration of 1-5 mg/ml measured just before the next dose is administered. It requires 2 to 3 weeks of therapy before bromide serum concentration will enter therapeutic range and close to 4 months before steady state values are approximated. If seizure control is needed more rapidly than this, a total oral loading dose of 400 to 600 mg/kg of potassium bromide can be given prior to instituting the maintenance dosage schedule divided qid over 4-5 days. By dividing the loading dose, excessive sedation may be avoided in case the dog is especially sensitive to the sedative effects of bromide. The loading dosage should be mixed well with food to avoid the induction of vomiting. Be sure to stress to owners that it is important to keep the salt content of the diet consistent to prevent marked serum concentration fluctuations of bromide.

The most common adverse effect of bromide therapy is polyphagia, and it is recognized in about 25% of the dogs on therapy necessitating changing to a low calorie diet such as canine R/D or W/D to prevent excessive weight gain. Polydipsia and polyuria are less common with KBr therapy than with PB therapy, but these adverse effects are sometimes recognized. Personality changes that can occur are; irritability leading to snapping at people or other animals, seeking constant attention from the owner, aimless pacing behavior, and most commonly, depressed mental level as a result of sedation. Clinical signs of bromide toxicity are sedation, incoordination, and in dogs, pelvic limb weakness and/or stiffness is observed, easily misdiagnosed as pelvic limb stiffness due to osteoarthritis, since specific neurologic deficits are absent. Bromide toxicity can be seen in dogs that have renal insufficiency because the halide ion is excreted by the kidneys. There has been an association made between the use of bromide in cats and the onset of a reversible respiratory disease.

Primidone
Primidone is metabolized in the liver to phenylethylmalonic acid (PEMA) and PB. Phenobarbital levels should be monitored in dogs on primidone as they correlate better with anticonvulsant efficacy than primidone levels. The same side-effects that phenobarbital create are seen with the use of primidone. The target therapeutic ranges are also the same. Primidone is advised for use in those patients who have proven refractory to phenobarbital although its efficacy has not been proven. Otherwise there is no evident advantage of primidone over the use of PB as a first choice AED. The conversion rate of primidone to PB is close to 4:1. Therefore the use of 250 mg of primidone equals the use of 60mg of PB. Conversion from primidone to PB should take place slowly (1/4 of the dose each month). In the dog, the use of this drug has resulted in progressive hepatic injury, which seems to be more common than that seen with PB.

Gabapentin
Gabapentin is a recent addition to the human anti-convulsant market, which has primarily been used as an adjunctive drug for humans with uncontrolled partial seizures with and without secondary generalization. Gabapentin is well absorbed from the duodenum in dogs with maximum blood levels reached in 1 hour after oral administration. The elimination half-life of gabapentin in dogs is 3-4 hours in dogs, meaning that it may be difficult to attain steady state levels in dogs even with tid dosing. The dose at present estimated to be necessary to achieve some effect in dogs is 30 to 60 mg/kg divided tid to qid. It may be that its use in dogs demands higher doses making its expense prohibitive. In dogs, gabapentin is metabolised in the liver, therefore liver function needs to be closely evaluated when dogs are on this treatment; it is excreted nearly 100% through the kidneys, with 60% being the unchanged parent drug. The author has used this drug with no deleterious effects, in addition to PB and KBr. In a study of 11 dogs, 45% demonstrated improved seizure control with success based upon a 50% reduction in seizure frequency. However, many dogs still exhibited multiple days on which there was cluster seizure activity. Forty-five percent (5/11) of the dogs in this study also demonstrated sedation and ataxia after the addition of this medication.

Levetiracetam
Levetiracetam was approved in November 1999 as a human add-on therapy for the treatment of partial onset seizures, with or without generalisation, in adults. Studies show that levetiracetam displays potent protection in a broad range of animal models of chronic epilepsy. Levetiracetam is water-soluble, is not metabolized by the liver, is excreted by the kidneys and is free of significant drug-drug interactions. The dose range documented for dogs is estimated to be 10-30 mg/kg q 8hrs PO. Levetiracetam has been documented as the most well tolerated anti-epileptic drug in humans, with adverse reactions equal to that of placebo. Overall, this drug is proven to be a highly effective adjunctive therapy in humans to control seizures refractory to treatment.

Zonisamide
Zonisamide has been shown to be both effective for focal and generalised seizures in people. It is metabolized mainly by hepatic microsomal enzymes, and the half-life in dogs is approximately 15 hours. The dose suggested for use as an add-on drug in dogs is 10 mg/kg q12hrs PO. A high safety margin has been demonstrated with chronic dosing studies in dogs. A recent clinical trial has shown
that the use of this drug has decreased seizure frequency by over 50% in approximately 50% of dogs on polytherapy, additionally enabling a reduction in the concurrent dose of PB. Five dogs had an increase in seizure frequency. Mild side effects (e.g., transient sedation, ataxia, vomiting) occurred in six of the dogs. Nine of 11 idiopathic epileptic dogs refractory to PB and or KBr responded to zonisamide in another study, with a mean of 70% reduction in seizure frequency. As for levetiracetam, seizure control was noted to subside after a couple of months in several dogs on zonisamide.

**Pregabalin**

Pregabalin like gabapentin is a structural, but not functional analogue of the neurotransmitter gamma-aminobutyric acid (GABA). Pregabalin has shown greater potency than gabapentin in preclinical models of epilepsy and pain in people. Pregabalin is active in a number of animal models of epileptic seizures including maximal electroshock-induced tonic extensor seizures in mice and rats, hippocampal kindled rats and threshold clonic seizures from the convulsive agent pentylenetetrazol and genetic mouse models, with a greater potency than gabapentin. There is no protein binding or hepatic metabolism, it is renally excreted with no drug-drug interactions identified. Although a prospective study is currently underway evaluating the use of this drug in dogs with refractory epilepsy, no current data exists on its effects.
Consolidation
Consolidation continues to be a major driver of change in our veterinary world. Pharmaceutical companies, distributors, and individual veterinary practices are all exploiting the cost-saving benefits of merging.

Management groups like VMG, local veterinary medical associations, and buying groups are leveraging their size to bring their membership valuable leadership resources, legal advice, management support, and cost savings. Expect more activity associated with these groups in 2018 and beyond. At the lecture we’ll also discuss how these groups are driving practice values higher.

Consolidated practices have more resources to aim at complex and expensive aspects of modern business like online marketing, new client acquisition, online visibility, and client retention. They’re also able to focus more attention on the difficult business of finding veterinarians, licensed veterinary nurses, and other skilled, experienced support team members. Their efforts are partially impacting the number of veterinary professionals available to work at individual practices and are driving wages up for everyone.

Veterinary practice wage studies done by the VHMA will be shared with attendees at all 2018 Fetch dvm360 conferences, along with commentary on what the data means by VHMA president, Jim Nash.

Some aggregating companies, businesses who are consolidating practices, are offering associate veterinarians unique partnership opportunities that allow them to be business owners, to earn more money, and to use sweat equity as the down payment to their partnership. It’s a way to retain valuable veterinarians and gives young doctors a shot at more income, but without the responsibilities of full practice ownership. You’ll find out more about one such model at the conference.

Distribution and sales
In an effort to reduce costs, manufacturers are deciding whether a direct approach to the consumer through online marketing is right for them. How the debate is going will be discussed at the conference. Inventory management will continue to get smarter in 2018 and more automated. Expect to see your client communication service companies, your management software, your apps, and your online pharmacies gathering more data about your business and your clients and using that information to help you sell your services and products more effectively.

Client connection
Apps continue to trend in 2018 as an effective way to stay connected with clients. We’ll share how practices are using rewards programs, through apps, as a way of incentivizing client behavior like early drop off and compliance to preventative medical services like dentistry.

Veterinary practices are also pulling away from more formalized client care. As a way of bonding better with the people that they serve, some practices are on a first name only basis with their clients, team members are dressing in street clothes, and hospital design and layout is shifting from clinical to comfy. You’ll see examples of this and learn how some practices have completely rebuilt client flow through the practice for efficiency and client connection.

Recruiting and training go digital
There are exciting advances in the way we’ll be finding and training employees of the future.

The latest data shows that our way of finding, interviewing, and hiring professionals is not working. At the lecture, you’ll learn of a digital approach to finding team members that saves time and secures employees that stay with the job.

Too, the VHMA has partnered with ACT to develop a new certificate program in training, available to all practice managers, that helps them to optimally train employees. You’ll be impressed to see this extensive, time saving, online approach to improving your team’s performance.

Payment options
Recent studies show that clients have reached their limit with respect to price increases. In our lecture, we’ll discuss how companies like Vetbilling.com and pet insurance companies are improving our client’s compliance to our recommendations and helping them to finance veterinary services. Companies like CareCredit have also launched initiatives that help new clients find practices that offer CareCredit financial services. More at the lecture.
Marketing
It seems that every year, marketing becomes more complicated and more expensive as everyone turns increasingly online for the best exposure to new clients. At the 2018 classes, we’ll teach you where to focus your marketing energy and money and how to manage the process efficiently. Digital marketing is definitely the way to go in 2018, but unless you know what you’re doing, the digital marketing ‘way to go’ will lead you nowhere.

We’ll do our best to squeeze all of this into our 50-minute lecture. As the conference season nears, keep an eye out for a resource list, available on the Fetch dvm360 website, that will provide you with more information as well as inside contacts at the referenced organizations who will be able to provide you with more information specific to your needs.
Types of gastrointestinal diets

Nutritional management of gastrointestinal disease is a broad topic incorporating both acute and chronic diseases of the stomach, small intestines, pancreas, gallbladder, and colon. As such, the goal of this lecture is to introduce the types of gastrointestinal diets available and help the clinician decide which is best based on their nutritional components. There are four broad categories of diets for managing gastrointestinal disease: low residue, fiber enhanced, low fat, and hypoallergenic. Homemade diets can also be used for managing disease, but vary tremendously based on their formulation.

Low residue diets

Low residue diets can be defined as having protein digestibility >87% and fat and carbohydrate digestibility >90%. These diets typically have refined ingredients and are low in fiber (<3-5% on a dry matter basis). The benefits of low residue diets are they can speed movement of food through the stomach and ease absorption in compromised intestine. Examples of low residue therapeutic diets include Purina EN,1 Hills i/d,2 and most Royal Canin gastrointestinal diets.

Fiber enhanced diets

Fiber enhanced diets may be designed for gastrointestinal disease or diabetes mellitus. Fiber can be categorized based on its solubility in water or its ability to be fermented by intestinal bacteria. Soluble fiber sources dissolve in water to form a thick, viscous gel, which will slow the movement of ingesta through the intestines. Soluble fiber sources also tend to be fermentable and can produce gases and physiologically active byproducts within the intestine. Insoluble fibers do not dissolve in water and tend to have low fermentability. Thus, they can be viewed as metabolically inert and provide bulking of stool and water absorption as they move through the intestine. Insoluble fibers tend to speed up evacuation of feces by stimulating colonic stretch receptors. Ideally, fiber enhanced diets would contain a combination of both soluble and insoluble fibers to provide a balanced effect. Too much of either type of fiber could result in soft stools, constipation, or excessive gas. Also, note that crude fiber listing on the guaranteed analysis of pet food labels do not capture the soluble fiber fraction of the diet and may underestimate total dietary fiber (TDF). Examples of fiber enhanced diets marketed for gastrointestinal disease include Hill’s w/d, Royal Canin Gastrointestinal High Fiber, and Purina DCO.

Low fat diets

Low fat diets typically have fat contents between 18-25 grams/1000kcal. Lower fat diets can be useful for reducing pancreatic stimulation and speeding movement through the stomach in vomiting animals. Examples of low fat diets marketed for gastrointestinal disease include Royal Canin Gastrointestinal Low Fat, Hill’s i/d low fat, and Purina EN low fat. Low fat diets are not appropriate for weight loss because they are not fortified with nutrients to offset low calorie intake. Weight loss diets are also typically lower in fat than standard diets, but the fiber content and calorie density may not be appropriate for animals with poor appetites or vomiting. In addition, weight loss diets usually are not restricted as low in fat as the gastrointestinal low fat diets.

Hypoallergenic diets

Diets designed to reduce symptoms of food allergies can be placed into two broad categories: novel protein or hydrolyzed. Novel protein diets use uncommon protein and carbohydrate ingredients to lessen the chance of exposure and a subsequent allergic response. In humans, most food allergens are glycoproteins that range in size from 14,000 to 40,000 daltons. Proteins within range are large enough to activate B and T cells, but small enough to pass through mucosal membranes and interact with the immune system. Hydrolyzed proteins are low molecular weight peptides (<18,000 daltons) with reduced antigenic potential because they are too small to bind with immunoglobulins.1 As a result, they are less likely to elicit a response from the dog’s immune system. Free amino acids are not allergenic but cannot be used due to their bitter taste and high osmolarity. There are many of hypoallergenic diets on the market. Novel protein diets are available over-the-counter (OTC) or by veterinary prescription. OTC tend to have a higher likelihood of contamination with common pet food proteins as compared to veterinary therapeutic diets.2 Examples of novel protein therapeutic diets include the Hill’s d/d products and Royal Canin Selected Protein diets. Hydrolyzed diets include Purina HA, Hill’s z/d, Royal Canin HP, and Royal Canin Ultamino.

1 Purina, St. Louis, MI
2 Hill’s Pet Nutrition, Topeka, KS
Specific types of gastrointestinal disease

Vomiting

Vomiting is the most common clinical sign of gastric disease. Dietary goals for vomiting are to minimize gastric irritation, promote gastric emptying, normalize motility and prevent gastroesophageal reflux. Fat and fiber delay gastric emptying, so choosing a lower fat, low residue diet is usually ideal to manage vomiting and reflux.

For acute, frequent vomiting food may be withheld for 24 hours. Small, frequent meals (3-6 per day) can also speed passage of food through the stomach.

Small intestinal disease

Acute small intestinal (SI) diarrhea with or without vomiting will often benefit from a low residue diet. As normal digestion and absorption may be compromised. Small frequent meals are recommended and early feeding for the intestines is best.

Inflammatory bowel disease is described as a group of chronic, idiopathic inflammatory disorders of the gastrointestinal tract. Severity can vary from mild to life threatening protein losing enteropathy (PLE). Clinical signs depend on section of bowel affected. Key nutrition factors for IBD include avoiding excessive dietary protein to minimize antigens that elicit an immune response (PLE is an exception and requires high protein), feed a low residue diet as absorption may be impaired, utilize a novel protein and/or hydrolyzed protein diets, and feed small, frequent meals.

Protein losing enteropathies such as lymphangiectasia require a low fat diet. Long chain triglycerides, the most common form of dietary fat, stimulate lymph flow and increase protein leakage through the lymphatic vessels. Lymphangiectasia can be primary, but is often secondary to IBD. A low fat, high protein, low residue diet is desired for these cases. Our nutrition practice usually assumes cases of lymphangiectasia are secondary to IBD unless proven otherwise, and also utilize hydrolyzed or novel protein diets for management. A low fat, low residue diet that is hypoallergenic and high protein is difficult to obtain. Our practice often utilizes Purina Feline HA for our canine patients as it meets most of these criteria. A portion of the fat from this diet comes from medium chain triglycerides (MCT) that passively diffuse from the GI tract to the portal system and partially by pass the lymphatic system. Thus, calorie density is maintained while lowering long chain fatty acids.

Short Bowel Syndrome develops from massive resection of the small intestine and may result in malabsorption due to lack of surface area. Cobalamin deficiency may occur if ileum is resected. These cases benefit from low residue diets. Moderate to high fat, energy dense foods that are low to moderate in fiber are ideal. Supplementation of fat soluble vitamins and cobalamin may be needed and patients should be fed small, frequent meals.

Dysbiosis of bacterial flora in the intestines can lead to diarrhea and may be present in 50% of dogs with diarrhea. Dysbiosis is often associated with exocrine pancreatic insufficiency (EPI). Laboratory evaluation involves a normal to high trypsin mediated component to the disease.

Pancreatic disease

Pancreatitis is the most common disease of the pancreas and can be acute or chronic in nature. Patients with acute pancreatitis patient will benefit from early enteral nutrition. Unless vomiting is uncontrollable with anti-emetics, small amounts of nutritional support should be given as soon as possible. Typically a nasoenteral feeding tube is used and a liquid product enteral product is used. For chronic pancreatitis in dogs the main nutrient of concern is fat. A low fat diet is best and the degree of fat restriction will be dependent on case severity. Protein can also stimulate the pancreas, so high protein diets should be avoided. Cats with acute pancreatitis is can be treated similar to dogs with a low fat, moderate protein diet. Cats with chronic pancreatitis typically do not require diet changes as diet doesn’t appear to be a factor in their disease progression or outcome.

The exocrine pancreas secretes numerous enzymes to digest fats, proteins, and carbohydrates. Animals must lose approximately 90% of their functional capacity before exocrine pancreatic insufficiency (EPI) produces clinical signs. EPI characterized by chronic small bowel diarrhea with steatorrhea and voluminous diarrhea. Patients also have ravenous appetite with weight loss. The best treatment for EPI is supplementation with pancreatic enzymes. Diet modification doesn’t appear to make much difference in these cases. However, absorption of fat soluble vitamins (A,D,E, and K) may be impaired and cobalamin may need to be supplemented until clinical signs are well controlled.

Large intestinal disorders

Colitis is a common disorder with many causes. In general, high fiber diets are helpful with colitis to support the growth of beneficial bacteria and to help with water balance. If a high fiber diet does not improve large bowel diarrhea, a low residue diet may be used to minimize nutrients reaching the colon. Finally, if the diarrhea is not responsive to fiber or high digestible diets a low allergen diet may be used in case there is an immune mediated component to the disease.

Flatulence is an annoyance to pet owners and the dietary goal is to reduce intestinal gas production. Low residue diets with a fat content lower than their current diet may be helpful. Highly fermentable carbohydrate sources should be avoided (beans, cruciferous vegetables). Alpha-galactosidase (Beano) may also decrease flatulence by improving digestion of carbohydrates. Outdoor exercise may also help expel gases in a less offensive environment.
**Prebiotics and probiotics**

Prebiotics are starches and fibers resistant to digestion. Examples include: Fructooligosaccharides (FOS), Mannanoligosaccharides (MOS), Galactooligosaccharides (GOS), Fermentable fibers. Indications for prebiotics include antibiotic-associated diarrhea, traveler's diarrhea, gastroenteritis, normalizing bowel function, colitis, and irritable bowel problems. According to the food and agricultural organization (FAO) and the world health organization (WHO), probiotics are defined as "Live microorganisms which when administered in adequate amounts confer a health benefit on the host." Ideally probiotics should originate in the species being treated, be nonpathogenic, be resistant to digestion by gastric acid and intestinal enzymes, able to adhere to the intestinal epithelium, and be capable of influencing host immune responses. Probiotics can promote normalized microflora and may have role in allergies. They can also help to inhibit binding of pathogenic bacteria. Many products may not contain the numbers of viable bacteria they claim.

**References**


Effective Weight Loss Plans: They Do Exist
Angela Witzel, DVM, PhD, DACVN
University of Tennessee
Knoxville, TN

Estimating needs of patients
The Daily energy requirements (DER) are the average daily energy requirements of an animal. DER can be divided into four parts: 1) Resting energy requirement (RER)-energy needed to maintain normal body functions such as respiration 2) Exercise energy requirement (EER) - energy exerted through muscular activity and exercise 3) Thermic effect of food (TEF) - energy burned through digestion and absorption 4) Adaptive thermogenesis (AT) - energy used to stay warm or cool. We always begin to estimate DER by calculating RER and then adding in a lifestage factor to account for exercise and other metabolic factors. When using the resting energy calculation, it should be based on your patient’s IDEAL weight. So if the dog or cat is overweight, the equation is performed using the weight they should be, not their current body weight.

Calculating resting energy requirements for dogs and cats

\[70 \times \text{Body Weight}_{\text{kg}}^{0.75} \quad \text{or} \quad [(30 \times \text{BW}_{\text{kg}}) + 70]\]

The equation raised to the 0.75 power is more accurate. The linear equation can be used in animals between about 6-60 pounds

Example: 5 kg cat

\[70 \times 5^{0.75} = 70 \times 3.34 = 234 \text{ kcal/day}\]
\[(30 \times 5) + 70 = 150 + 70 = 220 \text{ kcal/day}\]

**Daily energy needs = Lifestage factor x RER**

<table>
<thead>
<tr>
<th>Life Stage</th>
<th>Canine Factor</th>
<th>Feline Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult intact</td>
<td>1.8</td>
<td>1.4</td>
</tr>
<tr>
<td>Neutered</td>
<td>1.6</td>
<td>1.2</td>
</tr>
<tr>
<td>Senior</td>
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<td>1.1</td>
</tr>
<tr>
<td>Obese prone</td>
<td>1.2-1.4</td>
<td>1.0-1.2</td>
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<tr>
<td>Weight loss</td>
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<tr>
<td>Growth 2 - 3</td>
<td>2 - 5</td>
<td></td>
</tr>
<tr>
<td>Gestation</td>
<td>1 - 3</td>
<td>1.6 - 2</td>
</tr>
<tr>
<td>Lactation</td>
<td></td>
<td></td>
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<td></td>
<td>2 - 6</td>
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</tbody>
</table>

In the example above, if the 5 kg cat was a neutered male, you could take the 234 kcal/day you calculated and multiply by a lifestage factor of 1.2 to estimate daily energy needs. 234 x 1.2 = 280 kcal/day.

Consequences of obesity

a. Overweight cats 2.9 times as likely to be taken to veterinarians because of lameness
b. Obese cats
   i. 3.9 times as likely to develop diabetes mellitus
   ii. 2.3 times as likely to develop nonallergic skin conditions
   iii. 4.9 times as likely to develop lameness requiring veterinary care
c. In dogs, obesity increases the incidence of musculoskeletal disease
d. Exacerbates pre-existing cardiovascular and respiratory disease
e. Obesity and lifespan
   i. 48 dogs were paired. One dog was fed 25% less food than its pair mate 8 weeks of age until death.
      1. Median life span was significantly longer for the group that was fed 25% less food - 11.2 years versus 13 years
      2. Lean dogs had less incidence of chronic disease like arthritis
      3. Causes of death were similar

Treating obesity

1) Formulate a weight loss plan
a. Rule out other medical conditions like hypothyroidism and Cushing’s disease
b. Estimate current BCS and body fat %
   i. Either use 5 or 9 point BCS scale
c. Estimate ideal body weight
i. Can use body weight at approximately 1 year of age (depends on breed size)

ii. Can use breed size charts to help with estimation

iii. Average female cat 7-10 lbs, male cat 9-12 lbs

iv. Often based on clinical experience

v. A weight history is helpful. Most animals are close to ideal at the onset of maturity (approximately one year of age).

d. Determine the current caloric intake through dietary history

i. Owners will forget, omit, lie etc.

ii. Food journals are helpful

iii. Most large pet food companies have calorie content on websites

iv. Sometimes impossible to accurately estimate food intake.

e. Calculate desired caloric intake

f. Implement an exercise plan

2) Feeding for weight loss

a. Two categories of weight-loss food

i. Over the counter light and lean products

ii. Prescription weight-loss diets

b. Benefits of weight loss foods

i. When a diet is reduced well below feeding recommendations, deficiencies in protein and nutrients can occur

ii. Weight loss diets are balanced to provide both adequate nutrients and energy restriction

iii. Less calorically dense - the ability to digest and absorb food is inversely proportional to the amount of food eaten

c. Feline weight loss diets

i. Canned foods seem more satiating in cats

ii. “Catkins” diets

1. Low carbohydrate, high protein diets – designed to mimic natural carnivorous diet of cats

2. Dry forms are very dense in calories

3. Canned forms are about average in caloric density

iii. Traditional higher fiber diets

1. Less calorically dense

d. How much to feed?

i. Calculate the resting energy requirements (RER) using one of the following formulas:

\[ 70 \times \text{BodyWeight}_{\text{kg}}^{0.75} \quad \text{or} \quad \left[ (30 \times \text{BW}_{\text{kg}}) + 70 \right] \]

The equation raised to the 0.75 power is more accurate. The linear equation can be used in animals between about 6-60 pounds.

Example:

5 kg cat - \( 70 \times 5^{0.75} = 70 \times 3.34 = 234 \text{ kcal/day} \) or \( (30 \times 5) + 70 = 150 + 70 = 220 \text{ kcal/day} \)

Resting energy requirements should be calculated using IDEAL weight, NOT current weight. You can then calculate a patient’s daily energy requirements by multiplying by a lifestage factor for weight loss. Daily energy needs = Lifestage factor x RER.

ii. Dogs – usually feed at 1.0 to 1.2 times resting energy requirements

iii. Cats – usually feed at 0.8 to 1.0 times resting energy requirements

3) Monitoring

a. Recheck in two weeks to determine if losing weight too fast, too slow, or if there are other concerns

b. **Ideally, weight loss will be 1-2% of body weight per week**

c. Calculation: divide the weight lost by the weight on the last visit. That gives total percent lost. Then divide by the number of weeks since the recheck and multiply by 100.

d. Example: 15 pound dog lost 0.8 pounds in two weeks and now weighs 14.2 pounds.

\[ \frac{0.8}{15} = 0.053 \]

\[ 0.053/2 \text{ weeks} = 0.026 \times 100 = 2.6\% \text{ per week} \]

e. Weigh-in monthly if two-week recheck is good
Feeding Tubes: Who, What, When, and How
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Anorexia is a common occurrence in sick patients and the appropriate nutrition can lead to improved immune function and wound healing, faster recovery rates, and improve a patient’s quality of life. Choosing when and where to place a feeding tube is an important step for treating patients. In general, aggressive nutritional support in the form of a feeding tube or parenteral nutrition should be considered after three days of complete anorexia or about 5 days of partial anorexia (consuming <50% of resting energy requirements). After a patient is rehydrated, a loss of >5% of their previous body weight is a sign of malnourishment. Some laboratory parameters can also be used to assess nutritional status, such as low albumin and elevated creatine kinase, but these are often altered due to surgery and may not change significantly with acute anorexia. However, patients with low protein concentrations are in greater need of nutritional support to prevent further muscle wasting and protein catabolism. Another consideration when assessing the need for nutritional support is a patient’s anticipated length of anorexia. For example, if a patient has been anorexic for three days post operatively, but has a sudden positive change in attitude and alertness, one may decide to wait one more day before placing a feeding tube. In contrast, a surgeon may preemptively place a feeding tube in a historically non-anorexic patient during surgery if they are expected to be anorexic following a procedure. Once the need for nutritional support is established, the route of nutrition must be chosen (see figure 1).

If a patient will not consume enough calories voluntarily to meet their resting energy requirements, a feeding tube should be considered. Enteral feeding using the gastrointestinal tract is always preferred to parenteral (intravenous) nutrition. Enteroctyes in the intestine rely on nutrients within the GI lumen for energy. Therefore, enteral feeding prevents villous atrophy and may reduce the risk of bacterial translocation through the intestines. Enteral feeding also promotes peristalsis and often improves patient appetite. The type of feeding tube chosen is based on expected duration of anorxia, a patient’s ability to undergo anesthesia, and the need to bypass certain regions of the GI tract.

Nasogastric and nasoesophageal feeding tubes
Feeding tubes placed through the nasal cavity can either extend to the caudal esophagus (nasoesophageal tube) or into the stomach (nasogastric tube). The small diameter of the tube (5- to 8-Fr) usually does not elicit gastric reflux; however, nasoesophageal tubes are preferred to minimize this potential side effect. The main advantage to using nasal feeding tubes is that they can be placed with little or no sedation. They are appropriate for patients needing 3-7 days of assisted feeding, but often become irritating to the nasal cavity if used beyond 7 days. The small diameter of nasal feeding tubes limits their use to liquid formulations. To determine if the feeding tube is in the gastrointestinal instead of the respiratory tract, two view chest radiographs should be performed. One can also attach a syringe to the end of the tube and pull back. If there is negative pressure, the tube is probably in the GI tract. You can also place the end of the tube in a cup of water and look for bubbles of air. If a capnometer is available, high levels of CO2 coming from the feeding tube would be indicative of respiratory tract placement. One could also inject 1-2 cc of sterile saline into the tube and watch for coughing.

Esophagostomy tubes
Esophagostomy tubes are simple to place and can be used for several months of assisted feeding. They require heavy sedation or anesthesia for placement, and the most common complication is tube site infection. Esophagostomy tubes are larger in diameter (usually 12- to 19-Fr) than nasal feeding tubes, so most canned foods can be blenderized with water and used for feeding.

Gastrostomy tubes
Gastrostomy tubes (G-tubes) are useful for patients that require long-term assisted feeding or need to bypass the esophagus. Gastrostomy tubes can be placed surgically or percutaneously using an endoscope. G-tubes must also be left in place for a week to 10 days before being removed so that a stoma can be formed. The larger diameter of the gastrostomy tubes allows for a great variety of foods, including dry kibble soaked in water.

Managing feeding tubes
After placing a feeding tube and allowing the patient to recover from anesthesia, one should begin with a few milliliters of room temperature water. If no vomiting occurs, food can be initiated. Most patients with feeding tubes have been anorexic for several days and are prone to diarrhea, ileus, and refeeding syndrome. Therefore, feeding should begin with only 1/3 of their resting energy requirements on the first day, divided into 4-6 small meals. Before each feeding, one should attach a syringe and attempt to aspirate fluid from the stomach. If less than 5cc is aspirated, continue with feeding. If more than 5cc is collected, skip the feeding and consider a promotility agent like metoclopramide. Next, inject 3-5cc of warm water to assure tube patency. Food injected into the syringe should be room temperature or warm and given slowly over 5-10 minutes. If using a nasal feeding tube, constant rate infusions using a syringe pump can also be used. After feeding, flush the tube with 3-5cc of warm water to prevent clogging. If no vomiting or diarrhea
occurs in the first day of feeding, then increase to 2/3 of RER on day two, and achieve full RER on day three. Once a patient is able to tolerate feeding their RER, the frequency of meals can decrease and the volume increase. Avoid giving more than 20mls/kg per feeding in cats and 60mls/kg per feeding in dogs.

**Feeding tube complications**

Using feeding tubes can be rewarding, but they are not without risk or frustration. Complications can be categorized as mechanical, gastrointestinal, metabolic, and infections. Mechanical complications include tube obstruction, dislodgement, or premature removal. To minimize the risk of obstruction, the tube should be flushed well with water after giving food or medications. One should also try and limit medications administered through the tube to liquid preparations if using a nasal or esophageal tube. After a tube is placed, a mark should be made to show where the tube exits the body. This will make it easier to recognize a tube that has dislodged.

Gastrointestinal upset from feeding tubes is a common occurrence and can be minimized by slowly initiating feedings with small, frequent meals and restricting total amount giving for the first few days. Never feed foods that are cold from the refrigerator as this can stimulate vomiting. Liquid enteral products are more likely than canned or dry diet to produce soft stools. Most critical care diets are also high in fat and should be used with caution in patients with fat intolerance. If a patient had gastrointestinal upset prior to tube feeding, care should be aimed at treating the primary disease.

Metabolic complications of feeding tubes stem from the development of refeeding syndrome. Refeeding syndrome is a response to nourishment, particularly carbohydrates, following long periods of anorexia (approximately 5 days) or malnourishment. When a patient is malnourished, their intracellular concentrations of certain electrolytes are diminished due to lack of intake. When a carbohydrate-containing meal is given, insulin is released and glucose enters the cells. This stimulates adenosine triphosphate (ATP) production. Potassium, phosphorus, and magnesium follow insulin into the cells to support glucose metabolism. As a result, blood concentrations of these electrolytes can fall dramatically. Hypophosphatemia is the most common manifestation of refeeding syndrome and levels below 1.5 mg/dl are associated with muscle weakness, respiratory distress, heart failure, hemolysis, and death. Hypokalemia is also associated with muscle weakness, cardiac arrhythmias and death. Hypomagnesemia can also lead to muscle cramps, tetany, seizure, and exacerbate cardiac arrhythmias. Magnesium is essential for potassium uptake into cells and magnesium depletion should be suspected in cases of refractory hypokalemia (Adkins 2009). Patients at risk for refeeding syndrome should have electrolytes measured before providing nutrition. If phosphorus, potassium, or magnesium are below the reference range, patients should be supplemented before giving food. Once a meal is given, a chemistry and electrolyte panel should be repeated within 12-24 hours. One should also check a packed cell volume to look for red blood cell hemolysis and monitor for hyperglycemia. Refeeding syndrome typically occurs within the first 24-48 hours, but may take up to 4 days to manifest clinically.

Infectious complications can occur with any type of feeding tube. Nasal feeding tubes that are misplaced, dislodged, or that exacerbate vomiting can result in aspiration pneumonia. Cellulitis and abscess formation at the incision site of esophagostomy or gastrostomy tubes is a common occurrence. This risk can be minimized by not securing tubes too tightly to the body wall and keeping the site clean and protected. In some cases, the tube may have to be removed to resolve infections and cellulitis. Septic peritonitis a risk in patients with gastrostomy tubes if the tube is dislodged before a stoma can form. The risk of peritonitis is higher in patients with percutaneously, rather than surgically placed tubes. It is important in percutaneous tubes that the button or balloon of the tube is large enough to secure the tube within the lumen of the stomach. The exterior of the tube should also be well secured and protected from the animal. A body wrap or e-collar may be needed.
Many consumers desire commercial pet foods that are less processed and closely mimic the diet a pet would eat in its natural environment. For this reason, pet food manufacturers have developed and marketed foods under the categories such as natural, organic, grain-free, holistic, human grade, and raw.

**Natural diets**
The Association of American Feed Control Officials defines the term “natural” as “A feed or ingredient derived solely from plant, animal or mined sources, either in its unprocessed state or having been subjected to physical processing, heat processing, rendering, extraction, hydrolysis, enzymolysis or fermentation, but not having been produced by or subject to a chemically synthetic process and not containing an additives or processing aids that are chemically synthetic except in amounts as may occur unavoidably in good manufacturing practices.”

While pet owners often envision the term natural to be synonymous with unprocessed, it is clear from the definition above that this is not the case. As a result, many owners may choose and pay a premium to feed a diet marketed as “natural” that has similar manufacturing practices as a diet without such a marketing claim.

**Organic diets**
Pet food companies can advertise the term “organic” if they meet the requirements set forth by the USDA National Organic Program. Note that natural and organic are not interchangeable terms.

**Holistic diets**
The term “holistic” has no AAFCO definition and is unregulated with regard to pet food. Any pet food could use the term “holistic” in marketing their product. The term currently has no meaning in pet food.

**Human grade**
AAFCO defines human grade as the following: Every ingredient and the resulting product are stored, handled, processed, and transported in a manner that is consistent and compliant with regulations for current good manufacturing practices for human edible foods. This definition only deals with the storage and production of the food, not the ingredients themselves.

**Grain free diets**
The term marketing term “grain free” has no official AAFCO definition. Grain free is not synonymous with carbohydrate free. Most grain free diets use potatoes, peas, or tapioca as carbohydrate sources. These diets also vary greatly in their protein, fat, and carbohydrate contents. Those that are low in carbohydrates tend to be high in fat and calories, so weight can be an issue for some animals. It is difficult to assess the impact of grain free on food allergies. Wheat, but not corn, is reported in dogs to be a common allergen. However, the alternative carbohydrate sources used in grain free diets also have allergenic potential and increased feeding of these foods may cause more dogs and cats to develop allergies to these sources.

**Raw diets**
The desire to feed raw meat-based diets has risen tremendously in recent years. It is a nutritional philosophy of feeding domesticated dogs and cats as if they were still in the wild and the concept that processing food and ingredients degrades them and decreases their nutritional value. In the wild, prey that is caught by hunting is consumed immediately by predators. Harmful bacteria have little time to grow and cultivate in the flesh consumed. Despite that, it is common for dogs and cats that hunt and eat prey to become infected with parasites. Farming and manufacturing processes also cause the raw meat in found in grocery stores to be contaminated with harmful pathogens, which is why consumers are always advised to cook meat before consumption.

Despite many anecdotal claims, there have been no scientifically documented advantages to feeding raw meat diets to dogs and cats. There are several classes of raw food diets available and I tend to think of them like a Clint Eastwood movie...

**The good**
If a pet owner has a strong desire to feed their pet a raw food diet then a commercial raw diet that has undergone high-pressure pasteurization is highly recommended. High-pressure pasteurization occurs at a cold temperature so the meat is not cooked but pressure is increased above the point that bacteria can survive. This process is a great way to decrease the risk of bacterial contamination of raw food.
The bad
Assuming they meet AAFCO nutritional standards, commercially available raw food diets have the benefit of being complete and balanced. However, unpasteurized raw food diets carry a serious risk for bacterial contamination, not only of the food and the pet, but of the people and household. Though some pets do not become sick when infected with Salmonella, they can shed it in their saliva and feces, which means that their entire body may be contaminated with bacteria. All humans are at risk for Salmonella toxicosis, but it can be a fatal disease in young children, elderly, and immunosuppressed individuals. Other bacteria that may be found in raw foods include Campylobacter, Clostridium, Escherichia coli (E. coli), and Listeria monocytogenes. Commerically prepared raw diets may meet the nutritional requirements of your pet, but they can still result in the spread of harmful bacteria to humans and may make your pet sick.

The ugly
Aside from the health risks associated with consuming raw meat, home-made raw food diets are often nutritionally incomplete and unbalanced. Out in the wild, dogs and cats consume the entire prey, including the organs where many nutrients are found. Raw food diets that are not formulated for sources of fat, calcium, and other required amino acids and vitamins cause pets to become at serious risk for malnutrition. Other risks include esophageal obstruction or tearing from consumption of raw bones.

Homemade diets
Homemade diets are also growing in popularity among dog and cat owners. Home cooking is often more expensive and time consuming than feeding commercial pet foods. In addition, the vast majority of recipes available online and in books are not complete and balanced. It is highly recommended that pet owners wanting to prepare a homemade diet for their sick are referred to a veterinary nutritionist (many provide telemedicine consultations). A reputable source for homemade diet recipes for healthy pets is [www.balanceit.com](http://www.balanceit.com).

The use of homemade diets can be a clinically useful tool when developed in conjunction with a board-certified veterinary nutritionist ([www.acvn.org](http://www.acvn.org)). Examples of cases that may benefit from home cooking include elimination diets for food allergies, increasing palatability for sick patients, and managing disease combinations that do not have a commercially available option.

Vegetarian diets
Ethical beliefs are a common reason people adopt a vegetarian or vegan lifestyle and eliminate contact with animal products fed to their own dogs or cats. There are also certain diseases, including urinary stones, hepatic encephalopathy, and food allergies, that may benefit from a vegetarian prescription diet. However, there is concern about the health and safety of some vegetarian and vegan diets, especially for cats. Cats are obligate carnivores and require animal fats and protein in their diet to receive essential nutrients to thrive. Due to their anatomic, physiologic, biochemical, and enzymatic make-up, dogs and cats are not well adapted to an all-plant based diet.

Since some essential nutrients are limited in plant material, the risk of nutritional deficiencies increases when animal tissues are eliminated from the diet. In order for a vegetarian or vegan diet to be considered balanced and nutritious, many nutrients that are normally found in animal protein need to be added to the diet. Another concern when considering any commercial diet is whether or not the food actually contains all of the nutrients claimed on the label. A recent study evaluated 24 commercially available vegetarian dog and cat food to determine if labeling requirements were met and if the diets contained the minimum required amount of protein and essential amino acids, which are the building blocks of protein [1]. Only 8 diets met all labelling requirements and actually contained the minimum amount of protein and essential amino acids required by dogs and cats. Of the diets examined, 3 were therapeutic diets that required a prescription from a veterinarian to obtain. All three of these diets met labeling and nutrient requirements. A similar study evaluated two commercially available vegan diets for cats for nutritional adequacy. Unfortunately, both diets were deficient in multiple essential nutrients and could not be recommended as the sole source of nutrition for cats.[2]

Based on these studies recommending a vegetarian or vegan diet comes with a set of precautions that must be considered. Recommending a therapeutic vegetarian diet may provide more nutrient oversight and better quality control measures compared to less well-established brands.

Ketogenic diets
Ketogenic diets refer to the concept of feeding low carbohydrate diet to induce a state of ketosis. A major difference in metabolism between dogs, humans, and rodents is that it is difficult to induce a ketogenic state in dogs. To induce ketogenesis dogs typically have to eat 80-90% long-chain fatty acids on a metabolizable energy basis. This level exceeds the National Research Council safe upper limit for adult dogs and can reduce diet acceptance [3]. Although feeding medium chain triglycerides promotes more ketone production, the term ketogenic for low carbohydrate diets in companion animals isn’t always appropriate. Nonetheless, the concept of feeding low carbohydrate diets for certain medical conditions is gaining popularity, especially in the management of cancer.
Tumor cells typically have glucose metabolism rates up to 200 times higher than those of their normal tissues of origin. While normal cells undergo oxidative phosphorylation in the presence of oxygen and anaerobic glycolysis when oxygen is low, tumor cells consistently undergo aerobic glycolysis. This process is inefficient in its ATP or energy production compared to oxidative phosphorylation and produces high levels of lactate while also requiring high concentrations of glucose. In theory, eating a diet low in carbohydrates may limit glucose available to tumor cells while providing higher levels of fat and protein for the host to metabolize and use for energy. Another mechanism by which carbohydrates may promote tumor development is through increased insulin release. Insulin has been shown to stimulate cancer cell development and may also stimulate cancer mechanisms such as proliferation, protection from apoptotic stimuli, invasion and metastasis. While it is theoretically possible that a very-low-carbohydrate diet could help to reduce the progression of some types of cancer, present evidence is only preliminary. In the 1980s, seminal animal studies by Tisdale and colleagues demonstrated that a ketogenic diet was capable of reducing tumor size in mice [4]. Whether reducing carbohydrate intake prevents or slows the progression of cancer in dogs and cats remains to be seen.

**Summary**

Diet trends in our pets often follow similar trends to the human nutrition world. Because pet food manufacturing is a big business, marketing claims often take precedent over nutritional science in the choices of consumers. As veterinarians, it is important that we are aware of which diet trends are potentially harmful or beneficial and help direct our clients to the best diets for their pets.

**References**

What You Can and Can’t Learn From Pet Food Labels
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The Association of American Feed Control Officials (AAFCO) is an organization that establishes the regulations and policies for pet food. However, AAFCO does not have any legal authority over pet food. State feed control officials and the Food and Drug Administration (FDA) are charged with maintaining the safety and quality of pet foods within the United States. In addition, the United States Department of Agriculture (USDA) regulates the processing of animals for slaughter and meat and the National Organic Program.

AAFCO and FDA labeling requirements of pet food require the following components:

- Product name and brand name
- Species the food is intended to feed on the principal display panel (PDP)
- Quantity statement on the PDP
- Guaranteed Analysis including minimum percentage of crude protein, minimum percentage of crude fat, maximum percentage of crude fiber, and maximum percentage of moisture
- Ingredient statement with ingredients listed in descending order by weight
- Nutritional adequacy statement
- Feeding directions
- Statement of caloric content in terms of metabolizable energy or as fed basis as kcal/kg and kcal per familiar household measure (e.g., cup, can, treat, piece)
- Name and address of the manufacturer or distributor

Nutritional adequacy statement

The nutritional adequacy statement helps the consumer know whether the food is complete and balanced nutritionally. This statement can be determined in one of 3 ways:

1. The product completes and AAFCO feeding protocol for the intended life stage. This means the product has been fed to dogs or cats for one or more life stages under strict guidelines and found to meet a minimal standard.
2. The product is formulated to meet the nutrient requirements for the intended life stage established by the AAFCO nutrient profile. This means the nutrient profiles were calculated from the ingredient list or a chemical analysis of the food was done and compared to the AAFCO profiles for the species and life stage(s).
3. The product is nutritionally similar to a lead product that completes an AAFCO feeding protocol for the intended life stage. This method uses the same nutritional adequacy statement as a product that has completed an AAFCO feeding protocol.

Some chews and bones including are exempt from AAFCO label requirements unless the manufacturer makes a claim of nutritional value on the label. However, even these treats must meet FDA label requirements, including the name and location the manufacturer or distributor, proper product identification, and the listing of the ingredients by weight. Other types of commercial treats must state that the product is a treat or snack on the PDP.

Pet food quality assessment

Assessing the quality of a pet food from the label is nearly impossible. The guaranteed analysis provides no information regarding quality or digestibility of the diet. You also get very little information from ingredient lists. Labels list ingredients in descending order according to pre-processing weight. However, a specific component of the diet may come from two different sources, which would be listed further down on the list. For example, if a company uses only one type of corn ingredient, it is likely to be listed in the top two or three of the ingredients list. However, if you use different forms of corn such as “whole corn”, “corn gluten meal”, and “rolled corn”, then these ingredients can be listed further down in the list because they would be present in lesser amounts despite the fact that the diet is in reality “corn-based”.

Though there are many limitations, the nutritional adequacy statement is the best way to assess pet food quality based on the label alone. This statement ensures that if this product is fed as the sole source of nutrition, it will adequately sustain an animal during maintenance, pregnancy, or lactation, or will promote growth. It will include wording such as “complete and balanced”, the life stage or life stages for which the food is intended (adults, pregnancy, lactation, growth), and how the product was found to be adequate.

Another method for assessing the quality of a pet food is to call the company and ask a few simple questions:

1. Does the company employ a full-time qualified nutritionists?
2. Who formulated the company’s foods and what are his/her credentials?
3. Are the diets tested using AAFCO feeding trials or by formulation to meet AAFCO nutrient profiles? If the latter, do the diets meet AAFCO nutrient profiles by formulation or by analysis of the finish product?
4. Where are the diets produced and manufactured?
5. What specific quality control measures does the company use to assure the consistency and quality of the ingredients and the end product?
6. Can the company provide an average/typical nutrient analysis for the dog or cat food in question or their lead product? This is different from the guaranteed analysis which is only minimums and maximums.

Companies should freely provide the information above and those who refuse to discuss the questions or provide nutrient profiles should be viewed with suspicion. As another resource to pet owners, AAFCO has a comprehensive consumer friendly website to address pet owner questions related to pet food (talkspetfood.aafco.org).

While determining the quality of a pet food is difficult, there have been recent improvements in the safety and quality control standards for pet food manufacturing. The FDA Food Safety Modernization Act (FSMA) signed into law in January of 2011 is aimed at preventing foodborne illness in both human and animal foods sold in the US. For animal food, it ensures that manufactures establish current good manufacturing practices (cGMPs) and preventive controls. While most manufacturers must be in compliance with these regulations by September, 2017, small companies have until September, 2020 to meet all regulatory standards.

References
Fatty acid nomenclature
A FA (FA) is a hydrocarbon chain with a carboxyl group (COOH) on one end and a methyl group (CH₃) on the other end. FAs are categorized based on length and bonding structure. Short-chain FAs contain less than 6 carbons, medium-chain FAs have 6-12 carbon atoms, long-chain FAs have 13-31 carbons, and very long chain FAs have more than 32 carbon atoms. Saturation is a term used to describe the pattern of single and double bonds between carbon molecules within the FA chain. Saturated FAs do not have double bonds, which allows the carbons to be “saturated” with maximal number of hydrogen atoms. Unsaturated FAs contain double bonds that prevent maximal saturation with hydrogens. Unsaturated FAs are further categorized based on the number and location of their double bonds. As the name implies, monounsaturated FAs contain only one double while polyunsaturated FAs, also called PUFA’s, have multiple double bonds between carbon molecules. Omega-3 and omega-6 fatty acids are types of PUFA’s. Omega 3 or n-3 means the first double bond occurs on the third carbon from the methylated end (aka. omega end) while omega 6 or n-6 means the first double bond occurs on the 6th carbon from the methylated end.

Important dietary FAs
Essential fatty acids are those fats that cannot be synthesized in sufficient quantities to meet the metabolic demands of an animal and must be obtained in the diet. For dogs and cats, the omega-6 fatty acid linoleic acid. Cats also require arachidonic acid, another omega-6 FA. Alpha linolenic acid (ALA) is an essential omega-3 fatty acid. Docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) are important omega-3 fatty acids for therapeutic use. Traditionally these fatty acids have not been considered essential, but more recent evidence shows DHA, in particular, is required for neural development. Therefore, most consider these fatty acids to be essential during pregnancy and growth. In addition, the National Research Council includes minimal dietary intake requirements for DHA and EPA in their recommendations for both dogs and cats. To summarize, the following fatty acids should be included in the diet of dogs and cats:
- Linoleic acid
- Arachidonic acid (cats)
- Alpha linolenic acid (ALA)
- Docosahexaenoic acid (DHA)
- Eicosapentaenoic acid (EPA)

Alpha linolenic acid is found in high concentrations in some plants such as flaxseed. Many commercial products will advertise high concentrations of omega-3 fatty acids based on the ALA content. There is minimal conversion of ALA to EPA and DHA (<10% in dogs and cats). Therefore, if one wishes to achieve the therapeutic benefits of EPA and DHA, supplementation with high concentrations of ALA is a poor strategy.

Therapeutic use of fish oil
The highest dietary concentrations of EPA and DHA are found in marine-based animals. These fatty acids are synthesized by algae and consumed up the food chain. Common sources of EPA and DHA supplements include algae, krill, and fish oil (salmon, sardine, menhaden, and anchovy).

EPA and DHA reduce inflammation by competing with omega-6 fatty acids in the production of leukotrienes and prostaglandins. Omega-6 FA tend to produce pro-inflammatory end-products while omega-3 FA produce less inflammatory products. The anti-inflammatory properties of EPA and DHA have been studied in several disorders within veterinary medicine.

Inflammatory skin disorders
In a crossover study of 16 dogs with idiopathic pruritus, atopy, or flea allergy supplementation with EPA and DHA significantly improved pruritus, alopecia, and coat character compared to control.¹ A randomized, double blind, placebo-controlled clinical trial in 60 dogs with atopic dermatitis found supplementation with fish oil and borage oil (omega-6 FA supplement) reduced prednisolone dosage when compared to placebo (mineral oil).² The steroid-sparing effect was delayed, taking approximately 2 months to be significant. Omega-3 FA supplementation was also found to reduce cyclosporine dosages in a randomized, double-blind, placebo-controlled study of 36 dogs with atopic dermatitis. The median daily cyclosporine dosage was reduced by 45% when supplemented with high concentrations of omega-3 fatty acids (ALA, EPA, and DHA). Pruritus scores were also improved in the supplemented group compared to placebo (mineral oil).³
Cardiovascular disorders
Research indicates that dogs with heart failure have low concentrations of EPA and DHA. In dogs with heart failure due to dilated cardiomyopathy, fish oil treatment reduced the inflammatory markers PGE2, IL-1, and reduced cachexia when compared to placebo. In addition, fish oil had antiarrhythmic effects and reduced atrial fibrillation in dogs with experimentally induced cardiac pacing. Boxers with right ventricular cardiomyopathy had reduced frequency of ventricular arrhythmias when supplemented with fish oil.

Renal disease
Fish oil administration in dogs with experimentally induced chronic kidney disease has been shown to reduce proteinuria, prevent glomerular hypertension, and reduce proinflammatory eicosanoids.

Osteoarthritis and joint health
Several studies show that dogs and cats with joint disease benefit from supplementation with omega-3 fatty acids. When 127 client-owned dogs with osteoarthritis were fed a high or low omega-3 diet for 6 months in randomized, double-blinded study, owners reported improved ability to rise and the ability to walk. However, veterinarians did not notice clinical differences. A randomized, double-blinded trial evaluating 38 client-owned dogs with osteoarthritis for three months found significant improvements in assessments by veterinarians, along with lameness and weight-bearing scores, for the treatment group. Force-plate analysis also indicated a significant improvement in weight bearing for the treatment group. Cats with osteoarthritis also seem to benefit from fish oil supplementation. In a 10-week randomized, double-blinded, placebo–controlled, cross-over designed study of 16 cats with osteoarthritis, those supplemented with fish oil had higher activity levels, more walking up and down the stairs, higher jumps, less stiffness and more owner interactions.

Hyperlipidemia
While initial treatment of hyperlipidemia should include a low-fat diet, fish oil supplementation may be used as adjunct therapy. In humans, omega-3 fatty acids can reduce serum triglyceride concentrations by up to 50%. Fish-oil supplementation significantly reduced triglyceride concentrations in a study of young, healthy dogs. This provides some evidence that fish oil may helpful in the treatment of canine hypertriglyceridemia.

Cancer
Tumor cells are more sensitive to oxidative damage than normal cells. DHA undergoes peroxidation easily, resulting in oxygen radicals. When combined with chemotherapies that are pro-oxidant, this may help kill tumor cells. In one study, high DHA concentrations led to longer disease-free intervals and survival times in dogs with lymphoma.

Fish oil dosage
While there is some evidence to support supplementation of fish oil for certain medical conditions, actually dosing fish oil appropriately can be confusing. Most medical condition appear to benefit from combined EPA and DHA dosages of approximately 70 mg/kg. Studies evaluating osteoarthritis often use dosages closer to 140 mg/kg. A typical fish oil capsule at the drugstore will often market the capsule as 1000 or 1200 mg. This amount typically reflects the entire capsule, not the dosage of EPA and DHA. Regular strength human fish oils usually contain about 250-350 mg of EPA and DHA combined.

As with all nutritional supplements, care should be taken to use high quality products. Companies recommended by the UTCVM nutrition service include Nordic Naturals, Ascenta, Nature Made, and GNC Triple Strength. Many of these companies make highly concentrated products that can make dosing easier.

Therapeutic diets designed for osteoarthritis and joint disease (jdl, JM, JS) already contain therapeutic levels of EPA and DHA and do not need additional supplementation. As more therapeutic diets are developed and fish oil becomes more prevalent, one should review company product guides to avoid over-dosing fish oil. In general, combined EPA and DHA concentration between 1.0-1.2 grams/1000kcal will provide anti-inflammatory dosing, depending on calorie intake. Target ranges for osteoarthritis are about double this amount.

Adverse effects of fish oil
The most common clinical side effect of fish oil supplementation is gastrointestinal upset. Undigested FAs act as a substrate for bacteria and can cause secretory diarrhea. Therefore, one should start with low doses and gradually work up to higher doses over several weeks.

While high doses of fish oil may reduce platelet aggregation, most studies in dogs and cats have not detected appreciable changes in clotting ability at the dosages listed above. Wound healing is dependent on the presence of inflammation. Those study results are mixed, high doses of omega 3 FAs may reduce wound healing and high doses should probably be avoided in perioperative periods.

EPA and DHA undergo peroxidation easily, resulting in free oxygen radical formation. Since vitamin E is the main antioxidant for lipid peroxidation, dietary vitamin E requirements can increase with high concentrations of dietary PUFAs. Signs of vitamin E deficiency include muscle weakness, retinal degeneration, and steatitis.

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Medium chain triglycerides
Most medium chain fatty acids are transported via the portal vein directly to the liver and are not required to form micelles within enterocytes. Once they arrive at hepatocytes, they are metabolized into carbon dioxide, acetate, and ketones.

Seizure disorders
Ketones have long been known to suppress seizure activity, though the exact mechanism remains unclear. Hypotheses include 3-hydroxybutyrate having structural similarities to the neurotransmitters glutamate and GABA, acetone has direct anticonvulsant activity, and that ketones promote higher cerebral uptake of leucine. The therapeutic use of MCT has been garnishing more attention lately, particularly in brain-related disorders. MCT's produce more ketones per gram than long-chain triglycerides. This allows diets with MCT to induce ketosis while maintaining higher protein and carbohydrate content than the classical ketogenic diets utilizing primarily long-chain fatty acids. MCT-ketogenic diets in children reduce the frequency of seizures in children with idiopathic epilepsy. A 6-month prospective, randomized, double-blinded, placebo-controlled cross-over study in 21 dogs with presumed epilepsy found a diet high in MCT (10% metabolizable energy) significantly lowered seizure frequency (2.31/month, 0.11) compared to placebo diet (2.67/month, 0.33-22.92/month, P=0.020). While this study showed some degree of efficacy, the amount of MCT oil utilized is much lower than what is currently used to treat epileptic children and more research is needed to determine the optimal dosage in dogs. In addition, MCT oils have not been evaluated as a monotherapy for epileptic dogs.

Cognitive disorders
MCT supplementation to improve cognitive function has also been investigated in dogs. A 6-month study using 24 older beagles (9.79 years (SD 0.84)) found that dogs fed a diet containing approximately 10% metabolizable energy from MCT had significantly better performance on most cognitive test protocols compared to the control group, with more difficult tasks showing greater effects.

Coconut oil
Coconut oil has been touted as a panacea for many ailments. The composition of coconut oil is almost 90% saturated fats, with MCT making up about 63% of the total fat. Most studies evaluating the therapeutic use of MCT have utilized a combination of caprylic and capric acid. Coconut oil contains only about 7% caprylic acid and its primary MCT is Lauric acid. Therefore, the results of studies utilizing MCT should not be extrapolated to coconut oil. Currently, no published studies evaluating the therapeutic use of coconut oil have been published for dogs and cats.

Adverse effects
The primary adverse effects of MCT administration include gastrointestinal upset and reduced palatability and/or feed intake.

Summary
The anti-inflammatory FAs EPA and DHA found in marine algae and animals are great for a variety of diseases. Standard dosage is one regular strength capsule per 10 pounds (about 300mg/10 pounds or 70 mg/kg of EPA + DHA). Osteoarthritis dose is double the standard dose. Begin fish oil supplementation slowly to avoid diarrhea.

References


Are These Drugs Legal in this State?  
Chemotherapy  
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Learning that a pet has cancer is a difficult experience. After the cancer diagnosis, caregivers are often stressed, anxious, afraid or overwhelmed and at times the word chemotherapy is scarier than cancer.

Recent advances in veterinary oncology have changed the way we look at the outcome of treating a patient with cancer. Less emphasis is placed on a cure at all costs and more emphasis is placed on tumor control, such that the patient can live with cancer as a chronic disease (e.g. like osteoarthritis or diabetes). Knowing how chemotherapy works and what to expect from the treatments can help pet owners decide on whether or not to pursue therapy and/or what might be best for their pets. In addition, the decision of whether to pursue chemotherapy treatments requires knowledge of the biologic behavior of the tumor, geographic constraints, the pet’s temperament and concurrent illnesses, and the caregivers’ time and financial commitment.

When do we use chemotherapy?
The primary goal of cancer therapy is to maintain or improve the animal’s quality of life. The secondary goal is to stabilize, decrease, or eliminate the neoplastic process. Chemotherapy may be used alone or in combination with other treatment modalities, such as surgery and radiation therapy.

Chemotherapy is recommended for the following patients:
- Induction therapy for advanced or multicentric disease
- Adjunctive therapy for micrometastatic disease
- Primary or neoadjuvant therapy for unresectable disease or to decrease tumor burden prior to surgery
- Palliative therapy for alleviation of clinical signs

How does chemotherapy work?
Chemotherapy acts on rapidly dividing cells (tumor/cancer cells, epithelial cells, mucosal cells (GI tract), and bone marrow). These agents may damage a cell's genetic material (DNA) or affect a cell's ability to synthesize protein. However, chemotherapeutic drugs cannot distinguish between malignant cancer cells and normal cells. Fortunately, these normal tissues continue to grow and repair themselves, so the injury caused by chemotherapy is rarely permanent.

What are the typical side effects of chemotherapy?
Chemotherapy is generally very well tolerated in our patients vs our human counterparts. Chemotherapy is administered at lower dosages and frequencies in an attempt to minimize/prevent side effects. All chemotherapeutics have the potential to cause "bag" effects - - bone marrow suppression, alopecia and gastrointestinal signs as these tissues are the most sensitive to chemotherapy. However, these signs only occur in ~10% of patients. Keeping in mind that any animal can have an unexpected reaction to any medication.

Bone marrow suppression may be detected around days 7-10 following chemotherapy administration; therefore, a complete blood count is usually recommended 1 week following chemo administration. Suppression of the bone marrow may lead to a significant drop in the white blood cell count (nadir) resulting in increased susceptibility to infection. The infection usually comes from the animal's own body (such as bacteria normally found in the intestines, mouth, skin, etc.). Severe infections are uncommon but may require hospitalization for intensive supportive care, including intravenous fluid and antibiotics. Most patients are treated as outpatients with oral antibiotic therapy if the white blood cell count is low but the pet is feeling well. Modifications to subsequent doses are made in either scenario.

Alopecia does not typically occur in our patients, but areas that are clipped for surgery, catheters, etc can take longer to grow back. Wire-haired or non-shedding breeds are more susceptible to alopecia during chemotherapy. Hair loss is generally most evident on the face and tail. Whiskers and the long hairs over the eyes often fall out in cats. The hair will regrow once chemotherapy is stopped, but cosmetic changes such as color and texture have been reported.

Gastrointestinal signs such as vomiting, anorexia or diarrhea can occur approximately 3-5 days following chemotherapy administration. Most signs are mild and self-limiting; however, there are times that oral medication is necessary. In rare instances, the gastrointestinal intestinal signs are severe enough to warrant hospitalization and supportive care.

Some chemotherapeutic agents have unique toxicities (e.g. sterile hemorrhagic cystitis with cyclophosphamide) that are discussed at the time of administration.
How is chemotherapy given?
The administration of chemotherapy, route and frequency is patient dependent. The tumor type, stage, and general health of the pet help dictate the appropriate protocol.

Some drugs are oral medications that are given at home. Others are injectable that require administration within the clinic with treatments being given weekly to every third week.

The duration of the chemotherapy depends on the tumor type, burden, response to therapy or the development of adverse effects. Some pets receive chemotherapy for the rest of their lives. In others, treatments may be spread out or discontinued after a period of weeks to months provided that the cancer is in remission. Chemotherapy can be resumed when the cancer relapses.

We usually recommend that every patient receive at least 2 cycles of chemotherapy and then be evaluated for response to therapy before making the decision to continue the treatment, change drugs or discontinue chemotherapy.

Metronomic chemotherapy
Recently, the concept of low-dose, continuous delivery of cytotoxic agents (“metronomic” chemotherapy), has been introduced. Metronomic chemotherapy is a relatively new type of chemotherapy that refers to the continuous/chronic administration (e.g. daily or every other day) of low doses of oral chemotherapeutic drugs with few or no rest periods. This treatment works not only by killing the cancer cells directly (like ‘traditional’ chemotherapy), but also by depriving the tumor of its blood supply by stopping new blood vessels from forming, and helping the immune system to mount a response against the tumor.

Metronomic chemotherapy typically consists of the combination of low doses of oral cyclophosphamide, chlorambucil or lomustine along with a non-steroidal anti-inflammatory drug and in some cases, an antibiotic.

This method of chemotherapy administration is utilized when we suspect microscopic cancer cells are present, but at levels where we are unable to detect them; when the primary tumor is adequately controlled (e.g., with surgery and/or radiation therapy), and there is no evidence of spread and that patient has undergone the current standard of care of treatment; when the pet isn’t a good candidate for conventional chemotherapy; when the caregivers simply cannot travel to the hospital as frequently as would be required for other protocols; where visible tumors are detected (e.g., metastases) and the pets are still feeling well in an attempt to slow down progression.

There are many advantages to this approach for the management of the cancer patient:

• Lower drug doses = lower risk of adverse effects/need for supportive medication
• Much milder, self-limiting adverse effects
• Less intensive and less frequent monitoring of the patient = reduced number office visits + less stress to the pet + increased convenience to the pet owner
• Lower cost, as many of the drugs are relatively cheap and given at lower doses
• Less development of resistance
• Possibility of combination with other regular and orally given targeted therapies

What can be expected from chemotherapy?
In most instances, we are unable to obtain a cure for our veterinary cancer patients. Again, the primary goal of cancer therapy is to maintain or improve the animal’s quality of life. Chemotherapy is used to minimize the discomfort caused by a tumor or to slow down the progression of the disease and the prognosis is determined by the response and biologic behavior.

Clear methods of evaluating the tumor response to chemotherapy should be determined so that future treatment decisions can be made. These should include tumor measurements, evaluation of remission vs stable vs progressive disease, recurrence, metastasis, and quality of life measures. These findings help with the decision process in regards to continuing the current protocol, switching medication, or discontinuing the protocol.
Canine osteosarcoma

Osteosarcoma (OSA) is the most common primary bone tumor in dogs accounting for approximately 85% of bone tumors and certain breeds tend to be more susceptible. Any bone can develop osteosarcoma but typically long bones are more prone to developing cancer. Thus, appendicular osteosarcoma is the most common canine primary bone tumor. There are several primary bone tumors that can occur because of the structures located in the area that include: chondrosarcoma, osteosarcoma, synovial cell sarcoma, fibrosarcoma, hemangiosarcoma, and liposarcoma.

These are highly aggressive tumors, characterized by painful local bone destruction and distant metastasis with an eventual metastatic rate of 90%. OSA is largely a disease of middle age to older dogs and commonly affects the limbs of large or giant breed dogs, but can also occur in other parts of the skeleton (skull, ribs, vertebrae, and pelvis). The biological behavior, prognosis, and treatment of bone tumors depends on the tumor type, primary site, and extent of the disease.

We do not know the precise cause of malignant cancers of bones in dogs but abnormal bone cell growth and unusual hormone stimulation may be implicated. A history of previous fracture at the site of osteosarcoma is also not unusual. Excessive proliferation of cells to heal the fracture gives greater opportunity for mutation to a cancerous form. Body size and sex are also important in the development of bone cancers in dogs so genetic factors also have a role.

Clinical signs

The signs associated with a bone tumor may be nonspecific. Tumors in the limbs often cause various degrees of lameness and pain, and a firm swelling may become evident as the tumor size increases. It is common for pain to be intermittent initially and it may improve with pain medications. As the degree of discomfort increases, it can cause other signs such as irritability, aggression, loss of appetite, weight loss, and exercise intolerance. Some dogs may present because of a fracture, due to the weakening of the affected bone. Other clinical signs may vary, depending on the primary site and the involvement of other organs such as a cough associated with pulmonary metastasis.

Diagnosis

There are several diagnostic tests that can be performed to evaluate the extent of disease. Prior to treatment, staging with a complete blood count, serum chemistry profile, three-view thoracic radiographs, and abdominal ultrasound is suggested; additional advanced diagnostic imaging such as nuclear scintigraphy, CT or MRI is occasionally warranted. Although fine needle aspiration and cytology may yield a diagnosis, histopathology may be needed for definitive diagnosis.

Prognostic factors

Young age, large tumor size, high histopathological grade, tumor location in the humerus, and elevated ALP levels have been associated with a poor prognosis.

Treatment options

Canine appendicular osteosarcoma can be treated aggressively or palliatively.

Dealing with this tumor involves treatment aimed at two categories - life and limb. Treatment is designed to help with bone pain associated with the primary tumor and slow the progression of metastatic lesions.

1. No treatment/medical management: survival times of approximately 4-6 months. This includes the administration of single agent or combination oral analgesics; Rimadyl (or some other antiinflammatory), Tramadol, Gabapentin, Amantadine, Tylenol 3/4, etc.

2. Amputation only: survival times of ~4-6 months. Although losing a leg may seem like it would be a hindrance, our companion animals tolerate three legs well and amputation does not inhibit walking, running, or jumping (after the surgical recovery time). It is removal of the source of pain. While it does not extend survival time any longer than the administration of pain medications, it is QUALITY time that does not require the lifelong administration of pills.

3. Amputation followed by chemotherapy: survival time of ~12-18 months. Chemotherapy is administered once every 3 weeks for 4-6 treatments. The chemotherapy is administered in an attempt to slow down the progression of disease even when there is no evidence of pulmonary metastatic disease.

4. Radiation therapy: survival time of ~6-8 months. Radiation is used for local management of the tumor with the goal to alleviate clinical signs and discomfort from the tumor; chemotherapy would still be recommended to slow down the aggressive biologic behavior.
5. Bisphosphonate therapy: reported survival times of approximately 6-9 months. These drugs inhibit the cells that resorb bone and help with alleviation of clinical signs when attempts are made to spare the limb. This is still just local disease control. Adjunctive chemotherapy would still be recommended to slow down the progression of disease.

**Feline osteosarcoma**

Primary bone tumors are uncommon in cats compared to dogs and osteosarcoma is the most common tumor reported, accounting for approximately 70% of primary bone tumors. The age range for cats at the time of diagnosis is from 1 to 20 years, with a mean age of 10 years. Males appear to be more affected than females. The biologic behavior of this tumor is locally aggressive with a low metastatic potential.

Osteosarcoma occurs more commonly in the appendicular skeleton than the axial skeleton. The hind limbs are affected more commonly than the forelimbs and the most commonly affected flat bones include the skull, vertebrae, scapula, and pelvis. Osteosarcoma diagnosed in long bones occurs in the proximal tibia, distal femur and the proximal humerus.

Clinical signs are associated with the area of tumor growth. Osteosarcoma affecting the limbs results in swelling, pain, limited range of motion, and progressive muscle atrophy from disuse of the limb. Tumors arising from flat bones may result in swelling of the affected area. Difficulty chewing is a common sign of cats diagnosed with tumors in the mandible and maxilla. Nasal tumors can result in discharge and facial deformity. Vertebral involvement often manifests itself as neurologic symptoms and involvement of the pelvic bones causes difficulty in defecation due to narrowing of the pelvic canal.

Surgery/amputation is the treatment of choice for feline osteosarcoma. The biologic behavior of osteosarcoma in cats is less aggressive than dogs and adjunctive therapy is usually not necessary.
Canine transitional cell carcinoma

Bladder tumors are not common in the dog, but transitional cell carcinoma (TCC) is the most common tumor diagnosed, accounting for <2% of all neoplasms. This tumor typically appears as a mass arising from the bladder wall and extending into the bladder lumen. The masses usually occur in the trigone of the bladder and can extend into the urethra which is affected in over half the patients diagnosed with TCC.

This type of tumor has a moderate metastatic rate, but is extremely locally aggressive and often invades surrounding structures such as the urethra, ureters, vaginal vault and regional lymph nodes. In male dogs, TCC can extend to the prostate gland. Even though approximately 25% of patients will have metastasis at the time of diagnosis, and more than 50% will disseminate or spread to other areas of the body (lymph nodes, lungs and bone) during disease progression, this occurs late in the course of disease. Thus, the life-limiting problem is more often related to obstruction of the urinary bladder as the tumor grows leading to extreme difficulties with urination. Progression of disease often involves inability to control urination that can progress to inability to urinate.

Risk factors associated with TCC include exposure to topical insecticides for flea and tick control, exposure to marshes sprayed for mosquito control, cyclophosphamide administration, female gender, obesity, and certain breeds such as Scotties, Shelties, Beagles, Fox terriers, and Westies.

Diagnostic tests

There are several diagnostic tests that can be performed to evaluate the aggressiveness and extent of this tumor type. These tests include: a complete blood count, serum chemistry profile, urinalysis via free catch, radiographs, and abdominal ultrasound. Fine needle aspirates are not recommended due to the possibility of spreading the tumor cells to the abdomen. Ultrasound guidance can be utilized for diagnostic catheterization.

Two tests have been developed as an attempt at early detection of TCC which include the Veterinary Tumor Antigen Test and (VBTA) and CADET® (Cancer DETection) BRAF Mutation Detection Assay.

Dogs with TCC are very prone to developing bacterial infection in the bladder. Therefore, frequent urinalysis, culture, and treatment with antibiotics may be necessary. A secondary bacterial infection can result in a sudden worsening in symptoms in dogs with TCC, and these dogs will improve with treatment with antibiotics.

Clinical signs

Transitional cell carcinoma is typically seen in older dogs that present with symptoms suggestive of a urinary tract infection including straining to urinate, increased frequency of urination and blood in the urine. Dysuria, lethargy, difficulty defecating and weight loss have also been reported. Less commonly, dogs with TCC can have lameness due to spread of the tumor into the bones or a cough because of spread into the lungs. This tumor type can also lead to changes in mentality and/or seizures due to metastasis to the brain.

Treatment options

There are several treatment options; however, the disease is almost never cured. Treatment is geared towards maintaining a good quality of life for as long as possible.

Local therapy

- Surgery
  - Because these masses typically occur in the trigone and can extend into the urethra complete surgical excision is unlikely. At times, surgery may be indicated as an attempt to obtain tissue for a diagnosis, to remove the TCC within the bladder when the cancer is away from the trigone, and to restore or maintain urine flow. Partial cystectomy, ureterocolonic anastomosis, debulking and bladder reconstruction have all been attempted.
- Radiation therapy
  - This involves the delivery of multiple, small fractions of radiation to the tumor in an attempt to decrease tumor size or slow local progression and metastasis. This is still a relatively experimental procedure in dogs but has proven beneficial in people with bladder cancer.

Systemic therapy

The mainstay of TCC treatment in dogs continues to be systemic medical therapy which usually consists of chemotherapy, cyclooxygenase (COX) inhibitors (non-selective COX inhibitors and COX-2 inhibitors), and combinations of these.

1. Piroxicam: This is a non-steroidal anti-inflammatory drug (NSAID) that inhibits an enzyme called cyclooxygenase (COX). COX contributes to inflammation and is thought to be upregulated in many types of cancer including TCC. The goal of piroxicam administration is to decrease tumor-associated inflammation and may also slow tumor
progression via antiangiogenesis. Piroxicam is well tolerated but can lead to kidney damage and gastrointestinal ulcers when used long term so lab work (CBC, chemistry, urinalysis) is recommended every 6-8 weeks during administration and must never be given with other NSAIDs or steroids as this increases the risk of stomach ulcers.

2. Mitoxantrone and carboplatin are injectable chemotherapy agents most commonly used in the treatment of TCC. Both drugs are administered through a carefully placed intravenous catheter and given once every 3 weeks for 6 treatments.

3. Vinblastine is an injectable chemotherapy agent that was only recently found to have efficacy against TCCs. Vinblastine is given as an intravenous injection at 2 week intervals with duration dependent on the patients response to therapy.

**Urinary diversion and stents**

- Cystostomy tubes
  - When secondary lower urinary tract obstruction is observed, urinary diversion techniques are pursued to help alleviate straining and improve urine flow. These are placed to allow the caregivers to manually evaluate the bladder.

- Stents
  - These can be placed in an attempt to create a patent urethra.

**Prognosis**

Overall, the prognosis for dogs with TCC is still guarded to poor. Factors that have been identified as negative prognostic indicators include extensive tumor burden throughout the bladder, detection of tumor beyond the bladder and prostate involvement.

In general, the survival times for patients diagnosed with TCC treated with piroxicam only is approximately 6 months. The addition of chemotherapy extends that survival time to approximately 12 months.

**Feline transitional cell carcinoma**

The literature is sparse in regards to bladder cancer in cats. Transitional cell carcinoma (TCC) is still the most common tumor diagnosed; however, lymphoma and various mesenchymal cell tumors have also been reported. The median age at diagnosis is around 15 years of age and there is a male predilection when compared to females. This tumor is most commonly reported in domestic short haired cats, but it has also been reported in Siamese, Abyssinian and Somali cats.

The clinical signs for cats with TCC are like those seen in dogs and can include hematuria, stranguria, inappropriate urination, pollakiuria and concurrent urinary tract infection is common.

Most cats present with masses in the bladder wall. Less than half occur in the trigone and urethral involvement is not commonly reported. The metastatic rate has not been clearly defined as the studies on feline transitional cell carcinoma are small. There have been reports that include a 27% metastatic rate at the time of diagnosis and the lungs and lymph nodes are most commonly affected.

Treatment for cats with transitional cell carcinoma consists of surgery, NSAIDs, chemotherapy and radiation therapy. Tumors located at the apex of the bladder are treated primarily with partial cystectomy. Reported overall median survival time for approximately is approximately 6-8 months; however, cats with surgically resectable disease may have better long-term prognosis.
Canine lymphoma

Lymphoma is a systemic form of cancer that affects lymphocytes which are a type of white blood cell of the immune system. These malignant lymphocytes affect lymphopoietic tissue, including the lymph nodes, spleen and liver; although it can involve any organ system of the body. The most common form is the multicentric generalized lymph node form. Most dogs continue to feel well in the early stages; however, decreases in appetite, activity and potential problems with breathing can occur if the lymph nodes and associated organs are heavily involved.

Lymphoma is the most common tumor type that we diagnose in veterinary medicine. Treatment response rates are high, cure rates are low (< 10%). Approximately 80 - 90% of dogs will achieve a remission (the disease ‘goes away’) following chemotherapy; however, most dogs will experience a relapse of their cancer at some point in the future. Patients can be completely normal one minute and experience lymph node enlargement or visceral organ involvement overnight. Luckily, this aggressive nature of the disease generally results in the rapid induction of remission and a decrease in tumor burden. As a matter of fact, lymph nodes can literally melt away within 6 - 72 hours following the administration of medication.

Can your patient be cured?

Unfortunately, no. We can achieve a pretty durable remission with most chemotherapy protocols, but unfortunately, we never achieve a cure. It is best to focus on a realistic outcome which is the longest possible survival with good quality life. Different treatment protocols are associated with different “disease-free” intervals. See below for more details.

Does your patient need further tests?

There are several diagnostic tests that can be performed to evaluate the aggressiveness/stage of lymphoma. These tests include: a complete blood count, serum chemistry panel, urinalysis, abdominal ultrasound, fine needle aspirate of lymph nodes, spleen or liver, lymph node biopsy, immunohistochemistry, flow cytometry, PARR, canine lymphoma blood test, bone marrow aspirate, etc. However, in the dog, a lymph node aspirate is frequently adequate to make the lymphoma diagnosis.

Lymphoma classification.

Lymphoma is classified by stages that have been extrapolated from human medicine (WHO staging system):

- Stage I: one lymph node involved
- Stage II: several lymph nodes in the same general area involved
- Stage III: all peripheral lymph nodes involved (multicentric form = most common)
- Stage IV: (± Stages I-III) spleen, liver, and/or anterior mediastinum in the chest involved
- Stage V: bone marrow, central nervous system, lungs, ocular, renal, nasal, skin, gastrointestinal tract, etc
  - substage a: no clinical signs
  - substage b: clinical signs

Lymphoma is further divided into two cell types: B cell lymphoma vs T cell lymphoma. 80% of patients are B cell lymphoma and have a 90% response rate to chemotherapy. T cell lymphoma is less responsive to therapy and survival times are shorter. Lymphoma associated with the gastrointestinal tract, skin, mediastinum, lungs and central nervous system is most commonly T cell; therefore, the survival times and response to therapy are generally cut in half.

What type of treatment is available?

Chemotherapy remains the mainstay of therapy and is generally very well tolerated in our patients vs our human counterparts; however, each patient is an individual and some dogs may require dose modifications or changes in drug type. The chemotherapy is administered at lower dosages and frequencies in an attempt to minimize side effects. Most signs are mild and self-limiting; however, side effects are not tolerated and at times medical intervention might be necessary. We have very effective drugs now to decrease the severity and/or prevent most side-effects such as nausea, vomiting or diarrhea.

There are several different protocols for the treatment of lymphoma that result in different survival times; keeping in mind that QUALITY of life over quantity is the most important goal.

Treatment 1 -

The CHOP chemotherapy protocol is considered the standard of care, but is also the most expensive and labor intensive of all of the protocols. Several different CHOP protocols exist; however, all represent combinations of cyclophosphamide (C), doxorubicin (hydroxy daunorubicin [H]), vincristine (Oncovin [O]), and prednisone (P). The CHOP protocol used in most dogs involves almost weekly treatments for 19-25 weeks.
Alternatives to CHOP-based protocols: There are alternatives to “CHOP” chemotherapy if cost or weekly time commitment is not appropriate for your client.

- **Treatment 2** - single agent doxorubicin is somewhat less effective than CHOP but is a less expensive and less time consuming alternative. Doxorubicin is given by intravenous (IV) injection once every 3 weeks for a total of 5 treatments. It typically results in 70% response (remission) rates with an average survival of approximately 8-9 months.

- **Treatment 3** - TANOVEA®-CA1 is the first FDA conditionally approved lymphoma treatment for dogs and is administered once every 3 weeks for 5 treatments with reported survival times similar to single agent doxorubicin.

- **Treatment 4** - single agent oral chemotherapy (e.g., CCNU [lomustine®]) may be attractive to clients declining injectable therapy.

- **Treatment 5** - single-agent prednisone (oral) therapy will often provide a short (e.g., 2-3 months) partial or complete remission and is very inexpensive.

- **Treatment 6** – administration of the lymphoma vaccine has been reported to provide anecdotal extension of survival following traditional chemotherapy of 6-12 months. The vaccine is initially given every two weeks for four doses, then a booster is given once every six months.

Prognosis

Overall, the median (average) survival of dogs with lymphoma who do not receive treatment is approximately 4 weeks, although this can vary considerably with the type and extent of the cancer at diagnosis. The average survival of dogs is extended to 12 months with most chemotherapy protocols, with approximately 20 – 25% of dogs living 2 years or longer. Again, this is an average and some dogs do better than the average and some do worse.

Feline lymphoma

Intestinal lymphoma (LSA) is the most common intestinal tumor in cats and is diagnosed in cats 10-12 years of age and older. Other reported locations include nasal, mediastinal, laryngeal, tracheal, renal, CNS, cutaneous, and peripheral nodal involvement.

There is a strong breed predilection as Siamese cats are overrepresented and patients that are FeLV/FIV positive are at greater risk for lymphoma development. In addition, there is an association between inflammatory bowel disease (IBD) and gastrointestinal (GI) lymphoma; therefore, implicating inflammation in the development of this cancer.

The clinical signs are dependent on the organ involved and can range from weight loss, anorexia, vomiting, diarrhea, intestinal thickening or mass effect(s), tachypnea, dyspnea, Horner syndrome, nasal congestion, sneezing, facial deformity, stridor, polyuria/polydipsia, renomegaly, seizures, abnormal mentation, plaque-like solitary or multifocal dermal lesions, often on the head or face, enlargement of a single lymph node or lymph node chain (Hodgkin-like) or generalized peripheral lymphadenopathy.

Staging tests include but are not limited to a complete blood count, serum chemistry panel, FeLV/FIV testing, urinalysis, abdominal ultrasound, and thoracic radiographs. Advanced diagnostic imaging such as CT scan or MRI may be indicated for nasal or CNS presentations. Fine needle aspirates of masses and/or involved organs is often adequate to provide a definitive diagnosis of intermediate to high-grade LSA and large granular lymphoma. Histopathology is needed for a definitive diagnosis of gastrointestinal lesions. Additional tests such as immunohistochemistry and PARR might be necessary.

Gastrointestinal lymphoma is divided into two forms. Enteropathy-associated T-cell LSA (EATL) types I and II. EATL type I is usually high-grade or large cell/lymphoblastic gastrointestinal LSA. This form is generally characterized by intermediate to large cells that display transmural invasion and often form masses. Large granular lymphoma is an aggressive subtype of EATL type I. Patients usually present with multiple masses within the GI tract and other visceral organs.

The more common presentation of gastrointestinal LSA is EATL type II. This form is characterized by diffuse superficial (mucosal/lamina propria) infiltration of small T-lymphocytes. It is referred to as low-grade, lymphocytic, small cell, or indolent GI LSA. The disease course is usually slowly progressive.

All forms of feline lymphoma are primarily a chemotherapy responsive tumor type with a few exceptions. Generally, cats with EATL type I are treated with multiple chemotherapy agents. The overall response rate is approximately 50-60% with survival times ranging from 4-8 months. A small portion of cats have reported survival times of 1-2 years. Cats diagnosed with large granular lymphoma carry a poor prognosis of 3 months or less. While cats diagnosed with EATL type II are treated with chlorambucil and prednisolone and have reported response rates of 70-80% and >2 years survival.

At times, cats can be treated with surgery when the disease is localized (some GI, CNS, cutaneous, nodal). Radiation therapy is reserved for localized presentations of lymphoma like mediastinal, CNS, peripheral nodal, laryngeal/tracheal, some cutaneous and is the treatment of choice for nasal lymphoma.

The survival times for feline LSA are variable and depend on viremic status, tumor burden/location, therapy pursued, and response to therapy.
Canine mast cell tumors

Mast cell tumors (MCT) are the most common skin tumor in the dog. They are primarily found in older dogs but they have been reported in dogs as young as three weeks of age. Breeds frequently diagnosed with MCT include Boxers, Boston terriers, Bulldogs, and Labrador retrievers, but most are reported in mixed breed dogs. The biology of MCT in the dog is closely correlated to the histologic grade. Low grade tumors are usually well differentiated, slow-growing, usually < 3-4 cm in diameter, without ulceration of overlying skin, variably alopecic, commonly present for more than 6 months and rarely metastasize. High grade tumors are poorly differentiated, locally invasive, rapidly growing, variably sized, with ulceration of the underlying skin and inflammation/edema of surrounding tissues and rarely present for more than 2-3 months before presentation. High grade tumors are usually anaplastic and highly aggressive, with a high metastatic rate. Some anatomical locations such as tumors arising at or near mucocutaneous junctions (peripreputial/scrotal, muzzle, nail bed, aural, perianal, oral, etc) have been associated with more undifferentiated tumors and higher metastatic rates.

Diagnostics

MCT are initially diagnosed with fine needle aspiration cytology and aspirates of any regional lymph node is recommended in all cases. Other staging tests including bloodwork (CBC, chemistry), urinalysis, abdominal ultrasound, and rarely bone marrow aspiration are recommended in cases that have poor prognostic features (bad location, rapid growth, high grade, etc.). Histologic grade can be determined by incisional biopsy if the tumor is located in an area where wide surgical excision is not possible. Recently a cytologic grading scheme has been investigated to classify tumors as high grade if it was poorly granulated or had at least 2 of 4 findings: mitotic figures, binucleated or multinucleated cells, nuclear pleomorphism, or > 50% anisokaryosis. The cytologic grading scheme had 88% sensitivity and 94% specificity relative to histologic grading.

Prognostic factors

There are several factors that have been linked to a favorable outcome for patients diagnosed with mast cell tumors to include low histologic grade and Boxer breed. Location (muco-cutaneous junctions and bone marrow), regional/distant metastasis, high histologic grade, tumor > 3 cm in diameter, mitotic index (> 5/10hpf) or other cell proliferation indices, clinical signs, rapid growth/ulceration, recurrent disease, and c-kit mutations have been associated with a poor prognosis.

Treatment

Surgery remains the mainstay of therapy for MCTs, and is curative in a large percentage of cases. Surgical margins are based on tumor grade. Three centimeter margins and at least 1 fascial plane below the tumor is still recommended if there is adequate tissue surrounding the tumor and the grade is unknown.

If the tumor is small and easily resected, surgical excision without performing any further diagnostic tests is recommended.

If the tumor is large or located in a site that is not easily resectable, or the patient has negative prognostic factors, or if the treatment is likely to be expensive or create a functional problem, then additional diagnostic tests are warranted to assist with surgical planning and ensure that there is no evidence of metastasis. In addition, chemotherapy and/or TKI inhibitors might be recommended to decrease the tumor burden prior to surgery.

Complete excision and grade are assessed by a pathologist to help dictate the next course of action. If the tumor has been completely removed and is of low/intermediate grade, then no further treatment is recommended other than ‘wait and watch’ – which means routine recheck examinations to ensure the tumor has not returned and no additional lumps and bumps are present.

If the tumor is low/intermediate grade and was not completely excised then the following options are available:

1. Take a ‘wait and watch’ approach without treatment but with routine evaluations to document tumor recurrence at the earliest possible time. Approximately 50% of mast cell tumors will recur if not completely removed the first time. There is a risk that the recurrent tumor will behave more aggressively and may be more difficult to deal with.

2. Perform a second surgery in an attempt to remove the microscopic disease. This is not always possible due to location.

3. Post-surgery radiation therapy is an excellent option for long term control. Approximately 85% of dogs will have long-term (5-year) control or cure when radiation therapy is administered following surgery.

If a high grade tumor is not completely excised, surgery and radiation therapy are still viable options for local control of residual disease. However, adjunctive therapy (chemotherapy and/or tyrosine kinase inhibitor therapy) is still warranted if a high grade tumor is completely excised or not. The first 6 months are the most critical. If we can get a patient through the first 6 months, then there is a 50:50 chance of > 2-year survival.
1. Chemotherapy typically involves the administration of 4 weekly injections of vinblastine followed by 4 injections at every 2 week intervals along with oral prednisone. Additional chemotherapeutic agents such as CCNU have also been utilized.

2. Recently, a new class of drug has been developed to treat mast cell tumors. Palladia (toceranib) is Tyrosine Kinase Inhibitor (TKI) that works by shutting off tumor growth ‘light-switches’ and/or shutting off new blood vessel supply to growing tumors. This is an oral drug that is given every other day or three days a week for many months or even years. This drug is currently used for high grade tumors, tumors that are not removable with surgery following traditional chemotherapy, and patients at high risk for recurrence or metastasis following surgery and chemotherapy.

Ancillary treatment
Mast cells are key players in the inflammatory response and can be activated to release a wide variety of inflammatory mediators, by many different antigens including allergens, pathogens and physiological mediators producing clinical signs like local or generalized hives, redness, swelling, or itchiness; therefore, antihistamines (e.g., Benadryl) might also be prescribed. In addition, mast cells have been reported to increase the likelihood of gastric ulceration resulting in nausea, vomiting, anorexia, and gastrointestinal bleeding which at times warrants the administration of gastroprotectants such as pepcid, omeprazole, Carafate as well as antinausea medications like cerenia or metoclopramide.

Prognosis
High-grade mast cell tumors are significantly associated with shorter time to metastasis or new tumor development and with shorter survival time. In general, the median survival time 4-6 months for high-grade mast cell tumors but more than two years for low-grade mast cell tumors.

Feline mast cell tumors
Mast cell tumors are the most common splenic tumor, second most common skin tumor and third most common intestinal tumor in cats. MCTs are primarily a disease of older cats; however, it has also been reported in young cats. The only feline breed that has been reported to be at increased risk for MCTs is the Siamese. Most reports show no significant gender predilection and the etiology of MCTs is presently unknown.

The history and clinical signs of cats with MCTs can be extremely variable. Most do not show any clinical signs referable to their MCT; however, some may have signs associated with the release of heparin, histamine and/or other vasoactive amines.

Mast cell tumors are classified based on anatomic location as cutaneous and visceral forms. The cutaneous form is more common and is most often located on the head, neck and trunk. This form is further divided histologically into mastocytic and atypical forms. The mastocytic form of the disease is more common and is further classified into well-differentiated and poorly differentiated forms with the well-differentiated form being the most common histological type. The well-differentiated mastocytic forms behave in a more benign biologic behavior than the poorly differentiated malignant counterparts. The atypical form of this disease occurs most commonly in cats less than 4 years of age and have been reported to spontaneously regress within 4-24 months. The primary site for visceral tumor formation is the spleen followed by the gastrointestinal tract and should be thoroughly evaluated for cutaneous lesions.

Diagnosis
Feline mast cell tumors are also diagnosed via cytological evaluation of a fine needle aspirate. Pretreatment with antihistamines is warranted prior to the aspiration of visceral tumors as mast cell degranulation, anaphylaxis and death are potential concerns. Full staging consisting of a complete blood count, serum chemistry panel, thoracic radiographs, abdominal ultrasound and bone marrow aspirate are recommended for cats with evidence of visceral involvement, multiple cutaneous masses, diagnosis of an atypical tumor, clinical signs, and organomegaly.

Treatment
Surgery remains the mainstay of therapy when possible and there is currently no correlation between completeness of surgical excision and recurrence rate. Splenectomy is the treatment of choice for splenic MCTs with a good prognosis for long term survival. Conversely, the post-surgical prognosis for intestinal mast cell tumors is poor.

Other treatments with strontium 90, chemotherapy (lomustine, vinblastine, and cyclophosphamide), and receptor kinase inhibitors have been evaluated. Antihistamines and proton pump inhibitors are recommended for patients with gross disease, metastasis and patients diagnosed with the visceral form of the disease.

Prognosis
The prognosis for cats with mast cells tumors is dependent on location and form. Surgery is usually curative for cats with solitary cutaneous lesions. These tumors have low recurrence and metastatic rates. The poorly differentiated mastocytic form has a higher metastatic potential and is associated with a poor prognosis. Thorough evaluation is recommended for cats with multiple cutaneous tumors as they may be metastases from a visceral tumor. Cats with splenic MCTs have a good prognosis with long term survival from splenectomy alone. Unfortunately, the prognosis for cats with intestinal mast cell tumors is poor.
Diagnosis and Treatment of Canine Glaucoma

Mark Bobofchak,
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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 Humor Circulation

- Aqueous supplies the avascular lens & cornea with nutrients and removes wastes
- Produced in posterior chamber, moves through pupil to anterior chamber (AC)
- Rate of formation = rate of removal, so IOP remains constant
- Thermal currents in AC cause aqueous to rise against iris and fall against cornea
- Aqueous then exits via conventional and non-conventional pathways

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
Signalment can be a huge clue!

Diagnosing glaucoma

Primary glaucoma breeds
*classic breeds seen at ECFA

- Arctic breeds *
- Basset 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
- Springer spaniel
- Shih Tzu
- Shiba inu
- Shar Pei
Presenting signs are second biggest clue!

Glaucoma vs Exophthalmia

Chronic versus acute
- History
- Buphthalmos
- Lens subluxation
- Atrophied or cupped optic nerve

Chronic vs. acute
- 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 corneal 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

Hyposmotics

- 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
  - Travoprost

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
- Cyclocryothermy
- Goniovalve procedure
- Intrascleral prosthesis *
- Enucleation *
- Ciliary body ablation *

*Permanently blind, painful eye

Transcleral laser cyclophotocoagulation

Endolaser Cyclophotocoagulation
Cyclocryotherapy

Gonioimplant

Post op gonioimplant

Intrascleral prosthesis
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
The Canine Lens: Clarification of a Cloudy Eye
Mark Bobofchak, DVM, DACVO
Eye Care for Animals
Akron, OH

The Canine Lens

Caution: This topic can get a little foggy

Mark Bobofchak, DACVO
Eye Care for Animals
Akron, OH

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

Lens Development

• Anterior - 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 (humidity and temperature of ocular structures)
- 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

http://education.vetmed.vt.edu/courriculum/VM8054/EYE/ACCOMOD.HTM
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

Cataracts - Classification

- Density
  - Incipient
  - Immature
  - Mature
  - Hypermature

- Age of Onset
  - Juvenile
  - Senile

- Cause
  - Diabetes
  - Nutritional deficiency
  - Electrocution
  - Radiation
  - Metabolic

- Location
  - Capsular
  - Cortical
    - (anterior/posterior)
  - Equatorial
  - Axial
  - Nuclear

What is a cataract?

- Most cataracts due to...
  - Disruption of normal fiber arrangement
    - Diabetes
    - Trauma
  - Increase in proportion of insoluble (albuminoid) proteins
    - Age-related cataract
    - Hereditary cataract
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

- Do nothing
  - Incipient cataracts
  - No vision impairment
  - Not likely to progress

- Medical management
  - Topical anti-inflammatory
    - Phacoxylic agent

Management Options

- N-acetyl carnosine
  - Ocuvet™, Bright Eyes™
  - Powerful anti-oxidant
  - Shown to disaggregate crystallins in vitro
  - 2006 preliminary study
    - Significant reduction of opacity for immature cataracts and nuclear sclerosis
    - 80% of 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
    - 41.5 Dioptries - Dog
    - 53 Dioptries - Cat

Cataract Surgery Facts

- Ideal stage is immature or early mature
  - Early referral for evaluation
- 90-95% 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
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 cat

Cataract Surgery Facts

- Diabetes Mellitus
  - Nearly 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
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
- Endophthalmitis
- Capsule fibrosis
- Chronic uveitis
- Lens implant dislocation

Lens Luxation

- **Subluxation**
  - Partial zonule breakdown
  - Aphakic Crescent
  - Anterior chamber vitreous
  - Phacodonesis
  - "Jiggling" of the lens when the eye moves

- Treat with early surgery or miotics and anti-inflammatories
  - Pilocarpine
  - Demecarium Bromide
  - Latanoprost

- **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 dyskinesis
    - Focal corneal edema (endothelial touch)
  - **Often pupillary block glaucoma**
    - Apparent ciliary-iris fibers pass through the pupil to reach the intracameral angle.
    - Severe, nonresponsive glaucoma

- **Is this an emergency??**
  - Yes….if glaucoma present and vision intact (positive menace, dazzle, and indirect PLR)
  - If chronic and 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 (far-sighted)

- **Risks of ICLE**
  - Persistent glaucoma
    - Patients remain on lifelong hypotensive therapy
  - Retinal detachment
    - Diode laser photocoagulation 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

- **Lens Luxation**

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To Grid or Not to Grid...

Corneal Ulcers and Other Confusing Corneal Conditions

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To Grid or Not to Grid...
(Corneal Ulcers and other confusing corneal conditions)

Mark Bobofchak, DACVO Eye Care for Animals Akron, OH

Topics

- Ulcers
  - Indolent
  - Stromal
  - Descemetocoeles
  - Perforations
- Lacerations
- Autoimmune
- Dystrophy
- Degeneration
- Endothelial decompensation
- Corneal Sequestrum

Indolent Ulcers

- "Boxer Ulcer", Non-Healing Ulcer, SCCED, REE
- Older Dogs
  - > 6 years old
- Typical history
  - Progressive "redness"
  - Cloudiness
  - Intermittent blepharospasm
  - Good days and bad days
  - Older no known trauma

*Failure of epithelial-stromal adhesion complex formation
- NOT BM dystrophy
  - Older dogs
  - Normal BM in unaffected cornea
- Loss of BM
  - Normally cannot manually debride
  - Delays wound healing
- Stromal changes
  - Superficial acellular hyalinized zone
  - Barrier to adhesion complex
Indolent Ulcers

Medical Options

- Topical antibiotics
- Epithelial cell toxicity?
- Serum?
- Remend?
- EDTA
- Chondroitin Sulfate
- Cytosarclyte
- Substance P
- PIGAGs
- MMP inhibitors?
- Doxycycline

Surgical Options

- Debridement
- Cotton Swab vs. Blade
- Linear Grid Keratotomy
- Diamond Burr Debridement
- Multifocal Superficial Punctate Keratotomy
- CO2 Laser Keratotomy
- Thermokeratoplasty
- Keratectomy

Topical antibiotics
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- Serum?
- Remend?
- EDTA
- Chondroitin Sulfate
- Cytosarclyte
- Substance P
- PIGAGs
- MMP inhibitors?
- Doxycycline

Stromal Ulcers

- Increased proteolytic enzymes
  - MMP 2 and 9
  - Inflammatory cells
  - Epithelial cells
  - Fibroblasts
  - Bacteria
- Often rapidly progressive
- Varying degree of pain
  - Rosa

TREAT AGGRESSIVELY

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

Stromal Ulcers

Treatment Options

- Medical management
- Temporary Tarsorrhaphy
- Third Eyelid Flap
- Conjunctival Pedicle Graft
- Corneconjunctival Transposition
- Amniotic membrane / PStS graft
- Frozen Cornea Graft
- Corneal Collagen
- Crosslinking with Riboflavin

Size

Depth

Location

Corneal consistency
### Medical Management

**Culture/Sensitivity and Cytology**
- **Antiprotease/Anticollagenase**
  - Serum
  - EDTA
  - Doxycycline
  - N-acetyl-cysteine
  - Ilomostat (Galardin)

**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 synchiae
- Caution with KCS patients

**Systemic pain control**
- Tramadol
- NSAID

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**Systemic pain control**
- Tramadol
- NSAID

### Medical Management

**E-collar**
- IMPORTANT
- Exercise restriction
- Warm compresses
  - NOT for descemetoceles!!
- Frequent rechecks until cornea is stabilized and stromal thickness is improving

**Benefits**
- Cheap
- No anesthesia
- May have reduced

**Risk**
- May progress to perforation
- Frequent drops
- Dependent on owner

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Surgical Options

• Conjunctival Pedicle Graft
  • Effective technique
  • Immediate tectonic support and blood supply
  • Often leaves large scar
  • Ideal for large, peripheral, or malacic ulcers

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

Surgical Options

• Corneocconjunctival 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
  - Greyhounds
  - Progressive vascularization and pigment
  - Ventrolateral limbus
  - Third eyelid
  - Mineral degeneration

- **Superficial Punctate Keratitis**
  - Dachshund, Shetland
  - Multiple punctate opacities
  - +/- ulcers

  - CSA/Tacrolimus
  - Corticosteroids

- **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 ???
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 rule out 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
  - Hyperproteinemia
  - Postprandial plasma lipid elevation
  - Unilateral or bilateral
  - Avascular early
  - Treatment
    - ID and treat underlying cause
    - Topical anti-inflammatory 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 Disease

• 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 incipient ulceration due to bullae formation
  • Topical steroids
    • If underlying uveitis suspected
  • Thermokeratoplasty 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
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

Thank you for your time and attention!!

Questions??
Uveitis is a common cause of blindness and can be the initial symptom of life-threatening systemic disease. Inflammation of the anterior uvea (i.e., iris and ciliary body) is termed anterior uveitis, or iridocyclitis, and inflammation of the posterior uvea (i.e., choroid) is termed posterior uveitis, or choroiditis. Anterior uveitis is more easily diagnosed than posterior uveitis because the anterior uvea is easily visualized and accessible to simple diagnostic techniques. Ophthalmoscopy is required for recognition of posterior uveitis. Inflammation of the entire uvea is called panuveitis.

Causes of uveitis
Possible causes include toxic, traumatic, infectious, neoplastic, metabolic, immune-mediated, and idiopathic causes. Ulcerative keratitis causes uveitis but does so through a poorly understood "axon reflex". Immune-mediated or idiopathic disease is the most common cause occurring in approximately 60% and 70% of dogs and cats, respectively. Neoplasia accounts for approximately 25% and 13%, and infectious disease 18% and 30%, in dogs and cats, respectively.

Clinical signs and diagnosis
The clinical signs of uveitis are attributed to disruption of the blood-ocular barrier and release of various chemical mediators of inflammation, notably prostaglandins. The signs of uveitis are variable, and some mimic other ocular diseases. Conjunctival hyperemia and ciliary flush constitute varying degrees of a "red eye". Pupillary constriction, or miosis, is common and can be subtle or pronounced. Chronic uveitis may cause adhesions of the iris to the anterior lens surface, or posterior synechia, resulting in an immobile or distorted pupil. Aqueous flare is hallmark of uveitis and due to increased protein and cellular debris in the anterior chamber. Aqueous flare is most often subjectively graded on a scale of 1-4 with 4 being the most severe. Red and white blood cells within the anterior chamber are referred to as hyphema and hypopyon, respectively. Lipemic flare is seen in animals with concurrent uveitis and hyperlipidemia. Keratic precipitates (KPs) are accumulations of inflammatory cells on the ventral corneal endothelium. Decreased intraocular pressure (IOP) can be a symptom of uveitis but alone is not necessarily diagnostic. Differences in IOP between the eyes (e.g., 3-5 mmHg or more), even if values are in the normal range, are supportive evidence of uveitis when combined with other ocular signs. IOP values above 25 mmHg in the presence of uveitis indicate secondary glaucoma. Corneal edema is often present with uveitis but can occur with glaucoma, age-related corneal endothelial degeneration, and corneal ulcers. Ophthalmoscopy is necessary to assess the posterior segment. Choroiditis or chorioretinitis may manifest as mild retinal edema, small serous or exudative retinal elevations (or detachment), or retinal hemorrhages. Optic neuritis may also be present. Vitreous opacity or severe anterior uveitis may impede visualization of the fundus.

Systemic evaluation
An attempt should be made to determine the cause of the uveitis. The clinician must choose between extensive diagnostic evaluation for uveitis and associated client expense versus only symptomatic therapy. A history and general physical examination are indicated in all cases. A complete blood count and serum biochemical profile should be considered as minimum screening for cases where systemic disease is suspected. Additional tests might include urinalysis, thoracic radiographs, abdominal ultrasound or radiographs, and serology or urine antigen testing for selected infectious diseases. Bilateral anterior uveitis and unilateral (or bilateral) panuveitis with retinal involvement are suggestive of systemic disease. However, animals with systemic disease may occasionally present with only unilateral uveitis.

Consider the following systemic diseases

<table>
<thead>
<tr>
<th>Canine</th>
<th>Feline</th>
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<tr>
<td>Leptospirosis</td>
<td>FIP</td>
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<tr>
<td>Brucellosis</td>
<td>FeLV</td>
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<tr>
<td>Blastomycosis, Cryptococcosis, other</td>
<td>FIV</td>
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<tr>
<td>RMSF</td>
<td>Toxoplasmosis</td>
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<tr>
<td>Ehrlichiosis</td>
<td>Bartonellosis</td>
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<tr>
<td>Anaplasmosis</td>
<td>Blastomycosis, Cryptococcosis, other</td>
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<tr>
<td>Lyme disease</td>
<td>Neoplasia (lymphosarcoma)</td>
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<tr>
<td>Neoplasia (lymphosarcoma)</td>
<td></td>
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<tr>
<td>Uveodermatologic Syndrome</td>
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Miscellaneous diagnostic evaluation
Cytologic evaluation and microbial culture of aqueous or vitreous aspirates is beneficial in select patients. Aqueous centesis is performed following general anesthesia and utilizing an insulin syringe and 29-gauge needle directed into the anterior chamber at the temporal limbus. Aqueous centesis and cytology is a relatively low-yield procedure except in the diagnosis of lymphoma. In patients with posterior segment disease, vitreous or subretinal aspiration is more likely to yield positive results. General anesthesia is recommended, and a 22-gauge needle is directed nasally and posteriorly through the superotemporal pars plana of the ciliary body, approximately 6-8 mm posterior to the limbus. This technique should be reserved for eyes with marked vitreous opacity, endophthalmitis, or exudative retinal detachment (i.e., usually blind eyes). Care must be taken not to puncture the lens, and intraocular hemorrhage following either aqueous or vitreous aspiration is possible.

Treatment of uveitis
Topical anti-inflammatory treatment should be instituted immediately in an effort to avoid irreparable damage to the eye(s). Systemic anti-inflammatory treatment requires greater discretion. Treatment often includes a combination of anti-inflammatory, mydriatic/cycloplegic, and antimicrobial medications. Topical treatment may suffice for mild uveitis, but severe uveitis and eyes with posterior segment disease require systemic therapy. Corticosteroids remain the primary topical treatment for uveitis, including those patients where infectious disease is suspected. Recommended steroids include 1% prednisolone acetate, 0.1% dexamethasone alcohol, or difluprednate (Durezol). Acetate preparations are superior to phosphate preparations for ocular penetration. Dexamethasone can be acquired alone or in combination with neomycin and polymyxin (i.e., neo-poly-dex); the latter preparation is often less expensive than dexamethasone alone, but the antibiotics are of little benefit for treatment of uveitis. The frequency of application varies with severity of the uveitis and may be anywhere from q.24h to q.4h. Topical steroid treatment is contraindicated in the presence of a corneal ulcer.

Caution is advised before starting systemic corticosteroid treatment, particularly when bacteremia or mycotic infection is suspected. For patients where noninfectious or immune-mediated disease is suspected, prednisone can be administered at 1-2 mg/kg/day followed by a tapering dose as the eye(s) improve. Systemic NSAIDs (e.g., carprofen, meloxicam, deracoxib, robenacoxib) can be of benefit, but clinical experience suggests they are not as effective as oral steroids for treatment of uveitis. Anti-inflammatory agents such as azathioprine (Imuran) are not indicated for most cases of uveitis, but azathioprine is the drug of choice for patients with uveodermatologic syndrome.

Mydriatic/Cycloplegic drugs
Mydriatic/cycloplegic drugs such as atropine and tropicamide have several actions that include pupil dilation to reduce the risk of posterior synechia, paralysis of iris and ciliary body musculature to reduce painful spasms, and stabilization of the blood-aqueous barrier that has a nonspecific anti-inflammatory effect and aids in the return of normal intraocular pressures. Atropine 1% solution or ointment is most often used, but some ophthalmologists prefer 1% tropicamide solution. Tropicamide is a weaker drug with a shorter duration of action and tends to allow more pupil mobility compared with the wide mydriasis that occurs with atropine. The frequency of application of either drug will vary with the individual patient; twice daily treatment followed by gradual reduction is acceptable for the majority of cases. These drugs are contraindicated in the presence of elevated IOP. Atropine should be used cautiously in particularly old, debilitated, or small patients, as systemic effects may include constipation, tachycardia, and CNS signs. Cats are notorious for excessive salivation after application of atropine drops to the eye, but the salivation may be less severe with ointment than drops. Tropicamide has the same relative indications and contraindications but is less likely than atropine to cause adverse effects.

Antimicrobial therapy
Systemic antibiotic or antifungal treatment may be appropriate for specific infections. For empiric oral treatment where the cause is not clear and tests are pending, the author most often uses doxycycline (5 mg/kg BID) in dogs and clindamycin (12.5 mg/kg BID) in cats.

Lens induced uveitis
Lens-induced uveitis (LIU) occurs when the uvea is exposed to lens protein. Phacolytic uveitis is common in dogs with cataracts, where leakage of lens protein occurs through an intact lens capsule. In mild cases, signs may include conjunctival hyperemia, decreased intraocular pressure, or a darkened iris. In more severe cases, aqueous flare, keratic precipitates, synechia, and even hyphema may be present. Uncontrolled LIU can result in secondary glaucoma or phthisis bulbi. LIU should be controlled prior to serious consideration of cataract surgery. LIU is especially common in diabetic dogs and certain breeds with rapidly progressive cataracts such as the Bichon Frise and Siberian Husky. Phacoclastic uveitis occurs after spontaneous or traumatic lens rupture and is more difficult to control. It can quickly result in intractable glaucoma and secondary uveitis and is relatively common with diabetic cataracts (Wilkie DA, et. al., Vet Ophthalmol 2006; 9: 328–334).
Ocular disease is often associated with disseminated mycotic infection and may represent the first obvious symptoms. Mycotic infection is characterized by granulomatous inflammation that typically starts in the posterior segment and progresses to affect the entire eye (i.e., panuveitis). Itraconazole and fluconazole are the preferred treatments, but there is no consensus as to which is most effective. Terbenaﬁne may used in conjunction with these drugs. Though controversial, systemic steroid administration should be considered for dogs with ocular blastomycosis that have potential to retain vision in the affected eye(s). This is because the inflammatory response to the organism quickly destroys the eye unless dampened with steroids; oral NSAIDs may improve comfort but are of virtually no beneﬁt in controlling ocular inﬂammation associated with blastomycosis. The author often starts prednisone (0.7 mg/kg/day) and tapers treatment over the next few weeks as the eyes improve (Finn MJ, et al., Vet Ophthalmol 2007;10:299-303). Steroid treatment had no obvious effect on patient mortality, and treatment is tapered as the eyes improve.

Golden retriever uveitis
This condition was initially called pigmented uveitis, but given its predisposition and prevalence in the breed, it is also called Golden retriever uveitis (Sapienza JS, el. Al., Vet Ophthalmol 2000;3:241-246). Affected dogs are usually older than 4 years of age. Onset is insidious with gradual darkening and thickening of the irides, mild to moderate aqueous ﬂare, pigment clumps on the anterior lens capsule, posterior synchia, and uveal cysts. Lens capsular pigment is often but not always distributed in a radial manner. The uveitis is usually slowly progressive but often results in cataract and secondary glaucoma. The visual prognosis for affected dogs is guarded. It is not clear whether topical steroid treatment slows progression, and topical NSAIDs can prematurely increase the pressure. Hypotensive treatments are initially effective, but long-term pressure control is difﬁcult.

Uveodermatologic syndrome
Uveodermatologic syndrome in dogs is similar to Vogt-Koyanagi-Harada (VKH) syndrome in people and is caused by immune-mediated response against melanocytes. In dogs, severe uveitis and retinal detachment often develops in association with some degree of poliosis and vitiligo. Ocular signs often preceed dermatologic changes, and some dogs never show obvious dermatologic changes. There is predilection for the Akita, but it has been reported in a number of breeds (e.g., Samoyed, Siberian Husky, Sheltie). Affected dogs must be treated aggressively, but long-term management is best achieved with oral azathioprine at 1 mg/kg/day with gradual tapering of the drug.

Feline uveitis
Feline uveitis is often more insidious in onset when compared with canine uveitis, and florid clinical signs may be absent. Chronic uveitis is the most common cause of cataracts, lens luxation, and glaucoma in cats. In a retrospective study of 53 cats with uveitis, 69.8% were diagnosed with idiopathic uveitis. In other studies of cats with uveitis, approximately 82% of the cats had positive serology for one or more diseases including toxoplasmosis, FeLV, FIV, and coronavirus. Of course positive serology does not necessarily indicate causative role in the uveitis. The greatest association between serologic prevalence of an infectious disease and uveitis has been demonstrated for toxoplasmosis, suggesting that Toxoplasma gondii may have a prominent role in feline uveitis.

Neoplasia
Lymphosarcoma is the most common metastatic neoplasm to the eyes of dogs and cats, and anterior uveitis is the most common finding. Concurrent systemic involvement (e.g., lymphadenopathy, etc.) simpliﬁes the diagnosis, but the eyes may be affected prior to other systemic signs. Cytologic evaluation of aqueous (especially with visible exudates) or vitreous may yield neoplastic cells. Uveal melanoma is the most common primary ocular neoplasm of dogs and cats and is associated with uveitis in advanced cases. Primary ocular sarcoma is unique to cats, aggressive with potential for metastasis, and formerly known as post-traumatic sarcoma. These tumors are believed to arise from a damaged lens and can develop many years after ocular injury or initial uveitis. Blind feline eyes are best enucleated to reduce the risk of this tumor. Intraocular bleeding of undetermined cause should arouse suspicion for ocular neoplasia of any cause, particularly in geriatric pets.

Lipemic-aqueous flare
Lipid is normally excluded from the anterior chamber. However, breakdown of the blood aqueous barrier (i.e., uveitis) allows lipid to enter the anterior chamber resulting in lipid-laden aqueous. Even mild uveitis in the presence of hyperlipidemia can result in profound cloudiness of the anterior chamber. The uveitis is treated as for any other patient, and the anterior chamber clears with resolution of the inﬂammation. Systemic evaluation is advised to seek causes for hyperlipidemia and should include a biochemical proﬁle, cholesterol and triglyceride levels, and thyroid testing.

Intraocular hemorrhage
Blood in the anterior chamber (hyphema) or posterior segment is not a speciﬁc ﬁnding and can occur with any form of uveitis. The bleeding can be unilateral or bilateral. Possible causes include trauma, coagulopathy, systemic hypertension, certain infections, and primary or metastatic tumor. General physical examination is appropriate to assess for petechiae, lymphadenopathy, etc., and a platelet count or coagulation tests and blood pressure determination should be considered. Enucleation and pathology should also be considered for a permanently blind eye for which the cause of hyphema is undetermined.
Herpesvirus infections
Ocular disease due to feline herpesvirus (FHV) is common. It is estimated that 80% of cats are latently infected with the virus, and approximately 40% of these cats will suffer recrudescent infection in later life. These estimates are based on data that is several decades old, and the actual percentages may be higher. FHV affects cats of all ages, but the initial (or primary) infection usually occurs in neonatal and adolescent cats. Symptoms include bilateral conjunctivitis, respiratory disease, and fever. Most cats recover from the primary infection in 7-10 days without specific antiviral treatment, but neonatal cats are more likely to suffer serious corneal and conjunctival scarring. Recrudescent infection can be unilateral or bilateral and with or without respiratory signs. Unilateral conjunctivitis or ulcerative keratitis in the absence of respiratory signs is common in adult cats with recrudescent infection. Stress appears to be an important factor in precipitating FHV. “Stressful” events may include topical or systemic corticosteroids, concurrent systemic disease, anesthesia, hospitalization, acquisition of a new cat, or extended owner absences (e.g., vacation). Other conditions that may be associated with FHV include non-ulcerative (or stromal) keratitis, conjunctival and corneal scarring (symblepharon), corneal sequestrum, eosinophilic keratitis, keratoconjunctivitis sicca (KCS), blocked nasolacrimal duct, and possibly uveitis.

Herpesvirus keratitis
FHV keratitis can be ulcerative or non-ulcerative, but the ulcerative form is most common and is due to direct cytopathic effects of the virus. The non-ulcerative form (stromal keratitis) is primarily immune-mediated, occurs in response to viral antigen, and is characterized histologically by lymphocytic infiltrates. Corneal ulcers are most often superficial and irregular or geographic (map-like) in appearance. Linear to tree-branching ulcers (dendritic ulcers) are considered pathognomonic for FHV but are not the most feline ulcer in my experience. The author’s approach is to assume herpesvirus until proven otherwise, so antiviral treatment is recommended for most feline ulcers. Testing for herpesvirus is notoriously inaccurate, and a negative test in no way excludes herpesvirus as the primary causative agent. Stromal keratitis is immune-mediated, and improvement may occur with judicious application of topical steroids. In the author’s opinion, topical steroids should be used only if the cornea is negative to fluorescein stain and if prior antiviral treatments have been ineffective. Then, they should be used in conjunction with an antiviral agent.

Choices for topical antiviral agents include idoxuridine 0.1%, cidofovir 0.5%, and trifluridine 1% (Viroptic) solutions, ganciclovir 0.15% (Zirgan) gel, and acyclovir 3% or vidarabine 3% ointments. Of these preparations, only trifluridine and ganciclovir are commercially available for topical use, and the others must be compounded. Idoxuridine is the author’s first choice for topical treatment. Most of the antiviral drops are applied q.4-6h. for initial treatment, and the frequency is reduced as the eye(s) improve. Cidofovir is an exception and is typically applied q.12h.

Famciclovir (Famvir) is available for oral use as 125 mg and 250 mg tablets. Studies have shown it to be safe and tolerated well at doses of 40 mg/kg q.8h. for up to three weeks. The safety of long-term administration has not been evaluated. A CBC and biochemical profile is advised for initial treatment and periodically thereafter if long-term treatment is to be considered, as bone marrow suppression is possible. L-lysine is an amino acid that has demonstrated antiviral activity against FHV both in vitro and in vivo, though studies have yielded conflicting results. Several preparations are available for oral administration (e.g., Viralys, Enisyl, F, etc.). L-lysine may reduce the severity of FHV infection and the risk of recurrence, but L-lysine is not as effective as famciclovir or the aforementioned topical antiviral treatments.

Eosinophilic keratitis or keratoconjunctivitis
Eosinophilic keratitis (EOK) is an inflammatory condition of undetermined cause. The clinical appearance is that of corneal vessels in one or more quadrants with variably sized plaques of inflammatory cells that appear as pinkish-white or white corneal lesions. EOK can occur in one or both eyes, but unilateral involvement is most common. It occurs in cats of all ages with no proven breed predisposition, though it tends to occur more often in young to middle aged, neutered male, cats. The differential diagnosis should include FHV keratitis, neoplasia (i.e., squamous cell carcinoma), and mycotic infection, but the latter two conditions are rare. Fluorescein may be retained over inflammatory lesions, but affected cornea is usually thicker rather than thinner as would be expected for a herpetic ulcer. Also, cats with FHV keratitis are typically more uncomfortable than a cat with EOK. The diagnosis is easily confirmed by corneal (or conjunctival) cytology. Cytologic findings usually reveal a predominance of eosinophils, occasional mast cells, and a variable number of lymphocytes and plasma cells. Eosinophils and mast cells are not a feature of normal cornea, and the presence of even one eosinophil on cytologic examination is considered significant.

EOK is a disease to be controlled rather than cured. Recurrence is common, so the goal should be to find the lowest level of treatment necessary for control. Topical corticosteroids are the primary treatment, and 1% prednisolone, 0.1% dexamethasone, or 0.1% betamethasone preparations are preferred. Concurrent FHV infection can complicate treatment, especially if topical steroids are
used. Studies indicate that 50% or more of cats with EOK can be positive for FHV by immunofluorescent testing or PCR. For this reason, empiric antiviral treatment is appropriate for some cats with EOK. The author often uses topical idoxuridine concurrent with topical steroid treatments, usually at the same frequency. Steroids drops should be applied 2-4 times daily in initial treatment and then tapered as the condition improves. Subcutaneous or intramuscular injection of 20 mg methylprednisolone acetate (Depo Medrol) may be an adjunct to topical steroid treatment. Additional treatment options include topical cyclosporine or tacrolimus, and oral megestrol acetate. Response to topical cyclosporine or tacrolimus is variable in the author’s experience and in some cats ineffective. Oral megestrol acetate is usually effective but FDA approved for use in cats. Oral megestrol is not usually the first treatment choice because of possible complications that may include diabetes mellitus, metritis, mammary neoplasia, pyometra, weight gain, and behavioral changes. When used judiciously, weight gain is the only adverse effect that the author has routinely encountered. For the average 10-lb cat, a typical dose regimen is 5 mg p.o. once daily for 5-7 days, then 5 mg every other day for two weeks, then 5 mg once weekly for two weeks, then 5 mg once or twice monthly for maintenance. The lowest effective dose of megestrol should be used for maintenance. It is best to limit megestrol treatment to those cases where topical treatments are ineffective or deemed too risky. Biochemical profile is advised prior to treatment to assess glucose and liver enzymes, and caution is advised in older cats where the risk for diabetes may be greater.

**Corneal sequestrum**

Corneal sequestrum is a condition unique to the cat and has been reported rarely in other species. The exact cause is undetermined, but predisposing factors include corneal ulcers (usually chronic), lagophthalmos, entropion, and tear film abnormalities. Sequestra appear to represent an abnormal healing response and are probably preceded by corneal ulceration in most instances. Because most feline corneal ulcers are the result of FHV infection, it is not surprising that FHV has been isolated from sequestra. Toxoplasma gondii has also been detected in sequestra removed by keratectomy. In a study of nine cases, 33% of sequestra were positive for FHV by PCR, and 33% were positive for T. gondii. The significance of these findings is unclear. There is also a well-recognized breed predisposition for Persians, Himalayans, and Siamese cats, but any breed can be affected. Sequestra can be unilateral or bilateral. They appear as an amber, brown, or black plaque, of the central or paracentral cornea, but can occasionally occur in the peripheral cornea. There is usually concurrent corneal ulceration, vascularization, ocular drainage, and varying degrees of patient discomfort. Ultrastructural studies indicate the dark material in a sequestrum is consistent with melanin granules. Histologically, sequestra are characterized by degenerate collagen, ulceration, and a variable degree of inflammation. Blood vessels often extend to the lesion but do not infiltrate the sequestrum. The clinical appearance can be considered pathognomonic because corneal pigmentation (or pigmentary keratitis) is rare in the cat, and epibulbar melanoma occurs at the periphery (or limbus).

Surgery is the most expeditious way to resolve a sequestrum. For cats that are painful and for which the pain cannot be resolved with medical treatment, surgery is mandatory. A topical antibiotic is appropriate, and an antiviral drop (e.g., idoxuridine) should be considered for aforementioned reasons. Ocular lubricants can be helpful, and mucinomimetic agents containing hyaluronate or carbomer are recommended. Oral analgesic (buprenorphine) or analgesic/anti-inflammatory treatment (e.g., Metacam) may also be indicated for initial treatment. Medical management alone is acceptable for pets that are comfortable and for which the eye is minimally inflamed. Some sequestra will eventually slough from the corneal surface, but months are usually required before this occurs. It is also possible for a sequestrum to migrate deeper in the cornea, and globe rupture occurs in a small percentage of cases. The sequestrum can usually be removed by lamellar keratectomy, but a conjunctival pedicle graft or corneoconjunctival transposition may be required. Sequestra can recur after surgical removal, and it is important to advise clients accordingly. The literature suggests there is no difference in recurrence rate in pets that have had a graft procedure versus those that have not; this is contrary to the author’s experience, and a graft procedure seems appropriate in most instances.

**Glaucoma**

Glaucoma occurs less often in cats than dogs. The prevalence of feline glaucoma in the absence of antecedent ocular disease (i.e., primary glaucoma) appears low. Most cats develop glaucoma secondary to chronic uveitis or ocular neoplasia with the exception of aqueous misdirection discussed below. In cats, progression of glaucoma is usually insidious and florid clinical signs are often absent. Innate and poorly understood properties of the feline eye make it less prone to marked corneal edema, breaks in Descemet's membrane, and marked retinal atrophy that typify chronic glaucoma of dogs. The condition is often not suspected until buphthalmos is present. The incidence of secondary glaucoma appears higher in cats with idiopathic uveitis than in cats with uveitis associated with systemic disease. The pathogenesis is related to chronic lymphocytic and plasmacytic inflammation and infiltration of the ciliary cleft and trabecular meshwork with inflammatory cells. Because most cats with glaucoma have concurrent uveitis, drugs that cause miosis are generally avoided (e.g., demecarium, pilocarpine, latanoprost), as they may enhance the uveitis. Such drugs may be counterproductive with aqueous misdirection because of potential for pupil-block glaucoma. Latanoprost (Xalatan) works by increasing uveoscleral outflow from the eye, but the percentage of uveoscleral outflow in cats is low compared with dogs. One study indicated that latanoprost does not reduce the IOP of normal cats, and there is evidence it may be counterproductive in cats. The author typically only uses timolol, dorzolamide, or combination dorzolamide/timolol (Cosopt) in cats. Topical carbonic anhydrase
inhibitors such as dorzolamide (Trusopt) and brinzolamide (Azopt) are effective when used once or twice daily, but an occasional cat will develop lethargy or anorexia with these drugs. Timolol 0.5% solution is a non-selective beta blocker that is tolerated by most cats but should not be used in cats with asthma. It can also be used once or twice daily as can the combination dorzolamide/timolol drops. Blind and painful eyes are best managed by enucleation, and histopathology of the globe is advised. Diffuse iridal melanoma accounted for approximately one-third of all cases of feline glaucoma in one study. Ciliary body ablation (aka chemical ablation) with intravitreal injection of gentamicin or cidofovir is not recommended in cats because of their potential to develop primary ocular sarcoma. This is a rare but highly malignant tumor that is unique to the cat.

Aqueous misdirection occurs when aqueous is misdirected into the anterior vitreous; this results in gradual forward displacement of the lens, decreased anterior chamber depth, and increase in intraocular pressure. Lensectomy can be performed in affected cats, but topical dorzolamide treatment is effective for control of the intraocular pressure in most cats.

Hypertensive retinopathy
High blood pressure (systemic hypertension) is the most common cause of retinal detachment, hyphema, and acute blindness in cats. Most affected cats are over 10 years of age and have systolic blood pressures in excess of 200 mmHg (normal is below 160mmHg). Sustained elevation in blood pressure result sin hypertensive retinopathy. It is estimated that 80-100% of hypertensive cats have some degree of retinal disease. Ophthalmoscopic findings include tortuous retinal vessels, focal areas of retinal edema or hemorrhages, or complete detachment with extensive hemorrhage. Retinal degeneration develops with long-term detachment. Systemic hypertension can occur as a primary disease (i.e., no obvious cause) or secondary to renal failure (the most common cause in cats), hyperthyroidism, diabetes, etc. Approximately 50% of hypertensive cats have concurrent heart disease, and about 30% will develop neurologic signs without treatment.

The extent and duration of retinal detachment, and concurrent hemorrhage, determine the prognosis for return of vision. Retinal detachments of long duration (e.g., more than a week or so) or extensive intraocular hemorrhage is associated with a worse visual prognosis. Affected cats may have concurrent uveitis or glaucoma in association with the posterior segment disease and then require additional specific topical treatment. Amlodipine (Norvasc) at ¼ of a 2.5 mg tablet orally once daily is a good initial treatment and recheck of blood pressure advised in a few days. Additional treatments may be necessary to control the hypertension, and an assessment of the cat’s general health is appropriate (e.g., biochemical profile, thyroid testing, etc.). With prompt diagnosis and treatment, the retinas can reattach and the cat can regain useful vision.

Iris melanosis and melanoma
Cats of all ages may develop pigmented lesions of the iris in one or both eyes. In the early stages, focal pigmented lesions might be called an iris freckle or benign iris melanosis. In most cats, such pigmented lesions are static or progress slowly over months or years. These lesions can be a precursor to uveal melanoma. Fortunately, feline uveal melanoma does not tend to metastasize until late in the course of the disease. Many cats can retain discolored but comfortable and visual eyes for years. Ocular signs that suggest progression to melanoma include diffuse iris pigmentation, iridal thickening, dyscoria, uveitis, evidence that melanosis has extended into the filtration angle (via gonioscopy), and secondary glaucoma. One study indicated that cats with uveal melanoma and glaucoma have decreased survival times compared with cats in the general population. My approach is to provide guidelines regarding the best time for enucleation, but the client must be comfortable with this approach and understand there is no guarantee against metastasis. Periodic examination is advised by an ophthalmologist to monitor progression of the melanosis.
Common Orthopedic Soft Tissue Injuries of the Front Limb

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Forelimb injuries are common in the canine; unfortunately, many of them go undiagnosed or considered a “soft tissue injury” without a true diagnosis. The 3 most common orthopedic soft tissue injuries of the front limb are supraspinatus tendinopathy, biceps tendinopathy, and medial shoulder syndrome (MSS). With many forelimb lameness’s two major questions need to be asked: is this orthopedic or neurologic? If this is orthopedic then is it the elbow, shoulder or both? For many cases there can be an isolated shoulder problem, isolated elbow problem, or a primary problem with both, or a primary problem and then compensation to the other. While it is not the intention to cover every orthopedic condition in the forelimb it is important to learn to differentiate between shoulder and elbow lameness. A good solid orthopedic examination is key. For the elbow is there a reduction in range of motion? Does the dog resist elbow extension that could indicate an un-united anconeal process (UAP), is there a reduction in flexion that could indicate medial compartment disease (MCD) such as a fragmented coronoid process (FCP), is there any muscle spasm or tightness of during ROM in the elbow especially on the biceps? Is there pain on palpation of the medial aspect of the elbow specifically during flexion and supination (Campbell’s test) to indicated MCD? For the shoulder am I able to fully extend the shoulder, if not could there be an osteochondritis dessicans (OCD) lesion, can I extend the shoulder and abduct it with no pain or spasm, if not is there evidence of MSS? Can I fully flex the shoulder with no pain or spasm; can I palpate the supraspinatus and/or biceps with no pain or discomfort? Can I complete a biceps stretch test? The biggest thing to remember is we are not trying to make the patient cry out or scream, but rather we are looking for subtle signs of muscle spasm, reduction in range of motion, and possibly discomfort. Even the most stoic dog will show signs of muscle spasm or tightness. Don’t forget about some of the other players that can mimic orthopedic conditions such as a brachial plexus injury/tumor, a cervical lesion causing root signature pain, or even neoplasia.

It has been common practice to diagnose a “soft tissue injury” for forelimb lameness’s that we can’t seem to figure out. In many cases these injuries don’t resolve or become chronic. We should make every attempt to identify these injuries and move away from using the term “soft tissue injury” and figure out the problem. Dogs that tend to present with forelimb injuries are the “weekend warriors”, these are the dogs that lie around all week then become very active on the weekends. Given the lack of conditioning however, it does provide stabilization of the shoulder during the stance phase. It has been common practice to diagnose a “soft tissue injury” for forelimb lameness’s that we can’t seem to figure out. In many cases these injuries don’t resolve or become chronic. We should make every attempt to identify these injuries and move away from using the term “soft tissue injury” and figure out the problem. Dogs that tend to present with forelimb injuries are the “weekend warriors”, these are the dogs that lie around all week then become very active on the weekends. Given the lack of conditioning however, it does provide stabilization of the shoulder during the stance phase.

The diagnosis of supraspinatus tendinopathy is not well described and the prevalence seems to be increasing, this is likely due to advanced imaging such as MRI or diagnostic musculoskeletal ultrasound. These dogs will typically have a unilateral forelimb lameness that appears worse after exercise and heavy activity. They tend to be minimally responsive or non-responsive to NSAIDS and exercise restriction. Many will appear to improve during exercise restriction only to become lame after returning to normal activity. In some cases of biceps tendinopathy there may be a partial response to administration of an intra-articular steroid. However, this response is typically on temporary. It has been suggested that Rottweiler’s and Labs are predisposed to supraspinatus tendinopathy; however, this was suggested in the late 70’s early 80’s. At our practice we tend to see many Labs, and Border Collies with this condition. Currently the pathogenesis of both conditions is unknown; however, it has been demonstrated that there is a small distinct area of hypovascularity in the tendon of the supraspinatus. This in conjunction with repetitive microtrauma could result in microtears of the tendon and thus lead to the formation of fibrous tissue and in chronic cases calcification in the tendon. Even though the true cause is unknown overuse, which has been described in both human and animal models, is highly suspicious. The repetitive trauma results in a proliferative nodule; the typical inflammatory changes are not seen. During repair there is a poor or dysfunctional response possibly due to the lack of blood flow. The mechanical properties tend to deteriorate resulting in a decreased modulus of elasticity and maximal stress till failure. The origin of the biceps at the supraglenoid tubercle is said to be an area of hypovascularity, which may predispose it to mechanical failure causing either fraying or rupture. Given that the typical inflammatory changes are not seen histopathologically the term tendinopathy is better used then tendinitis or tendinosis. Because of the lack of inflammatory response patients require lengthy management and often respond poorly to treatments.

Supraspinatus and biceps tendinopathy

The supraspinatus is a large muscle that originates at the supraspinatus fossa and inserts on the greater tubercle. It is responsible for shoulder extension and is an active stabilizer of the shoulder. It has been shown to be active during about 65-80% of the stance phase. The biceps originates on the supraglenoid tubercle of the scapula then courses distally to insert on both the radius and ulna. The insertion becomes important when discussing potential medial compartment disease. Its primary function is for elbow flexion; however, it does provide stabilization of the shoulder during the stance phase.

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In looking at the distribution of the diagnosis of supraspinatus tendinopathy it has been shown just a few years ago there were very few cases with a supraspinatus diagnosis. For example in unpublished data from our practice of 327 cases, in 2006 there was less than 6 cases diagnosed with supraspinatus tendinopathy versus in 2013 there were over 90. Around 2010 is when the use of MRI and diagnostic musculoskeletal ultrasound (MSK US) became more commonplace and we began to recognize this disease condition. However, it could also be suspected that with the advent of more advanced imaging the diagnosis of supraspinatus injuries is being over-diagnosed.

From an examination standpoint we want to begin evaluating the dog standing and examine for symmetry of the supraspinatus muscles. Muscle atrophy can be one clue into a chronic problem. From an objective standpoint we want to take muscle mass measurements and compare it to the contralateral side. Furthermore, many of these dogs will have a decrease in shoulder flexion and will resist a biceps stretch test. Goniometry should be measured and compared to the contralateral side; normal shoulder flexion has been reported to be 54-59 degrees. During shoulder flexion palpate the insertion of the tendon for any discomfort, pain, or spasm or for the dog to begin pulling the shoulder away. Subtle changes noted may be a change in breathing (panting then stops), the pupils may dilate or the dog may begin licking its lips. Examination of the biceps tendon may reveal a tendon that is thickened and painful; furthermore, there may be decreased muscle mass in the affected forelimb. One of the biggest clues is a response to the biceps stretch test. This is completed by having the shoulder flexed and then placing the elbow into full extension. This will put a direct stretch onto the biceps. Many patients with a biceps tendinopathy either will not let you complete the biceps stretch test or they will be begin to show signs of discomfort and spasm.

After a though physical examination radiographs are typically the next course of action. In acute cases the radiographs are typically normal; however in chronic cases there can be mineralization in the area of the supraspinatus tendon or in the area of the bicipital groove. Granted while this is a nice finding it is very rare. The goal of radiographs is to help rule out other potential issues such as an OCD lesion, osteoarthritis, osteosarcoma, fracture/luxation, etc. MRI is most helpful in acute cases where increased signal intensity can be seen; however, MRI is expensive and requires general anesthesia. Furthermore, it is not very useful to perform recheck MRI’s to gauge healing. MSK US is another option to evaluate the canine shoulder. It will give us the tissue architecture so that we can determine if this is a true tendinopathy or an insertionopathy. MSK US may us to determine the severity (is this a grade I, II, or grade III lesion). Based on the grade of the lesion, it can help us to make treatment recommendations. Furthermore, following treatment we may be able to monitor healing by seeing a reduction in inflammation as well as a more normal appearance of tissue architecture. One big issue is that both advanced imaging modalities are very user dependent especially MSK US. Even the radiologists are still debating the best usage of these technologies as to what information we can get, what information we can’t get, and how to best achieve what we are looking for.

Arthroscopy is not commonly used to diagnosis a supraspinatus tendinopathy; however, it can be useful to evaluate for concurrent conditions. The supraspinatus is extracapsular so it can’t be viewed directly; however, with inflammation and swelling of the supraspinatus a bulge can be seen just adjacent to the biceps. MSS is commonly noted in cases with a supraspinatus strain. Arthroscopy can be utilized as both a diagnostic and therapeutic (biceps release) modality in patients with a biceps tendinopathy.

Previous treatment recommendations consisted of surgical excision of the calcification; however, this was met with variable success rates and the mineralization returns in almost all cases. Furthermore, when surgical removing the calcification one is also removing a portion of an active shoulder stabilizer. Extracorporeal shockwave therapy was an effective treatment in 2 dogs with chronic cases. At the author’s facility our current treatment recommendations are based on the exam, MRI, and MSK US findings. If I have a companion that is a first time offender with mild exam findings I will many times place them on an NSAID and/or muscle relaxer for 2 weeks with 4-6 weeks of exercise restriction AND formal rehabilitation therapy. Exercise restriction will prevent the issue from getting worse and help quiet down the inflammatory response. The formal rehabilitation will allow us to stretch out the tissue and facilitate healing. For the athlete, or companions that have failed the first go around of conservative management or patients with more moderate exam findings I will recommend either an MRI or MSK US. From the MSK US if it is a grade I (or they have not undergone rehab) we will recommend exercise restriction and formal rehabilitation therapy. For grade II lesions we will recommend regenerative medicine (PRP or stem cell/PRP) with an exercise restriction period of 8-12 weeks. Also, if the MSK US suggests any intra-articular pathology then we may recommend scoping the shoulder to address any other underlying conditions.

Rehabilitation for the supraspinatus and biceps tendinopathy is initially geared at releasing of trigger points, stretching and massage. Therapeutic US therapy can be used to heat the tissues to allow maximal stretching while laser therapy can be used to improve blood flow and stimulate healing while reducing inflammation. In the later stages of the supraspinatus and/or biceps strain we will incorporate UWT therapy; however, open water swimming should be monitored very closely as to not exacerbate the condition. Therapeutic exercises are initially isometric and geared towards improving fatigue of the muscles, after progressing from this stage eccentric exercises can be used. Regenerative medicine is a current promising treatment modality for supraspinatus and/or biceps tendinopathy. We tend to use combination therapy of stromal cell and PRP. The stromal cells are harvested from either fat or bone marrow. They help to diminish tissue injury, promote neovascularization, recruit and induce proliferation of resident stem cells, inhibit
fibrosis, act as a source of growth factors, and they are anti-inflammatory. PRP provides a great source of growth factors responsible for promoting angiogenesis, enhancing cellular proliferation, promoting extracellular matrix formation.

From a prognosis standpoint there is limited reported data in the literature. A study published recently from VOSM revealed that in dogs with grade I lesions or first time offenders we see about 42% respond to exercise restriction and rehabilitation therapy. Typically I tell owners we have about a 50% chance of improving their dog with exercise restriction and rehabilitation. Grade II or repeat offenders treated with regenerative medicine have well over an 82% response rate with the addition of exercise restriction, rehabilitation, and regenerative medicine. The biggest issue is many supraspinatus injuries may be secondary to another disease condition. So if a patient fails to return to normal activity then additional diagnostics to evaluate the joint are warranted.

**Medial shoulder syndrome (MSS)**

I tend to refer to the medial aspect of the shoulder as the “dogs rotator cuff” which is probably way off, but owners tend to understand it better. The shoulder joint itself is stabilized by both active and passive stabilizers. The active stabilizers include the supraspinatus, infraspinatus, and others (which is why many patients with MSS have concurrent supraspinatus tendinopathy). The passive stabilizers are the medial glenohumeral ligament (MGL), joint capsule, and subscapularis tendon along with others.

Much like supraspinatus and biceps tendinopathies, dogs suffering from MSS typically have an open diagnosis of unilateral forelimb lameness that is worse after exercise, and tends to be non-responsive to NSAIDS and exercise restriction. What makes this even more challenging is that some of them will have no lameness at all except during specific activities, which is commonly noted in agility dogs. You may note a shorten stride length or step length and owners may complain about them knocking bars, pulling up or refusing weave poles or taking wide sweeping turns. On big question is if MSS is a common occurrence or if it something rare? I think it is a common problem not only in the agility world but also in the weekend warrior that is not conditioned and goes out and over does himself or herself. But I believe also that it is rarely diagnosed and usually missed for some type of “soft tissue injury.” The MGL plays a role in shoulder stability and Dr. Bardet found that 69% of dogs with chronic forelimb lameness had pathology of the MGL; furthermore, MGL pathology is the most common form of shoulder instability. Unfortunately, there is no real information out there as to the clinical significance of damage to just the MGL. For example, one study looked at the stability of the MGL and found that by transecting the cranial arm there was no increased instability in the shoulder. It did result in marked inflammation and the authors speculated that with continued repetitive motion that overtime this could result in shoulder instability. So, this brings to light what structures really need to be damaged before it causes a clinical issue. Could a complete tear of the MGL result in instability, a complete tear of the subscapularis, or do both have to be torn? What if they are frayed or stretched, at what point does this result in a clinical issue? These are issues that have yet to be determined.

Dogs with MSS may show no evidence of lameness at presentation or may have a severe unilateral forelimb lameness. On examination one can appreciate pain on shoulder extension with abduction; along with pain there may be spasm on the medial aspect of the humeral head when palpated. With goniometry an increased abduction angle is noted. The typical normal angle is usually 30-32 degrees whereas dogs with MSS may have 50 or greater degrees of shoulder abduction. However, it is very important to compare to the contralateral side and in many cases this is a unilateral disease. Recently the use of abduction angles in determining MSS has been called into question. Jones, et al. presented a study where they looked at the accuracy and precision between and among observers as well as between fluoroscopic measured abduction angles. They found there was poor accuracy between the observer measured abduction angles and the fluoroscopic measured abduction angles. In addition the mean abduction angle with the intact medial structures was 28.34° whereas the mean abduction angle after complete transection of the MGL and subscapularis tendon was 35.63°. This means there was only about 8° of difference in abduction angles between cadavers with completely intact medial structures versus the same cadavers with completed transected medial structures.

Radiographs are typically normal in dogs with MSS; in really chronic conditions there may be some mineralization in the area of the supraspinatus and/or biceps if there is a secondary tendinopathy. MRI and MSK US are more beneficial for showing secondary supraspinatus or biceps tendinopathy; however dynamic instability can’t be seen with MRI. MRI can be used to pick up on tears of the MGL and subscapularis tendon. MSK US may reveal joint effusion, and a thickened or irregular joint capsule that is suggestive of intra-articular pathology; however, MSS can’t be fully differentiated from other types of intra-articular issues. Shoulder arthroscopy is beneficial for not only the diagnosis of MSS but also treatment in select cases. Arthroscopy allows for direct intra-articular observation as well as dynamic evaluation of tissues during ROM. During arthroscopy we are able to palpate the intra-articular structures to evaluate for any laxity.

The treatment of dogs with MSS is very controversial. Some consider this to be a surgical disease while others consider it to be a conservative/rehabilitative disease. From a surgical standpoint there is the option of thermal capsulorrhaphy (radiofrequency), prosthetic reconstruction, biceps transposition, or subscapularis imbrication. With thermal capsulorrhaphy a heat source is applied to the tissues to cause reorganization of the collagen. In other words it “shrinks” or “tightens” the tissue. This is accomplished by using a monopolar radiofrequency probe at 25W and 70°C in a striping technique. It creates scarring and the recovery of the tissues as well as immobilization is very long (5-6 months). One has to be extremely careful when performing this as permanent damage can be created

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to the tissues. Initially, Dr. Cook found a 93% improvement in patients treated with radiofrequency. A more recent study by Dr. Franklin found an 80% improvement with radiofrequency; however, the improvement was not significantly better than the patients that were treated non-surgically. In addition, thermal capsulorrhaphy is under intense review in human medicine due to the number of complications and the number of lawsuits. The American Association of Orthopaedic Surgeons recognizes the higher complication rate and lack of long-term efficacy. In addition most insurance companies consider it not to be medically necessary and will not provide coverage for it.

Prosthetic reconstruction is probably a better and safer alternative to thermal capsulorrhaphy. This can be done as an open procedure or as an arthroscopic assisted procedure. A recent study with at least 6 month follow up data reported a success rate of 93% and a complication rate of 15%. In addition, another paper showed that patients treated with surgical reconstruction have a higher likelihood of a successful outcome compared to non-surgical management. One problem area is trying to determine what patients benefit from surgery and which patients benefit from conservative management. In personal discussions with Dr. Peter Lotsikas, he seems to think that patients with complete subscapularis tears or multidirectional instability benefit the most from surgical reconstruction.

Conservative management is my initial approach (unless the condition is severe) where I place these patients in hobbles and place them in a formal rehabilitation therapy program for 8-12 weeks. Other conservative treatments such as extracorporeal shock wave therapy or regenerative medicine (stem cell/PRP) can be considered in addition to hobbles and rehabilitation therapy. From a regenerative medicine standpoint I may tend to inject PRP (my preferred starting point) or stem cell/PRP. Following this they are placed in hobbles for 4-6 weeks followed by continued strengthening via rehabilitation for an additional 4-6 weeks. Another modality that can be used for the conservative management in conjunction with hobbles and rehabilitation therapy is using extracorporeal shock wave therapy at the insertion point of the proximal humerus on the medial side as well as the glenoid cavity. This is completed initially, then 2 weeks later. At the 4 week mark they come out of their hobbles and begin strengthening exercises. In discussing this with Dr. Darryl Millis he sees about a 75% success rate

Above all else, formal rehabilitation therapy seems to be the most critical factor in success. The basic modalities can be used such as laser therapy, therapeutic ultrasound, etc. However, most of the improvement is going to come from the incorporation of exercises that strengthen the shoulder. Things such as the balance board, lifting the opposite forelimb or hindlimb, weight shifting, standing on a balance ball, walking in figure 8s or circles, and scapular stabilization exercises. Initially, this is done with the hobbles on then over 4-6 weeks the exercises are transitioned to having the hobbles off.

References
It Happens to the Best of Us: Orthopedic Complications and How to Address Them
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When it comes to orthopedics complications do and will occur even to the best of us. Unfortunately it’s one of the costs of doing business. However, there are ways to minimize complications that we will discuss such as having the appropriate diagnosis, wearing the appropriate personal protection equipment (PPE), having the appropriate instruments, and having the appropriate knowledge base. Also important is assessing outcome measures so that issues can be picked up and dealt with early. If a complication does occur one needs to be able to identify the complication as well as know some specific complications to be on the look out for such as infection, implant breakdown, or poor bone healing. Lastly, in the unfortunate event that a complication does occur then knowing how to deal with it is key. I think in a general sense it is much easier to prevent a complication from occurring than to deal with a complication. I go to great lengths to prevent complications as I don’t want to deal with it later nor do I want to deal with the increased morbidity of the patient or the frustration of the owner. To me what separates a great surgeon from a good surgeon is not the skill level of the surgeon, but the ability to anticipate trouble and be able to deal with a complication should it occur.

Orthopedics can be very fun, but at the same time can be very frustrating. For me I prefer it to be fun. Granted there are some frustrations that are unavoidable and I tend to cuss like a sailor. However, many frustrations are very avoidable. To minimize frustrations in the OR the correct approach is essential as well as the knowledge of the surgical procedure. Equipment is key as trying to use the wrong equipment will make life hard; so if your going to be doing lots of orthopedics I would suggest buying the correct instruments to use. Assistance is very helpful in the OR and visualization is key. You cant treat what you can’t see so having the knowledge of the approach is very important to ensure appropriate visualization.

Minimizing complications
The very first step in minimizing chances of complications is through achieving the correct diagnosis. The path to achieving the correct diagnosis is through a great physical, orthopedic, and neurologic examination. Remember, your hands and your brain are the best tools you have (and there is no overhead expense to these things!!). Once you have an idea of what’s going on, then with orthopedics additional diagnostics are commonly needed. Radiographs are the mainstay for orthopedic diagnostics; however, in some situations advanced imaging is needed such as a diagnostic musculoskeletal ultrasound, CT, and/or MRI. Of importance, a normal radiograph does not mean that pathology does not exist so it is important to know the limitations of the diagnostics that are being recommended.

As was just stated: radiographs are mainstay for orthopedic issues. However, one must take orthogonal views to determine and evaluate the extent of any injuries. This includes at least a lateral and AP radiograph to tell the whole story. I always take not only the injured limb but also the contralateral limb to look for differences. CT scans can be helpful especially with sacral fractures, spinal fractures, articular fractures, etc. Think of a CT scan like giving additional bony information in 3D. Both diagnostic musculoskeletal ultrasound and MRI are helpful for identifying soft tissue injuries such as tendinopathies, joint disorders, etc. Lets create a common case scenario: “Fluffy” is a 6 month old Yorkie that belongs to a long term client: Mrs. Smith. “Fluffy” jumped off the cough when the doorbell rang. She calls to see you immediately because he is holding the right front limb up. As usual your triple booked during your lunch but you agree to get her in anyway. If you have your technician get “Fluffy” to take radiographs and they only take a lateral of the shoulder and elbow, then the distal humeral fracture can be missed. In this case you wont see the fracture and will suspect a soft tissue injury and send “Fluffy” home on an anti-inflammatory. Had you taken orthogonal views you would have diagnosed the a Salter-Harris IV lateral condylar fracture. Knowing that this is an articular fracture would mean that surgical intervention is required and it is much easier when it is acute rather than if it is chronic.

Another aspect not to be overlooked when it comes to minimizing complications is appropriate PPE and patient preparation. With any surgery, but especially orthopedics a cap, mask, shoe covers, and gown are required. In addition, an appropriate hand prep through either traditional scrubbing, or alcohol based rubs is essential. Don’t forget about the patient when it comes to minimizing complications. There needs to be the pre-surgical assessment completed. In our hospital every morning during rounds we will shave a small area of hair around the surgical site to evaluate for any skin pathology. We will also, do a full examination in-depth look to ensure there is no evidence of pyoderma. Never complete an elective orthopedic surgery in a patient with pyoderma, especially if close to where the surgical incision will be. Once deemed appropriate for surgery and after anesthesia then the surgical site is shaved and prepped. Shave wide areas, we don’t want hair in the surgical field. Use caution in preventing razor burn, some speculate this could be a cause of surgical site infections (SSI). I have canceled surgeries due to severe razor burn. If razor burn is noted then it should be documented in the anesthesia record as an adverse event. Once shaved a rough prep is completed in the preparation room prior to moving the patient to the operating room. Once in the operating room the sterile prep is completed. The entire patient should be
draped in appropriately to create a sterile surgical field. Above all else bacteria LOVE implants and most of the bacteria come from the patient’s own skin. Use extreme caution not to rub instruments, suture, or implants through the skin (even the sterile prepped skin).

The surgical approach is the first step in a successful orthopedic procedure. The correct technique during the approach can lead to better visualization, ease of surgery, shorter surgery times, along with less tissue trauma and less morbidity. By causing less tissue trauma patients will be more comfortable and likely to use the limb sooner. If your going to do lots of orthopedics and want to minimize complications I would recommend investing in a good approaches book to the bones and joints of dogs and cats. And then, read over the approach prior to every surgery. Even as a surgeon now I still do this so I can create a visualization in my mind of what tissues I am dissecting.

Having the correct equipment is crucial for various types of orthopedic surgeries. As crazy as it sounds, make sure the equipment is sterile before you go into the operating room!! Just as important as having the correct equipment, the knowledge of how to use the equipment is vital. Trying to make an instrument do something it is not designed to do is a sure fire way to introduce frustrations and potentially a complication. Orthopedic instruments are expensive so if you don’t have the correct instruments or are unsure if what you have will be appropriate for that particular patient know the limitations and know when to refer the patient. Assistance is vital in the OR to minimize complications. Unfortunately, orthopedics is hard to do solo. You only have 2 hands, but most procedures require 4. If you are trying to retract as well as perform the procedure this will increase the likelihood of a complication occurring from lack of visualization.

Communication is the key to everything in life especially the practice of veterinary medicine. It is important to always discuss complications with owners and let them know that it is always a risk factor. Furthermore, address with the owner what complications can occur with the particular type of surgery you are recommending, how the owner will identify that something is wrong and how complications will be dealt with should they occur. I find that if I am very open and blunt on the front end it is much easier to deal with the issue on the back end should something occur.

Assessing outcomes
To know that we are truly improving, but to also know if we are not improving there needs to be the ability to assess outcomes from both a subjective and an objective standpoint. As health care professionals we often think that our patients are doing better than they really are because we all want our patients to improve. Assessing outcomes is also important to help make the decision to improve or change protocols. Additionally, outcome measures allow the veterinarian to pick up on complications. If a patient is not improving from an outcome standpoint it makes no sense to continue doing the same thing over and over. Instead the patient needs to be re-examined to figure out why their outcomes are not improving. There are a variety of ways to assess outcome such as the patients ability to return to function, improvement in their gait, improvement in joint function, improvement in muscle mass and range of motion, and improvement in pain. You can also take into consideration the owners or veterinarians impression. I would however, use extreme caution when taking into account the impression of others as it has been shown that visual impressions and subjective scales don’t correlate with findings of objective information.

Return to function is probably the best indicators of a successful outcome. If a dog is able to return to its previous function then I would say that the patient has healed. In some cases we wont know just how successful we are till we complete the post-operative and rehabilitation period and then go back to normal function. I call this the “sink or swim” period. Most patients will do really well during exercise restriction and formal rehabilitation therapy post operatively, but in some cases we wont know just how successful we were till get them back to normal activity. With some injuries we can’t expect the patient to completely return to full normal function so expectations may have to be adjusted. Our goal then should be to make them comfortable, but they will always have periods of stiffness, soreness, and lameness.

For companions in addition to the owner’s perceived improvement there are various pedometers, accelerometers, and GPS devices that can be used to give an estimate of the amount of activity at home. For canine athletes this can best be determined from them getting back to competition and can be determined when comparing to their previous times. For example fly-ball is easy to assess outcomes because the times are more reliable as the course does not change. However, for agility, lure coursing, and others it can be a bit harder since courses and obstacles can change. In these cases it is better to measure performance against common competitors.

Gait is something that is very important when it comes to assessing outcomes. Most commonly it is evaluated subjectively; there are various scales out there for usage. The nice thing about subjective gait assessment is that it is quick, inexpensive, and requires no equipment. However, experience is very important and it can be challenging especially if multiple limbs are involved. The reason for this is the human eye tends to fall on the most obvious abnormality contributing to the asymmetry. Unfortunately, the sensitivity of subjective gait analysis is less when compared to objective gait analysis, and there is not a very good correlation between subjective and objective gait analysis. From a post-operative standpoint an individual also needs to know what is normal versus abnormal during the various stages of healing with various conditions. From a gait standpoint I tend to use a 0-5 scale to record the subjective aspect of things. Objective gait analysis can provide very detailed information about the patients gait and progression. There are various way to do this such as with pressure sensitive walkways, force plate analysis, or a stance analyzer. However, objective gait analysis is
expensive, there is a learning curve to operating the equipment and interrupting the results. Lastly, getting the owners impression of home activity such as when they are taking the dog on a walk, how their gait is immediately after rising from rest.

One cheap objective way to evaluate static weight bearing is with the use of a bathroom scale. Hyytiainen found a 39% sensitivity and 85% specificity of static weight bearing in dogs with hind limb OA. This was compared to force plate analysis during walking and static weight bearing. They concluded that it is a reliable, simple, and cost effective outcome measure for rehabilitation of dogs with OA of the hind limbs.

When evaluating joint function the goal is to look at the quantity of the motion in terms of what is the maximum range of motion (ROM) and what is the comfortable ROM. Remember that dogs don’t typically engage complete ROM during day-to-day activities. The problem in veterinary medicine is we don’t know how much of the maximum ROM a dog needs to use. For example normal hip extension is approximately 160 degrees, but I usually consider anything from 150-160 degrees to be acceptable in terms of daily function. At the same time that 150-160 degrees has to be comfortable ROM. Also, look at the quality of the motion. Is there crepitus present; is there pain during ROM? From an objective standpoint in assessing joint function I like to use goniometry. It is an easy test to perform and gives lots of information. For me I measure joint angles of not only the affected limb but also the contralateral limb so I have something to compare to. While there are published values for Labradors I think using the good contralateral limb is probably a better representation for that particular patient. Goniometry should be measured at the initial consult and at each recheck examination or various points during the post-operative and rehabilitation period. If ROM is not improving or declining this is an indication that an issue is getting worse, a potential complication is present, or the current post-operative rehabilitation program is not adequate.

Measuring muscle mass is another outcome measure that should be completed as it relates with muscle strength and is an indirect measurement of assessing changes in muscle mass. Unfortunately, there is not a perfect correlation between muscle mass and muscle strength. Currently, measuring muscle strength in veterinary medicine is very difficult. Furthermore, it is inexpensive, quick, and easy to perform. I usually tell owners the good saying of if you don’t use it you lose it is very true for orthopedic patients. For example following a CCL rupture it only takes about 4 weeks for the dog to lose about 30% of muscle mass. Given that muscle mass usually takes double the amount of time to return, that means it will take 8 weeks to gain that 30% back. The gold standard for the hind limb is measuring at 70% of thigh length, clipped hair, the limb in an extended position, and the animal relaxed. I think the biggest thing with muscle mass is that you stay consistent with the way you measure it especially with each patient. For me in conjunction with goniometry I measure muscle mass at the initial consult and at each recheck or various periods during the post operative and rehabilitation program. Again I think it is important to measure the affected limb as well as the healthy contralateral limb to establish the baseline for that particular patient. If muscle mass is not improving or declining this is an indication that an issue is getting worse, a potential complication is present, or the current post operative rehabilitation program is not adequate.

Pain is very important from an assessment standpoint and can help with detecting complications. For acute pain physiologic parameters tend to be more helpful. From a post operative and rehabilitation standpoint behavioral changes are more useful for chronic pain. Many different scales have been developed such as visual analog scores, ordinal scores, and questionnaires. For example there is the CBPI that has been validated in dogs with OA, the Helsinki chronic pain index, which is an assessment of chronic pain, the Glasgow composite pain scale, and the Univ. of Melbourne pain scale, which is an assessment of acute pain.

I think paying attention to outcome measures is one very important way to know if you patient is improving as expected during the post operative period, but to also pick up on complications early. From what we have discussed you can add gait analysis, goniometry, and muscle mass to your tool belt. If a patient was non-painful then becomes acutely painful they need to be rechecked immediately. In the overall picture if there are trends of a plateau or declining measurements then this could indicate a potential problem or complication that needs to be addressed. I would not recommend pushing patients through post operative rehabilitation with the hopes that measurements will suddenly improve. I am not a fan of the saying “just keep with rehab and they will get better” which unfortunately is the remark made by many veterinarians during the recovery period or from individuals that can’t figure out what the problem is.

Specific complications and how to deal with them
Complications as much as they suck happen to everyone, even the best. The big 3 I will cover are infection, implant failure, and poor bone healing. The easiest thing to do is blame someone else. Don’t be quick to blame the owner, the dog, or the particular plate. Many times the reason for the issue is standing right in front of you if you were to look in a mirror.

1) Infection
I think one of the difficult things when talking about orthopedic surgery is discussing complications. Infection is a risk of any surgery, especially open fractures or if there is a break in asepsis. Sterile technique is very important when placing implants. The ultimate goal is to prevent osteomyelitis and potential sequestrum from occurring. Infection can occur due to hematogenous spread, direct inoculation from an open fracture or surgical contamination or less commonly direct spread from a focal soft tissue infection. In the acute phase after a bone has fractured the vascular channels are comprised which results in ischemia. Bone ischemia is a major predisposing factor for osteomyelitis. This ischemia is one of the key reasons why antibiotics alone are rarely effective. Around the
bone ischemia is a reactive hyperemia that is associated with an increase in osteoclast production. Along with increased osteoclast production there is also periosteal irritation that leads to periosteal reaction. The aggressiveness of the infection is noted to parallel that of the periosteal reaction.

The damage and ultimately the response to treatment of bony infections is dependent on the viability and stability of the fixation, the virulence and antibiotic sensitivity of the organism and the condition of the soft tissue envelope. The most common type of osteomyelitis is from direct inoculation, which is also known as post-traumatic osteomyelitis. The staph species dominate with *S. intermedius* and *S. pseudointermedius* being the most common. Some gram (-) bacteria can be associated with osteomyelitis. Fungal organisms are usually due to hematogenous spread. For an infection to occur the bacteria must contaminate and colonize the bone and surrounding tissues. The recipe for this to happen is vascular compromise plus tissue ischemia plus bacterial contamination. Its important to note that a stable fracture/osteotomy will heal in the face of infection, an unstable fracture/osteotomy will not heal in the face of infection and will perpetuate the persistence of infection. This means that if the bone is not healing after an infection then antibiotic treatment was unsuccessful or the fracture site is unstable.

When it comes to the clinical signs of infection there can be variances between patients depending on the cause and the duration. In general many of these patients will have lameness, atrophy, pain on palpation, swelling, draining tracts, and/or lethargy. If an implant is ever placed and there is an acute lameness or swelling it should be checked immediately. Technically, the gold standard for diagnosing osteomyelitis is through a positive microbial culture. It is important to ensure that the culture is collected with care so the sample is representative. Never collect the purulent material or fluid draining from a wound directly from the skin. Surface bacteria will result in contamination (and remember that some dogs will carry MRSP on their skin). Ideally, submission of actual tissue or implant is preferred over submission of a swab of tissue or fluid.

The radiographic appearance of osteomyelitis will reveal bone resorption, periosteal proliferation, increased medullary density, and possibly a sequestrum. A sequestrum is a well-defined segment of sclerotic bone surrounded by a zone of reactive tissue or bone known as an involucrum. The cloaca is an opening in the involucrum in which material including the involucrum may escape. Draining tracts are usually from the cloaca to the skin.

Treatment for an osteomyelitis should be aggressive to prevent the issue from becoming a chronic problem. Treatment in a general sense includes establishment of drainage either through an active means such as a Jackson-Pratt drain or through a passive means such as a Penrose drain or open drainage with daily bandage changes and delayed closure. Debridement should focus on removing all necrotic tissue, which includes bone, muscle, soft tissue, etc. Following debridement there should be copious lavage with a sterile isotonic solution. Don’t mix in things such as chlorhexidine or iodine as this can cause cellular destruction. If implants are in place and the fracture/osteotomy has healed then the implants need to be removed. If there are implants in place without a healed fracture/osteotomy then the implants should be removed. In addition, the bone edges will need to be debrided so there is healthy bleeding bone. Following this rigid stability will need to be completed in addition to appropriate antibiotic therapy.

2) Implant breakdown

Implants are under a constant load while the bone is healing, in fact it is a race against time to get the bone to heal prior to any implant breakdown. Cyclic strain due to excessive motion can cause implant breakdown. This can occur from overuse of the limb or poor confinement. We all tend to assume that over activity or poor confinement is the cause of implant breakdown, but unfortunately, poor fixation choice and technique are probably more commonplace. Implant breakdown in most situations will require a trip back to the operating room to remove the previous implants. However, before returning to the OR develop a new plan. This involves sitting down looking at the initial pre-operative radiographs followed by your immediate post-operative radiographs. The goal is to determine why there was implant breakdown then identify what needs to be done to improve the construct that will result in success. Once ready head back into the OR to place new implants.

3) Poor bone healing

Delayed union is healing of a bone that takes longer than expected to heal. The normal healing time frame for a bone is 8-12 weeks versus a nonunion, which is where the bone fails to heal regardless of healing time or if a delayed union is not addressed. A malunion is characterized by a healed fracture in an improper alignment. This may be noted as shortening of a limb, malalignment of the joint surfaces, rotational abnormalities, or varus and valgus deformities. Typically surgical correction is needed with mal-unions.

Initially there should be bone activity noted by 3-4 weeks post-operatively. If there is no bone activity at this early time period then intervention is need. Additionally, we should see continued radiographic activity every 4-6 weeks. If there is no activity noted over the course of 8 weeks or healing is not complete by 16 weeks then intervention is likely necessary. The goals of delayed unions are to figure a way to improve stability. Perhaps this involves adding a splint or returning to the OR to improve the rigidity. If there are poor healing properties to the bone this should be identified. There can be patient specific factors involved; however, this is unlikely in most cases. Excessive tissue manipulation during surgery is more likely the cause of poor healing properties. In general with bone the afferent blood supply is supplied through the nutrient artery, where the blood flow is centrifugal in that it progresses from the medullary cavity to the periosteum. Therefore, blood flow is from the nutrient artery to the metaphyseal arteries, and then the periosteal arteries. Once the bone is disrupted the medullary circulation is disrupted; therefore, we get an enhancement of existing
normal blood supply. Temporarily, there is a transient extraosseous supply from the soft tissues. Therefore, it is very important to preserve this blood supply and be kind to the tissues during surgery.

Nonunions are further broken down into viable and nonviable. Viable nonunions can be classified as hypertrophic where there is considerable callus formation but no bone healing, this is sometimes referred to as an “elephants foot”, or it can be moderately hypertrophic where there is a lesser degree of callus known as a “horses foot”. Both types of hypertrophic viable nonunions are typically caused from motion in the fracture site; therefore, more rigid fixation is needed. An oligotrophic viable nonunion is hard to distinguish from a nonviable nonunion due the fact there is no radiographic evidence of healing. Its cause is due to lack of cellular activity which are typically due to loose implants in the area of the fracture such as loose cerclage wires. Nonviable nonunions can be classified as dystrophic where there is no viable bone on either side of the fracture, necrotic where there is an infected section of bone such as a sequestrum, defect where there is a gap at the fracture site that is too large to heal, or atrophic where there is removal of dead bone by the host with no healing and often times resorption of the bone.

To address non-unions all the necrotic and fibrous tissue needs to be removed. There should be healthy bone ends with bleeding present. The area is then packed with cancellous autogenous bone graft and lastly rigid fixation is needed to promote bony union. Traditionally, viable non-unions are easier to deal with because motion is the issue so that needs to be corrected. Nonviable non-unions are tough because trying to regenerate large amounts of new bone is challenging. In situations of nonviable non-unions amputation may be recommended.

In summary, remember complications occur to everyone. Use every effort possible to minimize complications so they are an infrequent occurrence. Communication is key with owners so that everyone participates in the success of surgery. Remember to use the “tools in your tool belt” in assessing outcomes to identify complications early. They are easier to treat that way. Lastly, learn to identify specific complications especially the more common ones such as infection, implant breakdown, and poor bone healing.

References
My Dog Doesn’t Have Obamacare and Can’t Wait for a New Plan!  
What to Do About the Torn Cruciate

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My dog doesn't have Obamacare and can’t wait on the government to make up its mind; what I can do about the torn cruciate?

Cranial cruciate ligament (CCL) rupture is one of the most common orthopedic conditions encountered in the dog. In fact, over 1 billion US dollars are spent every year in dealing with the canine stifle. When dealing with hind limb lameness many dogs we see have some degree of hip dysplasia or degenerative changes in the hip; however, an acute lameness is typically not due to a hip problem. In fact 32% of dogs referred for hip problems actually have evidence of cruciate disease. About 33-50% of dogs will present with bilateral disease even if they have a unilateral lameness. Severe bilateral cruciate disease can often mimic other conditions such as severe hip dysplasia or neurologic disease. Therefore, a general rule of thumb is a hind limb lameness in a dog is cruciate disease until proven otherwise.

Personally for us, statements that we do not like are:

• All dogs that rupture their CCL must have surgery
• All dogs with CCL ruptures have joint effusion
• All surgical procedures (extra-capsular repair, TPLO, TTA, XYZ) have the same outcome
• A dog can’t return to pre-injury status following a CCL rupture
• Dogs don’t benefit from rehabilitation therapy either with a conservative approach or following surgery

Diagnosis

The diagnosis is typically straightforward and is based off the history, signalment, clinical signs, physical exam, and orthopedic exam. The history may include an acute or chronic hind limb lameness that may be mild to non-weight bearing. Interestingly, owners may report that the lameness has improved from initial injury. This usually corresponds to the timeframe from when the initial inflammatory response is ending. Regarding the signalment any age or breed can be affected. Typically we tend to see medium to large breed dogs that are around 3-8 years of age. The orthopedic exam is mainstay to diagnosing a CCL rupture. Findings may include a positive sit test where the dog will tend to sit with the affected leg projecting out to the side. Pain on hyperextension is usually the forgotten test but is very reliable. Most affected dogs will exhibit some degree of pain. Crepitus may be noted during ROM, and with chronic tears medial buttress formation may be noted. This is the peri-articular fibrosis that occurs. The classic findings for a CCL rupture are joint effusion, the cranial drawer test and the tibial compression test. A simple way to think about it, is that in an adult dog joint effusion will only be caused by a CCL rupture, septic arthritis, tick-borne disease, or immune-mediated arthritis. A medial patella luxation (MPL) will not cause the same degree of joint effusion, so if you have a patient will underlying MPL that develops joint effusion be thinking about a CCL rupture.

The cranial drawer test is testing for laxity in the CCL, but this is more of a passive test and does not mimic weight bearing. To perform the test one hand is placed on the distal femur with the thumb behind the lateral condyle. The other hand is placed on the proximal tibia with the thumb behind the fabella. The goal is to move the proximal tibia cranially in relation to the femur. Always check drawer in flexion and extension. When checking for partial tears the CCL has two bands, the cranio medial which remains taut in both flexion and extension and the caudolateral, which is taut in extension but lax in flexion. For example if the cranio medial band is torn and the caudolateral band is intact cranial drawer is only present in flexion because in extension the caudolateral band is taut. If the caudolateral band is torn and the cranio medial band is intact no cranial drawer is present because the cranio medial band is taut in both flexion and extension. Cranial tibial thrust is a test meant to mimic active weight bearing. The goal is to hold the stifle at a standing angle (approximately 135 degrees) and while holding the stifle still flex the hock. If the CCL is ruptured there should be a cranial displacement of the tibia. As with cranial drawer, tibial thrust should be checked in both flexion and extension.

Radiographic evaluation will help to see evidence of joint effusion with cranial displacement of the intrapatellar fat pad. With chronic CCL ruptures you may see evidence of OA and if you are lucky the stifle is sitting in drawer on the radiographs. Some people have proposed a stable stifle with joint effusion and a hind limb lameness may be evidence of a partial tear.

Treatment

When deciding on a treatment plan there is no one treatment fits all, but there are many, many, many options available. The reason there are so many options is because not one procedure or medical management technique is 100% perfect. I think one reason for this
is because what is considered our final outcome, a stable stifle, a patient that returns to activity pain free, elimination of OA, owner satisfaction, etc.? We will never be content on cruciate disease until we figure out the goals we want to achieve for an outcome.

When I approach a dog with cruciate disease I'm going to have the same conversation with each owner; however, depending on each case I may swing my conversation in one particular direction. Factors I consider when deciding on conservative vs. surgical treatment and which procedure are the patient, owner, and veterinarian factors. I look at the breed, the size of the animal, the age, the activity level, and what is that particular animals job. Are they a pet, an athlete, or a service dog? Regarding the owner I talk to them about their perceived outcome, their ability and willingness to follow directions post operatively, as well as finances. And then I look at my abilities such as what equipment I have available, what procedures am I comfortable doing, and what good and bad outcomes have I had with certain procedures.

When I first tell owners that their dog has a torn cruciate I try to cover 3 main options. Option 1 is we do nothing. By do nothing I mean we cage confine for 6 weeks with medical management (analgesia and NSAIDS) and (hopefully) formal rehabilitation therapy. The most important aspect here is confinement. These owners have to be aware the goal of conservative management is to allow peri-articular fibrosis to occur. This can’t occur with the dog remaining active. To break it down to them I tell the owners the dog must be kept in an area where he/she can stand up, lie down, and turn around. The dog eats, drinks, and sleeps in the crate. It only goes outside to urinate and defecate on a leash then back into the crate. I also throw the disclaimer in that in my opinion OA is worse with a rapid progression as long as the stifle is unstable and usually if this is a larger dog they wont return to full function. I also really push the fact that the dog will appear to be do “okay”; however, they have a very high chance of developing a meniscal tear. I tend to tell owners its not “if” but more of a matter of “when” they tear their meniscus. Personally, I am not a fan of this approach!

Option 2 is a conservative approach with exercise restriction, formal rehabilitation therapy, and a custom made stifle orthotic. While this approach parallels that of option 1, we can in theory attempt to help stabilize the stifle with a brace. In human medicine, knee braces are commonly used for multiple purposes. The human knee has been shown to enhance proprioception/joint position sense, permit the injured limb to relax, reduce fatigue in injured limb, provides some mechanical protection against impact, and slow movement down to allow muscles time to react and control motion. Categories of knee braces in human medicine include the following: prophylactic (prevent or reduce severity of knee injuries in contact sports), functional (provide stability for unstable knee, rehabilitative (allow protected and controlled motion during the rehabilitation of injured knees), and patellofemoral (improve patellar tracking and relieve anterior pain). Only functional knee braces are utilized in veterinary medicine.

In theory the brace should help limit tibial subluxation. At the authors institution (unpublished data) we did find improved objective gait analysis when a custom stifle brace was worn versus when not worn; however, the gait analysis was not improved equal to that of surgery. This data reveals that a brace is not considered equal to or meant to replace surgery; furthermore, it must be worn for the duration of the pet’s life.

Option 3 is the grey zone. It may be the morbidly obese dog that needs to lose weight before surgery. It may be the family that is saving up for the TPLO, but wants to do something prior to surgery. Or it can be any other number of reasons. The focus in this area is education. Client education is critical with regards to many aspects of their pet. This includes obesity reduction (through diet and exercise), pain mitigation (through NSAIDS and other oral medications), chondroprotection (through oral supplementation of Glucosamine, Chondroitin, Omega-3 FA, etc.. Seeing the big picture with these pets is needed to achieve a favorable outcome. Without it, you eat a fat, immobile dog in chronic pain.

Obesity: Checking thyroid function on all overweight dogs is recommended. If it is normal, then using a prescription diet tailored for obesity and arthritis is recommended. (MB uses Hills Metabolic and Mobility in his practice). Once initial inflammation is reduced (usually with NSAID, 1-2 weeks after injury) a rehab program of underwater treadmill walking is initiated. The goal here is to use the buoyancy of the water to reduce stress on the unstable stifle. This will allow for reduction of muscle atrophy and caloric burn without further injuring the patient. A protocol of 10-30 minute walks 2-3x a week (progressively building) is recommended. If possible, a photobiomodulation (therapy laser) treatment protocol is also initiated at these visits, with the goal being to reduce inflammation and promote tissue healing.

An at home program can include short leash walks, but a focus here on therapeutic exercises is used instead. Once initial inflammation is reduced, working on appropriate sit to stand exercises with the stifle in appropriate position is attempted. 10 reps per set, 10-2 sets per day. Initially we aim for 5/10 reps to be square, and then each week increasing our goal by one. The affected stifle should be against a wall, so as to minimize outward rotation. The goal here is fairly quick succession throughout the set. As soon as the patient ischium touches the ground the dog should be asked to stand again and repeat the exercise. Stretching and range of motion can begin at 1-2 weeks post injury, and then continue daily through week 8. The goal here is to maintain range of motion of the joint and minimize contraction.

The walks should be at a slow pace, on leash and on flat surfaces with good traction, Hill work and turning quick corners is not added until weeks 4-6.

Around weeks 4-6 as the fibrosis is being achieved, core strengthening with balance disks and wobble boards can be done in a slow safe and professionally administered manner.
My issues with stifle orthotics are as follows:

1. **Tolerability:** I can't ask the patient if he/she will tolerate the brace, I have had some dogs that don't mind it at all, others take time, and some just freeze or try to chew it. The other issue is given the different shapes and sizes of dog stifles the brace MUST be custom made. This means a mold must be made and sent to the orthotist and then sent back about 2 weeks later. It's a horrible feeling to have an owner pay the expense for a brace and then the dog won't tolerate it.

2. **Arthritic progress:** What I can tell an owner is that with surgery we can slow down and minimize arthritic progression. Without surgery we will have continued accelerated and worsening progression OA. Along that scale is a brace; I just don't know if the scale is closer to that of surgery or that of no-surgery?

3. **Meniscal damage:** What I can tell an owner is that with surgery we can minimize the chances of a meniscal injury. Without surgery there is a high incidence of meniscal injury. The problem is again along that scale I don't know where a brace will fall. Will it help protect the meniscus the same as surgery, or will it not make a difference such as doing nothing? This does bring up a good point about meniscal damage. A “meniscal click” will only get you about 30-40% correct at identifying a meniscal injury. If you add in a positive McMurray test and pain on hyperflexion that may improve to about 50%. Personally, I feel as if a dog has a meniscal tear they will not benefit from a brace because it will do nothing to help with the pain and discomfort. The problem is if at best you can diagnose a meniscal injury in 50% of patients then how does one approach determining if there is meniscal injury? A MRI could be considered but is costly and requires general anesthesia, arthroscopy could be considered but personally would be below the standard of care to go to surgery to identify a meniscal injury but not treat the CCL rupture. Therefore, if I have owners that want their dog in a brace then they must undergo a stifle ultrasound. If there is evidence of meniscal damage then that dog will not be a good candidate for a brace, if they don’t appear to have meniscal damage then we can give it a shot knowing that an ultrasound is not 100%.

**Cruciate disease** is a complex problem that does not have a clear cut answer. Ultimately, surgical intervention may be needed in any case that is initially managed without it. Proper education of the client is critical to establishing favorable outcomes.

**References**

Hip dysplasia (HD) was originally described in 1935 by Gerry Schnelle and has become one of the most common orthopedic conditions that leads to joint inflammation and secondary osteoarthritis. Unfortunately, even after all of this time the exact etiology is unknown but considered to multi-factorial. One such factor involved in the expression of HD is genetics. It is not a simple Mendelian pattern but rather a complex inheritance. This means there are multiple genes that are combined with environmental influences that lead to the clinical expression of HD. Joint laxity is considered the initiating cause of HD which in turn leads to hip subluxation and poor congruence between the femoral head and acetabulum. Multiple causes of hip laxity have been described such as abnormal hip development, biomechanics, genetic influences, increased joint fluid, pelvic muscle mass, nutrition, weight/growth, and hormonal and environmental factors. It’s probably safe to assume that HD and the subsequent arthritis are the clinical manifestation of all of these.

Nutrition is thought to be a large contributor to joint laxity and thus HD; however, no dietary deficiencies cause HD. Dietary excesses on the other hand can contribute to the development of HD. For example, increased calcium and vitamin D lead to alterations in endochondrial ossification, and delayed bone remodeling. Diets high in excessive vitamin C can lead to hypercalcemia and diets with a high anion gap lead to increased synovial fluid production, which in and of itself has been shown to be a risk factor for hip laxity. Feeding diets to promote rapid growth have been shown to have a higher incidence of HD and also cause early fusion of the acetabular growth plates.

Increased body weight is not a cause of HD, but it certainly has very important clinical consequences in susceptible dogs. Therefore, weight reduction is an effective preventative strategy. In the lifespan study of 49 Labradors it was reported that heavier dogs (dogs allowed to eat ad lib) developed radiographic OA on an average of 6 years earlier than the dogs in the restricted fed group. Furthermore, heavier dogs required long-term treatment for OA on average 3 years earlier than their restricted fed littermates.2

The diagnosis of HD is made from the signalment, clinical signs, physical exam findings, and radiographs. Affected dogs are typically large breed fast growing dogs such as German Shepherds, Rottweiler’s, Labradors, or Golden Retrievers. The age of presentation is typically biphasic and contributes to the type of treatment that may be recommended. Juvenile dogs will tend to present between 5-12 months of age with an acute onset of unilateral or bilateral hind limb lameness. These clinical signs are thought to be due to joint laxity. Histologically tearing of the joint capsule along with microfracture of the dorsal acetabular rim is seen. As dogs become older the long-standing joint laxity causes periarticular fibrosis, which may decrease or lessen the clinical signs. This is why some dogs will tend to have improvement in clinical signs until later in maturity when they present for clinical signs that are consistent with OA.

The severity of clinical signs depends on the stage/severity of the disease. Lameness can be intermittent, progressive, and range from mild to severe. In young patients with severe laxity a “popping” noise may be heard during ambulation. Both young and older patients may exhibit exercise intolerance and difficulty rising from pain and discomfort. Disuse muscle atrophy is a common finding and the gait may be characterized as either “swaying” or hopping. It is very important to remember that a non-weight bearing lameness is rare and thus other problems should be considered such as a cranial cruciate ligament rupture. Orthopedically pain in the hips along with crepitus may be noted. Many of these patients have decreased range of motion in extension and weight shifting to the forelimb. Evidence of joint laxity is determined through the Barlow, Ortolani, and Barden’s test. The Ortolani is performed with the patient in either lateral or dorsal recumbency and sedation is required in most cases. The first part of the ortolani is the Barlow test where a force is directed through the femur through the dorsum to subluxate the hip. The Barlow test is considered a provocative test in that it creates subluxation in a lax hip. The second part of the Ortolani test is the true ortolani maneuver where the limb is abducted and a click or clunk can be heard as reduction of the hip occurs. The clunk is considered a positive ortolani and indicative of coxofemoral laxity. Some surgeons will use the angles measured during an Ortolani test as indications for a triple or double pelvic osteotomy. The Barden’s test is performed with the dog in lateral recumbency; a direct lateral force is applied to the femur without abducting the limb. In the awake dog pressure on the medial thigh can cause discomfort and this should not be mistaken for hip pain. Any movement of the greater trochanter more than ¼ of an inch suggests laxity. Unfortunately, Ortolani and Barden’s only suggest laxity and do not predict later development of clinical signs of OA.

Radiographs are mainstay for the diagnosis of HD along with the characterization of the disease and any presence of OA. There are several ways to evaluate canine hips, which vary from using the hip extended view as what is done with OFA, or developing a distraction index as what is done with PennHip. OFA style radiographs are generally used in daily practice, this involves that the
pelvic limbs are fully extended and parallel, the pelvis is symmetrical and the pelvic limbs are internally rotated. Sedation and/or general anesthesia is usually required. Mal-positioned radiographs can lead to false assumptions. The two most notable and early signs with hip OA are the circumferential femoral head osteophyte (CFHO) and the caudo-lateral curvilinear osteophyte (CCO). The CFHO is a white line at the articular margin of the femoral head that may or may not extend completely around the femoral head. It is graded from I to III. The CCO is also sometimes known as a Morgan's line, it is a well-defined linear density on the femoral neck between the greater trochanter and the capital physis in dogs greater than 18 months of age. It is different from a puppy line in that a puppy line is an indistinct radiodense line on the femoral neck in dogs less than 18 months of age, its in a similar location to the CCO but it is more subtle, more diffuse and shorter than the CCO. A puppy line is considered self-limiting and is not clinically significant.

One big debate is between the use of OFA and PennHip for HD screening. OFA is a subjective scoring system based on the hip extended view. The problem is the hip extended view is an unnatural position for dogs and can mask subluxation because the view actually forces the femoral head into the acetabulum. It does identify OA and moderate laxity but is not a sensitive method to detect early or mild laxity. PennHip uses stress radiography to detect joint laxity and it can be predictive for the development of OA. It is a measure of hip laxity, not a certification process. A study in 2010 using the OFA database described a 1.5% increase in OFA excellent films, a 3.3% increase in OFA good films, and a 2.1% decrease in OFA fair films. To complicate matters it was found that in dogs with OFA excellent films 52% had DI >0.3 putting them into the OA susceptible range, 82% of dogs with OFA good had DI greater than 0.3, and 94% of dogs with OFA fair had a DI greater than 0.3. In other words the progress of eliminating HD is moving very slow. In fact at the current progress it will take about 44 years to move Labs from a hip score of 10 where is it currently to a hip score of 5, which is equal to an OFA excellent grade.

Physical rehabilitation has a multimodal approach within itself for managing HD. Physical modalities, manual therapies and therapeutic exercises can all be used to achieve relief from HD. Goals of rehab for the patient include: maintaining or improving muscle mass, building muscle support around the lax or arthritic joint (and all joints), reducing pain and weight loss (via exercise, when indicated).

Physical modalities can include theraphy (the use of cold and warm packs). The benefits of cryotherapy are established (pain relieving, vasoconstriction, etc.) and warm compresses can be used to relieve pain, cause vasodilation and also help to warm up stiff, tight tissues to begin other exercises.

Therapy LASERs (Light Amplification by Stimulated Emission of Radiation) have become very popular in recent years. There are different wavelengths, amplitudes, treatment times and other factors that must be considered. This process has also been called photobiomodulation. It has been proposed to activate cytokines and other tissue factors, decrease pain and inflammation and increased wound healing. Always use goggles for both the humans and patient to avoid damage to the eyes. It cannot be used over pregnancy or cancer.

Manual therapies are skilled hand movement techniques intended to: improve issue extensibility, increase range of motion (ROM), induce relaxation, mobilize or manipulate soft tissues and joints, modulate pain and reduce swelling and inflammation. These can include massage and joint mobilizations. The basic principles of joint mobilizations work from physiologic motions and accessory motions. Physiologic motions are normal active motion that is available at a joint. Examples: flexion, extension, abduction, internal rotation, etc. Accessory Motions are movements that cannot be performed actively. Examples: distraction, compression, glides, spins and rolls. There are 4 grades of mobilization, and the manipulation (used in chiropractic) is a 5th grade. Grades 1-4 are passive movements, with 1 and 2 not reaching initial resistance of the joint end feel. Grade 3 moves through the initial resistance to the end feel, but does not exceed it. Grade 4 mobilizations are compact with in the first and second resistance points. Grade 5 (manipulations) exceed the normal end feel of a joint.

Therapeutic exercises are the “meat and potatoes” of rehabilitation. These are designed to work a patient from a recumbent position back to normal (or as close as possible) activity following injury or insult. Exercises in this group can include cavaletti rails, working on balance boards, disks or other core strengthening equipment. Once walking on a flat non-slip surface is achieved, adding varying degrees of difficulty (up hills, through different traction, etc.) can be included. Sit to stand exercises and core strengthening with dancing exercises are also helpful. The key is to keep the patient moving and building.

Land treadmills – Can be useful devices for providing exercise. Small and medium dogs will work well on a human machine, but larger dogs will benefit from a canine treadmill. This is due to stride length and length of the belt. Having the dog walk on an incline will help build up the pelvic limbs.

Underwater treadmills – Can be used as both a diagnostic and therapeutic tool. The buoyancy of water will allow severely affected animals to utilize their limbs. There are also studies showing the benefit of underwater treadmill therapy for reducing obesity in dogs. With water at the level of the hock, there is a 9% reduction in perceived body weight, a 15% reduction with water at the stifle, and 62% reduction when at the greater trochanter. The non-slick, safe, contained surface an underwater treadmill provides is superior to walking in ponds, lakes or swimming in pools, in the author’s opinion.

Treatment for HD can be broken into prevention and/or laxity improvement utilizing the juvenile pubic symphysiodesis (JPS) or triple/double pelvic osteotomy (DPO or TPO). More definitive treatment can be accomplished with medical management, a femoral
head and neck ostectomy (FHNO or FHO) or a total hip replacement (THA). In immature dogs that are still growing with no evidence of OA then medical therapy can be attempted. This includes promoting weight loss, daily activity, and formal rehabilitation therapy to improve muscle mass, range of motion, and comfort. Many of these patients benefit from NSAIDS, chondroproctants, and omega-3 fatty acids. For those that are severely clinically affected or have failed medical therapy then either a JPS or DPO/TPO, FHNO, or THA can be considered. In mature dogs medical management is geared towards OA management. Older dogs that become refractory to medical management would then become candidates for either a FHNO, or THA. Regardless early detection is key, in susceptible breeds hip palpation should begin by 12 weeks of age. If they have a positive Ortolani or have a high DI after 16 weeks of age then JPS should be considered in at risk breeds. A JPS is a minimally invasive way to pre-maturely cause fusion of the pubic symphysis. This causes ventro-lateral rotation of the acetabulum with growth of the animal (resulting in ventroversion and improved femoral head coverage). To procedure is completed with a small incision to the pubic symphysis, electrocautery is then used every 2-3 mm along the symphysis at 40 watts for 12-30 seconds. Best results are achieved in patients before 16 weeks of age (20 weeks in giant breeds) resulting in about 10-15 degrees of ventroversion if done at 16 weeks. No real benefit is gained if completed in animals greater than 22-24 weeks of age. The resultant hip changes are similar to what is seen with a DPO/TPO; however, it is easier and faster with fewer complications and no implants are needed.

A FHNO has typically been reserved for smaller dogs and cats; however, larger dogs can also be candidates. It involves removal of the entire femoral head and neck and relies on the formation of a pseudoarthrosis. Even though owner satisfaction is high it is a salvage procedure with 62-65% return to normal function from a gait analysis standpoint. Probably the biggest complication with a FHNO is leaving femoral neck behind, other complications include shortening of the limb, patellar luxation, muscle atrophy, limited hip extension, recurrent lameness and chronic pain. In my hospital patients are required to undergo formal rehabilitation therapy beginning 3-5 days after surgery and continuing for 6-12 weeks.

In summary, HD has a complex pathophysiology with the predominant feature being joint laxity. There are many factors that contribute to joint laxity. Clinical signs will vary depending on the stage of disease, but remember an older dog that is acutely non-weight bearing will often times have a cruciate rupture with underlying HD. A thorough physical examination with good quality radiographs is needed. Early detection is key so that way a JPS can be performed.

References
Osteoarthritis (OA) is a chronic, progressive disease that affects both dogs and cats. It has been noted that up to 20% of adult dogs and 60% of adult cats have radiographic evidence of OA.\(^1\)\(^2\) Owners, themselves, are becoming increasingly aware that bone and joint problems are and issue with their pet. Much of this increased awareness has come through the use of the Internet and social media. The overall outcome of osteoarthritis is centered on destruction of the articular cartilage and breakdown of the joint. Because of this OA must be thought of as a global disease process rather than an isolated disease entity. There is considerable cross talk among the tissues that make up a joint. For this reason the joint must be thought of as an organ and the final pathway of OA is organ failure of the joint.

OA primarily affects diarthrodial joints. A diarthrodial joint is composed of the joint capsule, synovial lining, articular cartilage, and the surrounding muscles, ligaments, tendons, and bone. The joint capsule is composed of two layers: the outer fibrous layer and the inner subsynovial layer. Both layers have a rich blood and nerve supply. One explanation of pain associated with OA is distention of the joint capsule due to joint effusion. The synovial lining covers every structure in the joint except for the cartilage/menisci. It provides a low friction lining and is responsible for the production of synovial fluid. Two major cell populations are present in the synovial lining: type A synoviocytes and type B synoviocytes. Type A synoviocytes are macrophage-like cells that are responsible for phagocytosis. The type B synoviocytes have a more fibroblastic-like appearance and are responsible for producing hyaluronan acid (HA) and other enzymes.

The physiology of cartilage is important because damage to chondrocytes will not only lead to death of that particular chondrocyte but also an inflammatory response that creates problems with neighboring chondrocytes. Thus a downward, progressive spiral occurs which leads to destruction of the “work-horse” (chondrocytes) and loss of extracellular matrix production. The loss of ECM production leads to the loss of cartilage’s ability to soften and transfer loads to the underlying subchondral bone.

The pathophysiology of OA is described as a non-infectious disorder of diarthrodial joints. It is categorized by deterioration of articular cartilage, bone formation at synovial margins (osteophytes), peri-articular fibrosis, and a localized inflammatory response. For OA to develop there has to be some insult to the articular cartilage such as hip dysplasia, a cranial cruciate ligament tear, elbow dysplasia, or an articular fracture. Once the chondrocyte is damaged the inflammatory cascade begins and is followed by the release of multiple cytokines. The two main cytokines involved with OA are interleukin 1 beta (IL-1\(\beta\)) and tumor necrosis factor alpha (TNF-\(\alpha\)). IL-1\(\beta\) is responsible for the breakdown of the matrix, while TNF-\(\alpha\) drives the inflammatory response. Furthermore, prostaglandins are released, particular prostaglandin E2 (PGE2), which increases the release of metalloproteinases (MMPs). MMPs are responsible for the continued breakdown of the ECM.

In summary of OA inflammation: Osteoarthritis is a chronic progressively destructive disease that involves the entire joint. Inflammation is a key component of both joint destruction and pain. Acute pain resolves after the initial injury heals. Chronic pain involves structural changes of the dorsal horn, is more intense than acute pain and more difficult to control. Treatment considerations for osteoarthritis should address inflammation as well as pain.

Diagnostic approaches to osteoarthritis: Owners will typically complain about their pets have a reluctance to exercise, stiffness, lameness, inability to jump, or even some behavioral changes. Remember that cats are not small dogs, and they can have fewer signs. The biggest complaint from owners with cats suffering from OA is a reduction in activity, reluctance to jump, an unkempt appearance, and aggression. Orthopedically, dogs may show disuse muscle atrophy (ensure to rule out any neurogenic atrophy), a reduced range of motion, pain or discomfort on range of motion, crepitus, and joint effusion. Cats can be tricky to examine so allowing them performance tests is encouraged to see how the cat moves and interacts with its environment. One true test is to place the cat on exam table with its carrier below. Most cats will easily jump from the exam table to their carrier. Any reluctance to want to do so raises concern about possible joint pain.

Radiographs are key to aiding in the diagnosis of OA. However, just as with any diagnostic modality there are limitations. Radiographs only provide bony information, they are taken in a non-weight bearing position, and osteophytes are useful to diagnose OA but they are not pathognomonic for OA. Furthermore, the value of osteophytosis for staging OA is controversial and does not correlate with OA progression. Probably the biggest issue with radiographs is that they do not correlate with clinical signs. The radiographic key features of OA are: osteophytosis, enthesophytosis, effusion, soft tissue swelling, subchondral sclerosis, intra-articular mineralization (especially in cats), and subchondral cyst (rarely seen).
Other additional diagnostic modalities include CT, MRI, and arthroscopy. Arthroscopy is probably the most valuable means to objectively evaluate the cartilage. However, it is a surgical procedure and can be costly to perform. It does allow the evaluation of the cartilage, which can then be classified by the Modified Outerbridge score. One looming question is if you don’t perform arthroscopy and radiographs are helpful to diagnose but don’t help stage for monitoring for progression of OA is there some type of subjective based assessment? The answer is yes, the Canine Orthopedic Index (COI) was developed and validated in 2014 to provide reliable assessment of dogs with OA in terms of staging as well as response to treatment. It can be downloaded at www.canineorthopedicindex.com.

A multimodal approach to OA management is needed. Non-Steroidal Anti Inflammatory Drugs (NSAIDS) represent the cornerstone of therapy, but other modalities include: nutrition, chondroprotectants, additional analgesics, physical rehabilitation, weight control, exercise, an EPA rich diet and many new and emerging options. Let’s look through these individually.

Obesity is a growing issue in veterinary medicine. The effects of obesity on OA are twofold. Biomechanical stress contributes to clinical signs and progression of disease. Adipokines secreted by white fat cells contribute to the progressive inflammation of osteoarthritis. Leptin levels are elevated in obese dogs. In humans with osteoarthritis, increase leptin levels correlate with elevated MMPs and NO in synovial fluid. Adiponectin is anti-inflammatory, but levels are low in obese dogs. In human patients with knee osteoarthritis there is a significant correlation with adiponectin: leptin ratios.

Humans with increased body mass index (BMI) experience OA in non-weight bearing joints, which resolves with weight loss. Decrease in BMI, is associated with symptomatic relief from knee OA in man. Systematic review of canine studies found that preventing obesity decreases incidence of OA and weight loss reduces signs of OA. Additionally, diets rich in Omega-3 fatty acids have shown to be beneficial for both dogs and cats with OA. Additional nutritional supplements such as glucosamine, chondroitin, methylsulfonylmethane (MSM) and others have been shown potentially beneficial for our patients.

Physical rehabilitation has a multimodal approach within itself for managing OA. Physical modalities, manual therapies and therapeutic exercises can all be used to achieve relief from OA. Goals of rehab for the DJD patient include: maintaining or improving muscle mass, building muscle support around the arthritic joint (and all joints), reducing pain and weight loss (via exercise, when indicated).

Physical modalities can include thermotherapy (the use of cold and warm packs). The benefits of cryotherapy are established (pain relieving, vasoconstriction, etc.) and warm compresses can be used to relieve pain, cause vasodilation and also help to warm up stiff, tight tissues to begin other exercises.

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Disease modifying agents for OA are next to be discussed. Polysulfated glycosaminoglycan is FDA approved, disease modifying osteoarthritis drugs; for dogs and horses; water-based, for intramuscular injection Dosage: 2 mg/lb body weight, IM, twice weekly for up to 4 weeks (maximum of 8 injections). MOA: specific is not known; in vitro studies show; they inhibit serine proteinases; PGE2 synthesis; metalloproteases, hyaluronidases and others. Stimulate synthesis of protein, collagen, proteoglycans, and hyaluronic acid. There are studies showing it reaches feline cartilage via subcutaneous injection. This is extra label usage for this medication. Also, maintenance injections have bene anecdotally reported for both dogs and cats. Clinical studies on PSGAGs showed both good efficacy and safety. Treated dogs had statistically significant improvement in range of motion and total orthopedic score over placebo treated control dogs. 2.1% of dog had adverse reactions including: transient pain at the injection site (1 incident), transient diarrhea (1 incident each in 2 dogs) and abnormal bleeding (1 incident). These effects were mild, self-limiting; did not require interruption of therapy. Do not use in dogs showing hypersensitivity to PSGAG, or in dogs with known or suspected bleeding disorders. Use with caution in dogs with renal or hepatic impairment.

Adjunct analgesics for OA are numerous. They are used in addition to or replacement for NSAIDS. Research is scant on some of them. Amantadine – only drug studied to treat canine osteoarthritis. In dogs with osteoarthritis pain refractory to an NSAID, addition of amantadine improved physical activity. Amantadine might be a useful adjunct therapy for the clinical management of canine osteoarthritis pain. It can be dosed at 3-5mg/kg SID. Gabapentin – Calcium channel modulator – 5-10mg/kg SID-TID. Amitriptyline 0.5-1.0mg/kg SID-BID – cats and dogs. Local anesthetics – Lidocaine, bupivacaine, mepivacaine. Acetaminophen can be used in dogs.
but not cats. Opioids – morphine, meperidine, methadone, oxymorphone, hydromorphone, fentanyl, fentanyl patches, butorphanol, pentazocine, nalbuphine, buprenorphine, codeine and tramadol.  

Tramadol’s metabolism and elimination is rapid and variable among dogs. When administered orally or intravenously to the dog, metabolism of tramadol and all metabolites is rapid. There is much variability between dogs, possibly breeds. Pain control did not necessarily correlate with plasma levels of the active metabolite (O-desmethyltramadol). Tramadol effects on α-adrenergic or serotonin receptors may contribute to analgesic effects in the dog. Regardless of mechanism of action, studies suggest oral dose should be 5 mg/kg q 6 hours or 2.5 mg/kg q 4 hours. In the author’s opinion this is a very challenging drug to utilize effectively in practice due to these variables.  

Galliprant is a first-in-class non-cyclooxygenase (COX) inhibiting, non-steroidal anti-inflammatory drug (NSAID) in the piprant class. Piprants are a newly recognized drug class, established and defined by the World Health Organization in 2013 as prostaglandin receptor antagonists (PRA). Unique mechanism of action by antagonizing the prostaglandin E2 (PGE2) EP4 receptor. PGE2 its physiologic effects through binding of four different receptors, EP1, EP2, EP3 and EP4. The EP4 receptor has been identified as the primary receptor responsible for mediating pain and inflammation associated with osteoarthritis. Galliprant selectively blocks the EP4 receptor, thus blocking PGE2 elicited pain.  

Potential intra-articular therapies include regenerative medicine (platelet rich plasma with or without stem cell treatment), hyaluronic acid, or steroids. Discussion of regenerative medicine is beyond the scope of this proceeding. HA is a viscosupplementation that restores the physiochemical properties to the joint. From a molecular standpoint it stimulates production of ECM as well as continued production of HA from resident synoviocytes. It will also inhibit inflammatory mediators. It is important to use a product that closely mimics a dog’s HA such as Evervise from Everost (sold through Patterson). Evervise is about 2 million Daltons in size and is made from a fermentation process rather than rooster combs. Until further research is completed it is not recommended to combine an HA injection with any other drug as this may decrease the molecular weight of the HA or could lessen its efficacy. What has been shown is that approximately 80% of dogs respond well to HA, 10% respond fair, and 10% don’t respond. The duration of response is about 4-6 months of relief. When compared to regenerative medicine a response of about 9 months is expected following a platelet rich plasma injection and about 12 months or longer following a platelet rich plasma and stem cell injection.  

In summary, OA is a chronic progressive disease and the goal of management needs to be to slow and minimize the progression. Owners need to be well educated to know that it will progress and there will be flare-ups. Treatment needs to be multimodal and patient centered.  

References  
2015 AAHA /AAFP Pain Management Guidelines for Dogs and Cats.  
Adequan prescribing information. NADA 141038, Novartis animal Health, US, INC.
1. Acute Pain Myths
   a. Animals do not feel the degree of pain that humans do
   b. It keeps them quiet post-op; this is a good thing
   c. OHE, neuters, minor procedures don’t need medication at home
   d. Analgesics cause adverse events
   e. Pet owners won’t pay for pain control

2. Pain Scales in general—guide for use
   a. Intervals of assessment should be determined by anticipated pain from the procedure as well as the health of the animal and expected onset and duration of the drugs used.
      i. Hourly for the first 4 hours post-surgery
   b. Start after recovery from anesthesia: usually as they are trying to go into sternal recumbency
   c. Allow animals to sleep
      i. Be aware that cat-napping is not real sleep
   d. Continuous and distant observations are best, utilizing specific pain scale interactive observations periodically

3. Acute pain and validated scales
   a. Pain scales were developed for the assessment of pain from surgery
      i. Can probably be used for assessment of acute traumatic pain as well.
   b. Two pain scales discussed in this lecture
      i. Scotland: Newmetrica/Glasgow Feline Composite Pain Scale
      ii. Brazil: UNESO-Botucatu-MCPS
      iii. There are other pain scales out there.
   c. Botucatu
      i. Multidimensional Composite Pain Scale
      ii. Looks at a combination of physiological/behavioral/body position factors
      iii. Several pages long. Difficult to use in clinical setting.
      iv. Well-validated for postoperative pain.
   d. Newmetrica Pain Scale
      i. Can be downloaded for free for non-commercial use at newmetrica.com
      ii. Called the Glasgow Feline Composite Pain Scale
      iii. Utilizes
         1. Reaction to palpation and interaction. You must get this as a baseline prior to the surgical procedure
         2. Grimace scale
            a. Muzzle position
            b. Ear position
      iv. Suggested to intervene when post-op score, less the baseline, is >7
      v. Various questions with different scores possible
         1. Watching cat in cage
         2. Interacting with cat
         3. Palpating the wound

4. Acute pain and common sense
   a. Best to use a validated scale
      i. But as you walk past the cage…good for distant observation as discussed above
   b. Posture
      i. Cats can try to hide
      ii. But a hunched back is a way cats with withdraw into themselves and try to protect a painful region
   c. Recline
      i. Most normal cats will lie on their side, with all 4 paws close together, generally facing the front of the cage
      ii. Most painful cats will
1. Turn their backs to the front of the cage
2. They may lay flat out

d. Eyes
   i. Normal cats have a round or almond shaped eye opening
      1. As you draw a line from the lateral canthus to the medial canthus of each eye, the lines usually meet on or near the bridge of the nose
   ii. Painful cats have a squint, with the above mentioned line meeting further down the nose.

e. Normal behaviors
   i. Be aware of normal stretching behaviors: Cats do downward dog position even better than dogs
   ii. Grooming, usually starts at back end, and works their way up…not paying too much attention to any one area of body

5. Practice with your staff
   a. A good source of videos is available at animalpain.com It is a Brazilian site but they have PDF scores in English for each of the videos. This is a free site.

6. Chronic Pain
   a. Feline Musculoskeletal Pain Index
      i. Developed at NC State University
      ii. Download for free after filling out questionnaire
      iv. A variety of questions
         1. Each activity ranges from normal to impossible
         2. Read instructions each and every time
         3. Don’t let clients see prior score or answers
      v. The higher the score, the less impairment there is
      vi. Some questions will not be answered as they may not be applicable
         1. Directions on how to get score as a percentage of questions answered
   b. Behavioral changes in chronic pain states
      i. No validation
      ii. Some clients find it easier to answer
      iii. Best to compare to how cat used to act a few years ago
      iv. Samples
         1. Ability to groom…cat may be dirty or matted in certain areas
         2. Using objects to jump up on things. For example using a chair or ottoman to jump up onto a window sill or table
         3. Using objects to jump down from things. For example jumping down from a table using a chair
         4. Sliding down a cabinet or piece of furniture as far as possible prior to jumping.
         5. Inappropriate elimination
            a. Especially going right next to a litter box. Or only getting partway into a litter box because of height of sides or difficulty walking on litter substrate
         6. Difficulty going up or down stairs
         7. Does not initiate or engage in play

7. Final points
   a. Always try to use validated scales
   b. Be proactive whether it is acute or chronic pain
   c. Acute pain is hard to chase down without overmedicating.
      i. Importance of early detection and intervention

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Evaluating Acute and Chronic Pain in Dogs
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1. Causes of Chronic Pain in Dogs
   a. Osteoarthritis
   b. Cancer
      i. Usually osteoarthritis but any type of cancer can cause pain
   c. Neurological issues
      i. Intervertebral Disc Disease
      ii. Cauda Equina Syndrome/Lumbo Sacral Disease
      iii. Syringomyelia
      iv. Neuropathic pain
         1. E.g. diabetes, chronic OA pain
      v. Fibrocartilaginous Embolism
         1. Pain is usually fleeting although paresis issues stay
   d. Neuropathy Causes
      i. Trauma
      ii. Surgery
      iii. L-S lesions
      iv. Spinal cord injury
      v. IVDD
      vi. FCE
      vii. Discospondylitis
   viii. CNS tumors
   ix. Pancreatitis
   x. Inflammatory Bowel Disease
   xi. Saddle Thrombus
   xii. Polyradiculoneuritis
   xiii. Diabetic Neuropath
   xiv. Brachial plexus avulsion
   xv. Fractures
   xvi. Osteosarcoma
   xvii. Nerve sheath tumors
   xviii. Any cancer
   xix. Chemotherapy
   xx. Untreated acute pain
   xxi. Osteoarthritis

2. Chronic pain of OA
   a. Serves no real purpose
      i. Exercise and activity actually improves function
         1. Maintain muscle mass
         2. Maintain ambulation
   b. Considered a maladaptive pain
   c. Evaluation tools for OA
      i. Best evaluated by the owner
         1. In drug studies, things like force-plate analysis are being phased out
         2. Veterinary evaluations don’t carry the weight of owner evaluations
      ii. But! When reported owner observations don’t match with what you are seeing
         1. Realize many owners are optimistic about how well their dog is doing
         2. May be psychological/social/financial reasons for not wanting to report it
   d. Canine Brief Pain Inventory (CBPI)
      i. One of the main go-to evaluation tools in FDA studies
      ii. Adapted from a human tool
      iii. Validated
      iv. Can be use for initial evaluation of OA and for response to treatment
      v. Three parts
         1. 4 pain severity score questions (0 no problem to 10 as worst)
            a. Pain at its worst in the last 7 days
            b. Pain at its least in the last seven days
            c. Pain at its average in the last 7 days
            d. Pain as it is right now
         2. 6 pain interference score questions (0 no problem to 10 as worst)
            a. General activity
            b. Enjoyment of life
c. Ability to rise to standing from lying down
d. Ability to walk
e. Ability to run
f. Ability to climb

3. One overall quality of life question
   a. Overall quality of life in the last 7 days
      i. Poor-fair-good-very good-excellent

e. Client Specific Outcome measures
   i. Several out there
      ii. Cincinnati Orthopedic Disability Index is one example
      iii. You can make up your own questions if you like.
         1. As the client to think of activities, especially ones that were no problem at one point, that their dog has issues with now
         2. Scored from
            a. No problem-a little-quite a bit-severe-impossible
      3. CODI contains standard orthopedic questionnaire as well
         a. Walking
         b. Running
         c. Jumping
d. Getting up
f. Lying down
g. Climbing stairs
h. Descending stairs

f. Things to keep in mind
   i. Always ask clients to think back and compare their dog today as to what he was like a few years ago
      1. Too many people think problems are age related
   ii. Ask clients to be honest; describe how it is instead of what they want

3. Quality of Life Scale
   a. Common topic for dogs with chronic pain issues
      i. Many people see pain issues in their dogs, and don’t say anything until the problem affects THEIR quality of life: carrying dog in and out of house, soiling issues, etc.
   ii. Euthanasia should always be the last resort
   b. Vetmetrica
      i. Web-based HRQL instrument for the dog
      ii. 22 item structured questionnaire completed by the owner in about 5 minutes
      iii. Online data capture and computation of scores
         1. 4 domains of QOL
            a. Energy
            b. Happiness
            c. Comfort
d. Calmness
      iv. Looks at QOL on both a physical and emotional level
   v. Vetmetrica.com

4. Acute Pain Assessments in Dogs
   a. Glasgow Short Form Composite Pain Scale
      i. Distant observation
      ii. Take dog out of kennel and walk it
      iii. Palpation of wound
      iv. Overall assessment of disposition
   b. Colorado State University Acute Pain Scale
      i. Not validated
      ii. Distant and interactive evaluation
   iii. https://www.researchgate.net/figure/Colorado-State-University-Canine-Acute-Pain-Assessment-teaching-tool_fig1_49661913

5. Dysphoria
   a. Often an issue with dogs and opioids
      b. You can get an idea from palpation if it is dysphoria v. pain
      c. Not sure, intervene with some dexmedetomidine
      i. 5 micrograms/kg IV push
      ii. Not enough to sedate, but enough to stop dysphoric behavior
Therapeutic Lasers:  
Clinical Applications and Business Considerations  
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Basic properties of lasers
1. Why laser therapy?
   a. 20% of veterinary practices have lasers
   b. Client driven—they often ask for alternative therapies
   c. Used in Rehabilitation
   d. Much more research supporting use
   e. Better products on market
2. Too many clinics don’t use them at all or underutilize them
3. Terminology
   a. L_A_S_E_R = light amplification by stimulated emission of radiation
   b. Photobiomodulation Therapy (PBMT) is becoming the more popular term
      i. Includes LED therapy, but that is a different lecture
   c. Old terms
      i. Low level laser
      ii. Cold laser
      iii. High Intensity laser
4. Laser Tissue Interactions
   a. Photothermal
      i. Long pulses biological effect due to heating
      ii. Hair removal
      iii. Surgical lasers
   b. Photomechanical/sometimes called photoacoustic
      i. Short pulsed lasers cause ablation
      ii. Tattoo removal
      iii. Photorefractive keratectomy
   c. Photobiochemical
      i. The non-surgical lasers generally used in veterinary practice
      ii. Pain reduction
      iii. PBMT

Mechanism of action of lasers
1. What does PBMT do?
   a. Relieve Pain
   b. Increase microcirculation
   c. Modulate the inflammatory response
   d. Accelerate healing
2. Mechanism of action: Cellular level
   a. Chromophores
      i. Components of various cells and sub-cellular organelles which absorb light.
         1. Mitochondria
         2. Cell membrane
   b. Restoration of energy balance
      i. When enough mitochondria are engaged by CCO absorption, the organelles mediate a series of biochemical reactions that lead to therapeutic clinical outcomes
         1. Increased activity of Cytochrome C Oxidase
         2. Increased mitochondrial function
         3. Increased Oxygen consumption
         4. Increased ATP production
      ii. This leads to
         1. Reduction in pain
         2. Modulation of inflammation
         3. Increased microcirculation
3. How does laser affect pain?
   a. Serotonin increases
   b. Endorphins released
   c. Acetylcholine increases
   d. Bradykinins decrease
   e. Direct analgesic effect on nerves
4. Tissue Interactions
   a. Physics
      i. Power = Energy over time
      ii. 1 watt = 1 Joule second
         1. Joule is unit of energy
      iii. Laser dose is listed in Joules/cm²
      iv. The wavelength governs the depth of penetration into the tissue
   b. Tissue interactions with laser
      i. Can:
         1. Reflect
         2. Scatter
         3. Transmit through
         4. Absorb
      ii. Absorption is what we want
         1. Chromophores are exposed to laser light
         2. Causes alteration in cellular function
         3. Increases healing
   c. Clinical effects
      i. Angiogenesis
      ii. Neovascularization
      iii. Increased collagen production
         1. Reduces scars and improve elasticity
      iv. Increased muscle generation and decreases muscle atrophy
         1. Activates satellite cells responsible for muscle regeneration
      v. Increases bone formation by causing proliferation of osteoblasts
      vi. Increases cartilage production
         1. Increased chondrocyte productivity

Current evidence
1. What do we currently know about lasers
   a. Electromagnetic spectrum
      i. Infrared to near red. The closer to infrared the further it penetrates
   b. Classification
      i. Based on danger posed to eyes
         1. Class 1 and 2 are visible lasers. Safe for accidental viewing
            1. Printers
            2. Scanners
            3. Dvd and CD players
         2. Class 3R
            1. 1-5 mW
            2. Laser pointers, light shows
               a. OK if you don’t stare at them. But potentially hazardous
         3. Class 3B
            1. 5-500 mW
            2. Therapeutic laser
            3. Eye danger
         4. Class 4
            1. >500 mW
            2. Eye danger
               a. Direct viewing
               b. Diffuse reflection
   2. Penetration
      a. Determined by
         1. Wavelength
         2. Power
         3. Dose
      b. Proper penetration
         1. Avoid scattering
         2. Surface absorption
         3. Absorption by unwanted chromophores
      c. Wavelength between 650 and 1000 nm is best for this purpose
      d. Power
         1. Determines the number of photons at the depth of absorption/saturation of tissue
      e. Dosage
         1. Need to determine appropriate number of treatments
         2. Use good technique
3. Emission
   a. Continuous wave
      i. Saturates the tissue with photons faster than a laser emitting in a pulsed mode
   b. Pulsed
      i. Maybe better/safer when treating over an open wound or a painfully sensitive area

4. Comparison of lasers
   a. Example:
      i. Treatment area 300 cm²
      ii. Dosage 10 Joules/cm²
      iii. Target energy delivered 3000 Joules
   b. 5 mW laser 10,000 minutes
   c. 500 mW laser 100 minutes
   d. 3 W laser 16 minutes
   e. 10 W laser 5 minutes

5. Laser Safety
   a. Whether class 3 or 4, use eye protection
   b. Class 4 requires constant movement and adjustment for hair and skin pigmentation because of heat

Use rehab
1. Intervertebral disc disease
   a. Benefits
      i. Reduce cytokines and radicals that infiltrate the spinal cord
      ii. Stimulate neuronal sprouting and regrowth of severed axons
   b. Protocol
      i. 2-8 J/cm² daily until ambulation
      ii. Dorsolateral approach toward the intervertebral foramen and directly on spinous processes
      iii. Treat trigger points in muscles

2. Peripheral Nerve Injuries
   a. Benefits
      i. Peripheral nerve regeneration
      ii. Increased motor neuron survival
   b. Protocol
      i. 1-5 J/cm² daily for three days
      ii. Approach is like spinal cord injury
      iii. Then q2-3 days until resolution
      iv. Warn owners that this is minimum of two months…varies with distance from spinal cord, etc.

Other uses of laser: Pain and chronic conditions
1. Osteoarthritis
   a. Benefits
      i. Pain management
      ii. Reduction of Interleukin 1
      iii. Regrowth and replication of chondroblasts
      iv. Increased NO levels

2. Other chronic issues to consider
   a. Post op pain management
   b. Aural hematomas
   c. Otitis
   d. Lick granulomas
   e. Soft tissue injuries
   f. Wound healing
   g. Anal gland sacculitis
   h. Hot spots, dermatological issues
   i. Gingival stomatitis
   j. Idiopathic cystitis

3. Goal of treatment
   a. Applied until condition is manageable or goal is reached
   b. Administration protocol
      i. Aggressive phase: every other day (or even daily) for at least three therapy sessions
      ii. Transitional phase: gradual reduction in frequency of treatments
         1. Watch for relapse
      iii. Maintenance phase: Therapy as needed

4. Dosage for OA and other conditions
   a. 8-10 J/cm²
   b. Trick for determining dose:
      i. A CD/DVD is 100 cm²
      ii. Size of CD needs 800 to 1000 Joules total
Treating Acute and Chronic Pain in Cats
Michael Petty, DVM, CVPP, CVMA, CAAPM, CCRT
Animal Pain Center
Canton, MI

Acute pain

1. Unique needs of cats
   a. Difficulties in treatment once they leave the clinic
      i. Caregivers often unable to give oral medications
      ii. Caregivers often unwilling to give oral medications
   b. For some cats, each physical interaction causes a progressive worsening in their willingness to cooperate.
   c. Danger to staff administering oral medications or multiple injections to fractious cats.
      i. Cat bites are the #1 worker’s comp claim in veterinary clinics
   d. Hands-off approach to treating pain is the best
      i. CRI’s +/- diffusion catheters can present problems
         1. Twisted iv lines require a lot of baby-sitting
         2. Catheters get chewed out

2. NSAIDS
   a. Both NSAIDS licensed for use in cats have an injectable form and are very effective at treating pain and inflammation of surgery
   b. However, limited dosing options, especially with Metacam.
   c. Problems may be using in cats with renal disease and dehydration
      i. Many clinics only use them after surgery for this reason…but it is better to have on board prior to surgical insult

3. Sodium Channel Blockers: Bupivacaine and lidocaine
   a. Local anesthetics should be used insofar as possible for the prevention of pain
   b. Only class of drug that can stop 100% of pain
   c. Side effects are rare and usually involve overdose: Too much area to treat with a safe dose
   d. Ineffective in areas of infection
   e. Many possible routes
      i. Local
      ii. Regional
      iii. Epidural
      iv. Pleural
      v. Abdominal
      vi. Etc

4. Ketamine
   a. A neurolept analgesic with NMDA suppression
   b. Makes an OK analgesic when used by itself
   c. Makes great analgesic when used in conjunction with opioids, alpha-2’s and locals

5. Opioids
   a. Morphine
   b. Hydromorphone
   c. Methadone
   d. Buprenorphine
      i. Simbadol
   e. Can all be used parenterally watch out for dysphoria and hyperthermia

6. Alpha-2’s
   a. Very useful in cats
   b. Pain and sedation
   c. Best used in combination with opioids and or ketamine
   d. Higher doses when used as stand-alone drug…greater potential for side effects
      i. Even so, adverse events are uncommon
      ii. Reversible
7. New-ish drugs
   a. Onsior tablets and injectable – already discussed earlier
   b. Dexmedetomidine – already discussed earlier
      i. New DexDomitor 0.1 makes is easier to give to small animals like cats.
      ii. Less room for error with low strength drug
   c. Simbadol
      i. High concentration buprenorphine for sub-q use
      ii. One injection 1 hour prior to surgical procedure
      iii. Lasts for 24 hours
      iv. Excellent pain control
      v. FDA approved….no other buprenorphine products are
      vi. Can be used in combination with
         1. Locals
         2. Dexmedetomidine
         3. Ketamine
         4. Nsaids
         5. Always give simbadol first, wait one hour prior to other meds
      vii. Can be given three days in a row: many owners would rather come back than give a pill
      viii. Side effects
         1. Happy cats, euphoric and loopy, but warn the owners
         2. Dilated pupils
   d. Nocita
      i. Off label in cats
      ii. Three day bupivacaine

Chronic pain
1. NSAIDS
   a. Mode of action in cats
      i. As in dogs, NSAIDs have their action by the inhibition of Cox 2
         1. Cox 2 ultimately produces prostaglandins which then cause pain
      ii. As in dogs, big limitation in use is the potential for adverse events
         1. GI, Renal
      iii. Unlike in dogs, largest limitation is regulatory
   b. Meloxicam
      i. Metabolized by oxidation
      ii. Plasma half life varies from cat to cat, about at 24 hours +/-
   c. Robenacoxib
      i. Degrades to from y-lactam
      ii. Plasma half life 1 ½ hours
         1. Persists 24 hours in tissue
   d. Approved use
      i. Meloxicam one single injection
      ii. Robenacoxib 3 days
   e. Off label use with long term administration
      i. Meloxicam labeled dose is onto appropriate for more than a single injection
      ii. Half life of meloxicam can lead to accumulation in plasma, reaching toxic doses with daily administration
         1. Have to give smaller than labeled doses on a daily basis
         2. Has been shown to be safe and effective given long term at doses approved in the EU
      iii. Renal disease is not as big of an issue in cats as it is in dogs. In dogs, most renal disease is glomerular.
      In cats most renal disease is nephritis: can be helped by NSAID
2. Alternative Pain Medications
   a. Amantadine for suppression of the NMDA pathway, hyperalgesia and allodynia
      i. Not many people are using it in cats
      ii. No studies I know of
b. Gabapentin
c. Tramadol
   i. No efficacy studies in cats, high incidence of serotonin syndrome

3. Diet and Nutraceuticals
   a. Glucosamine and chondroitin sulfate supplements
   b. Mobility diets
   c. Soybean Avocado Unsaponifiables
   d. Weight loss

4. Acupuncture
   a. Acupuncture in cats is easier than it sounds
   b. Physical Therapy
      i. Hands on mobilizations and massage
      ii. Strengthening exercises
      iii. Laser therapy
      iv. Underwater treadmill

5. Home makeover
   a. Increase ability to move around and provide environmental enrichment
      i. Looking out windows
      ii. Increased social interactions with humans and other animals in household
   b. Litter box on each floor
      i. Geriatric cat litter
Treating Acute and Chronic Pain in Dogs
Michael Petty, DVM, CVPP, CVMA, CAAPM, CCRT
Animal Pain Center
Canton, MI

Diagnostic considerations
1. Radiographs should be part of every pain exam.
   a. Too many other issues that can mimic osteoarthritis
      i. Cancer
      ii. Dysplasia
      iii. Sequestrum
      iv. Panosteitis
      v. Fracture
      vi. Osteochondritis dessicans
   b. You want to be treating the correct disease
2. Neurologic Examination
   a. Rule out any neurologic issue…many of them mimic pain
   b. It is quick and easy to check most of the cranial nerves
3. Blood work
   a. Treat underlying conditions
   b. Know what may interfere with medications
4. The Owner’s opinion
   a. They may not know what issue they are describing, but often they are able to put their finger on the main issues.
5. When all is said and done, you need evidence of OA or other disease.
   a. It helps to start OA treatments early on

Pharmaceuticals
1. NSAIDs
   a. Considered the gold treatment of OA and many other chronic painful conditions
      i. Give the freaking label dose…having the owner decide minimum effective dose is lose-lose.
      ii. Owners want to spend the smallest amount of money possible
      iii. Are they determining lowest effective dose by administering validated pain questionnaires?
   b. If one NSAID doesn’t work, try another
      i. Some people like ibuprofen better, others like naprosen, etc.
   c. Have a hard discussion about gastric ulcers
      i. In my practice they are asked to stop and call me if
         1. They miss a meal
         2. Vomiting
   d. Monitor with lab work
      i. Start before the first dose
      ii. Every 3-12 months depending on age, comorbidities, etc.
2. Piprant class
   a. Grapiprant
   b. FDA calls it a non-cox inhibiting NSAID. The world health organization calls it a piprant
      i. Prostaglandin receptor antagonist
   c. Seems to have a better safety profile than NSAIDS
      i. But it is still new.
   d. Periodic monitoring: I monitor before starting and then run periodic age-appropriate blood tests
   e. Give according to package insert
3. NMDA antagonists
   a. Amantadine is main one
      i. Not labeled for pain in dogs
      ii. Has some good research behind it
      iii. Can be expensive
      iv. Usually needs to be compounded
1. 100 mg tablets/caplets
2. 3 mg/kg once daily.
b. Methadone also has NMDA activity, but impractical to give long term
   i. Cost
   ii. Stigma
c. Dextromethorphan
   i. Not absorbed well in dogs
d. Ketamine
   i. Can’t send it home
4. Gabapentin
   a. Works mostly by blocking calcium channels at dorsal horn of spinal cord
   b. Probably has its best effect by blocking neuropathic pain
c. Dosing
   i. Nonlinear absorption: in other words doubling the mg given doesn’t double the mg absorbed
   ii. Half-life around 6 hours, give it tid
   iii. Keep increasing the dose until you get the desired effect.
5. Cannabidiol
   a. Derived from hemp
      i. Should have little to no THC
   b. Ongoing studies right now.
c. Great anecdotal stories
   i. Pain
   ii. Appetite
   iii. Mood
   iv. Sleep
d. Buyer beware…get it from a source that tells you:
   i. The concentration
   ii. Results from an outside lab assay
6. Diets
   a. Weight loss diets and mobility diets
   b. Research shows that even a modest weight loss = using an NSAID
c. Mobility diets
   i. Green lip mussels/Omega 3 fatty acids
   ii. Glucosamine/Chondroitin sulfate
   iii. Herbal supplements
   iv. Other
d. No one company has the perfect mobility diet. Actions of many of the ingredients are anecdotal
7. Pain Modifying anti-arthritic agents
   a. Adequan
      i. Only one that is FDA approved for use in dogs
      ii. Meant to be given as a set of injections, not as a chronic treatment
         1. Use at label dose
         2. Use sub-q if you wish
      iii. I give it twice weekly until effect is noted, then once monthly +/- a week
   b. Glucosamine and Chondroitin Sulfate
      i. Must be given together in order to be absorbed/work
      ii. Evidence for it, evidence against it
      iii. I find it works best as a larger package of active ingredients
         1. MSM
         2. Avocado unsaponifiables
         3. Boswelria
         4. Turmeric
         5. Omega-3’s
      iv. Dasuquin Advanced
         1. Has most of the above things in it.
280
8. Other treatments
   a. Synovetin OA
      i. Radioactive tin for injection into arthritic elbow joints
      ii. FDA approved
      iii. Must have RAML certification
      iv. The radioactive tin is absorbed by synoviocytes and phagocytized by macrophages in the synovium
         1. These inflammatory cells are destroyed.

9. Physical Modalities
   a. Laser
      i. Recent advances in laser
      ii. My favorite is Companion
      iii. But if all you can afford is a IIIb, then get it
      iv. Use roughly twice weekly until you see the results you want then increase interval between treatments
         1. Some issues, such as IVDD should be treated daily
         2. All animals respond differently
      v. Don’t use in areas where you want to avoid increased blood flow
         1. Where a local block is still working
         2. Neoplasia
      vi. Don’t use near eyes
      vii. Caution in using over bony prominences
      viii. Be realistic on how deep the laser will reach
   b. Rehabilitation
      i. Poorly utilized in veterinary medicine
      ii. Mostly about hands on manipulations and directed exercises
         1. UWTM is only about 3% of rehab
      iii. Uses
         1. Post-orthopedic procedures
         2. Acute injuries
         3. Chronic pain of OA
         4. Neuropathies
         5. Muscle tendon and ligament tears and injury
         6. Vestibular syndrome
      iv. Homework
         1. Just as anyone here who has gone through PT, you know that it is also about the stuff you do at home
         2. Can’t maintain gains with just 1-2 weekly sessions
   c. Extracorporeal Shockwave Therapy
      i. Works by???
         1. Biologic response
            a. Inflammation followed by anti-inflammatories
            b. Turning a chronic condition into an acute one
            c. Release of growth factors
            d. Release of myofascial trigger points
         2. A few good studies out there that shows it works
      ii. Three types
         1. Piezoelectric (like your dental scaler)
         2. Electromagnetic (like a loudspeaker)
         3. Electrohydraulic (electricity through water producing sound waves)
         4. Piezoelectric and electrohydraulic are the two most common units in veterinary medicine
            a. Piezoelectric doesn’t hurt
            b. Electrohydraulic hurts
               i. Sedation
            ii. Maybe works better? Has good paper on it published in AAHA
   d. Massage
      i. Three types of massage
1. Petrisage
2. Effleurage
3. Tapotment

ii. All three techniques can be learned in *Canine Medical Massage* by Narda Robinson

e. Home Makeover
   i. Make it easier for dog and owner at home
   ii. Variety of things
      1. Ramps
      2. Harness
      3. Steps
      4. Non-slip surfaces
         a. Yoga mats
         b. Rubber backed rugs
      5. Baby gates: going up and down stairs all day could be an issue

f. Acupuncture
   i. Especially useful when a chronically painful animal can’t take medications for whatever reason
   ii. Medical acupuncture is an old treatment with a modern update
   iii. Many known actions
      1. Local response to needle insertion
      2. Humoral actions
      3. Effect on CNS
      4. Pituitary
      5. Others
   iv. Few contraindications
      1. Bleeders
      2. Skin infection
      3. Putting needle in or near a tumor
   v. Can’t acupuncture in the wrong direction
   vi. Pain
      1. Acute or chronic
   vii. Neurologic issues
      1. IVDD
      2. FCE
      3. Cauda Equina syndrome
   viii. Medical acupuncture for veterinarians
      1. [https://curacore.org](https://curacore.org)

g. Myofascial Pain Syndrome
   i. Commonly overlooked
   ii. Issue known for hundreds of years
   iii. Etiology
      1. Sustained contractions of muscles can cause cramping in set of muscle fibers
      2. These contractions or trigger points can become permanent
      3. Cinderella Hypothesis
         a. Whenever a limb has a sustained contraction, the same set of muscle fibers within any given muscle are the ones that maintain that contraction
         b. No matter how exhausted those muscles become, no other muscle fibers “help out”
         c. Eventually there is a depletion of ATP within the muscle fibers, which is necessary for the muscle to un-contract
   iv. Impact on DJD
      1. Chronically contracted fibers shorten overall muscle length
      2. This can lead to decreased joint width space
         a. Abnormal function of joint
         b. Increased inflammation and wear-and-tear
Acute pain management

1. Opioids
   a. Ideal for acute pain.
   b. No place for chronic pain
   c. Oral administration doesn’t work so well
      i. Exception is buprenorphine. But do you really want to send home an injectable opioid?
   d. Use as
      i. Part of local block-extends duration
      ii. Epidural
      iii. CRI
      iv. Parenteral
   e. Opioids are currently in short supply
      i. Consider the use of Simbadol
         1. No shortage
         2. Can use in dogs, but not at cat dose.
            a. Use dose and route in Plumbs for Buprenorphine in dogs

2. NSAIDs
   a. Give preoperatively
      i. Not in cases of renal issues
      ii. Don’t use if you are unable to monitor and treat low blood pressure issues
      iii. Don’t use in GI surgery
   b. Is a go-to pain reliever post op pain
   c. Grapiprant
      i. Only labeled for chronic pain
      ii. I have used it for acute pain in some of the above contraindications with good effect

3. Local anesthetics
   a. Should be used for every surgery insofar as possible
   b. The only true analgesic
   c. Methods
      i. Local
      ii. Regional
      iii. CRI
      iv. Epidural
   d. Nocita
      i. 3 day bupivacaine
      ii. Can only use in dog cruciate surgery. As if.
      iii. Cat study under way

4. Alpha-2’s
   a. Dexmedetomidine is most common in the US
   b. Great adjunct drug
      i. Opioids
      ii. Part of CRI
      iii. Rescue drug

5. Icing
   a. 15 minutes of icing twice daily provides up to 24 hours of pain relief
Dirofilaria immitis – heartworm
I.H. – mosquitoes
D.H. - dogs and wild canidae, marine mammals, ferrets, cats

♂ 12-22 cm (6-9 inches)
♀ 25-31 cm (12-14 inches)
Mf 300 - 325 x 6 - 7 µm
PPP 6 months

Epidemiology of heartworms in cats
• Often arrested development of heartworms in cats due to being aberrant host
• One heartworm can cause disease or even death
• Higher percentage of HW positive cats than dogs reported by Companion Animal Parasite Council (CAPC)
  o Due to lower compliance of use of monthly preventative use in cats

Clinical signs
• Respiratory disease is seen more frequently in cats
  o Often see eosinophilia and basophilia
• May present as sudden death

Diagnostic tests in cats
Use both antigen and antibody test
• Antigen Test Kits
  o Only detects adult female worms.
• Average worm burden in the cat is 1-2 worms and is frequently only males.
  “Asthma” like syndrome occurs 3-4 months post infection. Antigen test incapable of confirming HW as etiology
• Antigen tests:
  o Detect antigen produced by adult worms (Produced by adult, female worms)
  o First detection at 5-8 months P.I.
• Antibody tests:
  o Detect antibody produced against larval and adult worms
  o First detection at about 3 months
• Infrequently see microfilariae on Knott’s test

Feline heartworm treatment goals
• Relieve acute signs (usually respiratory) May be due to adult or larval infection
• Control chronic signs (respiratory, vomiting)
  o Prednisone (2 mg/kg-decreasing doses one month)
• Prevent reinfection - prophylaxis
• Rid patient of Adults via surgery (possible? advisable?)

Prevention
• All cats should be on yearlong heartworm preventative, regardless if indoor only pets
• Minimizing mosquito breeding environments around the house
Climate Change and Its Impact on Parasites: Are We Seeing the Tip of the Iceberg?
Richard Gerhold, DVM, MS, PhD
University of Tennessee
Knoxville, TN

Dirofilaria immitis – heartworms
I.H. – mosquitoes
D.H. - dogs and wild canidae, marine mammals, ferrets, cats

Reasons my climate change may be influencing expanding populations of heartworm disease.
• Climate change- warming environment can lead to increase in larval stage development in mosquitos
• Increase in mosquito populations in cooler months
• Allows certain mosquito spp to inhabit northern or higher altitude regions not previously occupied by vector species
• Urbanization- leads to more areas of standing water (flower pots, old tires, buckets, etc) for mosquitos to breed
• Incidence of canine and human dirofilariasis has increased in certain regions likely due to warming climates
• Similar trends of increasing infection are expected for other filarid species of humans, domestic and wild animals.
• This information has already been observed in certain northern states in the US.

Trichostrongyles
• Warming climate has potential to minimize hypobiosis leading to increased reproductive output of female worms.
• Increases duration of when infective larvae are available on pasture for consumption by definitive host

Ancylostoma
• Increase air and soil temperatures will likely lead to increased abundance of infective L3 larvae in the environment.
  o This has been documented with high incidence of human cutaneous larval migrans due to both canine and feline hookworm species during unordinary warm periods in various regions.

Lungworms
• Lungworm infections are dependent on gastropod intermediate hosts for development to third stage infective larvae
  o Development in gastropods is temperature dependent
• Warming trends strongly suggest higher infection rates of gastropods with various lungworm species including but not limited to Parelaphostrongylus tenuis, Protostrongylus spp. Aelurostrongylus, and Muellerius.
• These models have already been validated for lungworm infections in muskox and other arctic species.

Toxoplasmosis
• Cats serve as definitive hosts and numerous mammals and birds are the intermediate hosts
  o Most cats in the wild become infected shortly after weaning
  o Mice are the usual intermediate host and a normal predator-prey relationship exists between the cats and mice that enhances transmission
• In the environment, sporulation occurs in 1 – 5 days; under favorable conditions, sporocysts can survive about 18 mos.; can survive in fresh and salt water—Sporulation can occur faster in warmer environments – leading to more rapid production of oocysts in the environment.
  o Toxoplasmosis has been observed in certain arctic species
• Potentially increasing risk for domestic animals and humans.

Coccidia
• Similar to Toxoplasma above, in the environment, coccidian sporulation occurs in 1 – 5 days; under favorable conditions, sporocysts can survive about 18 mos.;—Sporulation can occur faster in warmer environments – leading to more rapid production of oocysts in the environment.
  o Higher oocyst production and ingestion leads to greater pathology due to parasite replication within the host cells.

Trypanosoma cruzi (Chagas disease)
• It is estimated that between 6 and 8 million people worldwide are infected and have Chagas disease, with estimates of up to 100 million people at risk for infection.
• Disease is transmitted through feces of infected triatomine bugs.
• There are 21 countries in the Americas (mainly central and south America) that are documented to be endemic for the disease.
• Disease emergence is occurring in Texas.
• Acute and chronic disease manifestations. Chronic disease often associated with heart and intestinal disease.
• With changing climates, the distribution of Chagas is pushing north with greater numbers of cases being documented in the southern United States, especially in hunting dogs and those used for border patrol.

**Trichomonas gallinae**
• Protozoa persisted longer in artificial water baths in warmer temperatures (~33 C) compared to room temperature (~25C).
• Warmer temperatures could lead to greater infection rates for birds. Perhaps may have similar implications for *T. foetus* in cats.

**Acanthamoeba and Naegleria fowleri**
• Both parasites are associated with sensory system infections in humans.
• *Naegleria fowleri* can lead to rapid, progressive, encephalitis that is often fatal.
• *Acanthameoba* can cause severe ulcerative keratitis
• Both parasites replicate higher in water where eutrophication occurs. Warmer temperatures are expected to increase bacterial populations which in turn can lead to increase in these parasites
Making Sense of Test Results for Tick-Borne Diseases
Richard Gerhold, DVM, MS, PhD
University of Tennessee
Knoxville, TN

Ticks and tick-borne diseases
Tick-borne diseases are extremely important and emerging diseases in the United States. The area in which you live will influence the diseases that are circulating in the environment. Although diseases such as Lyme disease has received a great deal of attention, other important diseases including ehrlichiosis, Rocky Mountain spotted fever, anaplasmosis and cyttauxzoonosis have been emerging in various areas. A good travel history is imperative given various species of ticks and tick-borne diseases are more common in certain geographical areas. More information on tick-borne disease distribution can be found at http://www.capcvet.org/parasite-prevalence-maps/

Diagnosis of tick-borne diseases: Serology vs PCR
Testing is warranted on animals with the aforementioned clinical signs. PCR testing (detection of pathogen DNA) is more sensitive than serology for detection of Rocky Mountain spotted fever and Anaplasma/Ehrlichia species during the acute phase of the disease, prior to the development of an antibody response. Therefore, if an animal presents with acute signs suggestive of tick-borne disease (i.e. fever, lethargy, thrombocytopenia, leukopenia, arthropathy, neurologic dysfunction), the best test for diagnosis is the PCR. Whole blood (EDTA) should be obtained for the test, prior to antibiotic administration.

Serology is useful for detection of chronic/persistent infections, during which the numbers of pathogens are lower or absent from circulation and cannot be detected as easily by PCR. This is particularly true for Lyme disease. This organism localizes in the tissues and is difficult to detect in the blood. It is important to note that antibodies to these tick-borne agents may persist for several months to years (especially for E. canis), so detection of antibodies does not distinguish current infection from previous exposure. Also, high seroprevalence to these agents has been documented in healthy dogs in endemic areas, such as the Southern USA, and most dogs exposed to Anaplasma or Ehrlichia species will not develop overt clinical disease. Therefore, PCR of skin or other tissues (not blood) and/or complete blood count is useful to determine if seropositive animals are currently infected and have clinical disease.

Identification of ticks
Tick bodies are divided into two primary sections including fused head and thorax and abdomen. All adult and nymphal forms have 4 pairs legs and no antennae and all larval forms have 3 pairs of legs. The importance of determining larvae vs other stages include to determine the likelihood of tick being infected with various pathogens. Unless transovarial transmission occurs, larvae are unlikely to be infected with pathogens, while nymphs and adults have higher likelihood include with pathogens in transstadial transmission. Whereas hard ticks have scutum, soft ticks do not have scutum. Ticks are great vectors due to their ability to be persistent blood-suckers which attach firmly & feed slowly, long life spans, may be geographically widespread, resistant to environmental conditions, high reproductive potential, and can pass infective agents through egg to next generation and/or through successive stages. Ticks bites in themselves can lead to wounds and Inflammation from salivary proteins. Secondary infection and disease can be due to toxicosis, local necrosis, and tick paralysis. Tick bites predispose animals to secondary attacks by myiasis-producing flies.

Soft tick have no scutum are soft, tough, leathery body, do not stay attached—instead take multiple small volumes of blood, and often feed at night.

Soft ticks include Otobius megnini (Spinose Ear Tick) transmits relapsing fever caused by a Borrelia spp. (different than Borrelia burgdorferi which causes Lyme Disease). Spinose ear ticks are more common in western states that are west of 100th meridian. Hard Ticks is largest family of ticks has a scutum (dorsal, hardened plate) that covers entire dorsum of males and forms an anterior shield in females. Hard ticks remain attached until engorged and then fall off to molt or lay eggs. General life cycle include:

- Egg \(\rightarrow\) 6-legged larva \(\rightarrow\) 8-legged nymph \(\rightarrow\) 8-legged adult
- Oviposition (egg laying) occurs off of the host

Nymphs and adults can be identified based on visual exam but often unable to distinguish larvae without microscopic exam. Nymphs and adults are more likely to harbor pathogens than larvae—this is why you need to be able to distinguish larvae (6 legs) from nymphs/adults (8 legs).

Tick species
All Dermacentor spp.
- Ornate ticks with eyes
- Basis capitulum (mouth part) is rectangular if viewed from above and has stubby palps
- Resembles Rhipicephalus (both have11 festoons, small rectangular patterns on posterior abdomen)

Dermacentor variabilis (American dog tick)
- Eastern half of U.S. and west coast, but rare in Central US
- Dogs, cats, humans, horses, cattle, fox, rodents, and other mammals
• Can cause tick paralysis in humans, dogs, etc.
• May take as little as 3 months, with favorable conditions, or up to 2 years
• Principal vector of *Rickettsia rickettsia* - Rocky Mountain Spotted Fever (RMSF) and others in Spotted Fever Group
• Infrequent vectors of tularemia, anaplasmosis, *Babesia canis*, *Cytaxuzoon felis*

**Rhipicephalus sanguineus** (Brown Dog Tick)

• Wide distribution
• *Rhipicephalus* ticks are similar in appearance to *Dermacentor*, except they have a hexagonal basis capitulum. All stages parasitize on dogs and will attach to other animals, but usually not humans
• Can survive indoors for months to possibly years without a blood meal
• Domestic & kennel problem due to tropical nature of tick and because it cannot survive outdoors in North America
• Vectors *Babesia canis voglei*, tularemia, *Ehrlichia canis*, RMSF

**Rhipicephalus (Boophilus) annulatus** (Cattle Fever Tick)

• Southern U.S. & Mexico—spreading North into lower Texas
• Parasitize mainly on cattle; also deer, horses, donkeys, sheep, etc. in other countries, not U.S. and Mexico
• 1-host tick in U.S. and Mexico—re-emerging cases on US/Mexican border—large concern for USDA
• First demonstrated tick-borne disease
• *Babesia bigemina* (Texas cattle fever) VERY important disease to cattle industry—causes severe anemia and death in cattle and is a reportable tick species!

All Amblyomma spp.

• Ornate ticks
• Long mouth parts & commonly 11 festoons—allows one to differentiate from *Ixodes* spp which lack festoons

**Amblyomma americanum** (Lone Star Tick)

• Wide distribution, but mainly in southern U.S.
• Large silver spot at apex of scutum on females—hence name “lone star”
• All stages feed on wild & domestic animals, birds, & humans and is significant pest for humans & animals
• Can transmit *Coxiella burnetii* (Q-fever), tularemia, *Ehrlichia chaffeensis*, *Ehrlichia ewingii*, RMSF, *Cytaxuzoon felis* (cats)
• Vectors agent of Southern Tick Associated Rash Infection (STARI) in humans
• Cause of STARI is currently unknown—may actually be the host reaction to tick saliva—leads to swelling and pain at bite region

**Amblyomma maculatum** (Gulf Coast Tick)

• Southeastern US in Gulf coast region, but has expanded range recently
• Ornate scutum—often confused with *Dermacentor*—examine mouth parts to differentiate
• Adults attack nearly all animals & humans and can transmit *Hepatozoon americanum* Hepatozoonosis—dog must eat tick to be infected with Hepatozoon

All *Ixodes* spp.

• Ornate ticks and no festoons, has distinct anal groove anterolateral to anal orifice
• Used for identification in NON-ENGORGED tick but can’t see groove in engorged ticks—use mouth parts instead

**Ixodes scapularis** (Black-Legged Tick)

• Wide distribution, in East, South, and Midwest U.S. Highest populations in upper Midwest and New England/midatlantic states
• Primary Lyme disease (*Borrelia burgdorferi*) vector in Eastern US and Midwest
• Vectors *Babesia microti*, *Anaplasma phagocytophila*

**Ixodes pacificus** (California Black-Legged Tick)

• Primary Lyme disease vector in the West Coast

**Tick-borne diseases**

**Tick paralysis**

Potentially fatal reaction to a paralyzing neuromuscular toxin secreted in the saliva of a female tick late in her feeding. Cattle, sheep, horses, dogs, and humans seem to be most affected.

Clinical signs include: headache, vomiting, general malaise, loss of motor function and reflexes, followed by paralysis that starts in the lower body and spreads to the rest of the body

Respiratory failure and death can result. Signs disappear rapidly when tick is removed, suggesting that the toxin is rapidly excreted or destroyed

**Lyme Borreliosis**

• Agent: *Borrelia burgdorferi*
Vector: Ixodes scapularis (Eastern and Midwestern US), Ixodes pacificus (Western US)


Animal health: Major cause of canine and equine disease, including endocarditis and joint pain. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.

Human health: Acute and chronic diseases including joint pain, heart disease, and neurological disorders. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.

Diagnoses: Lyme disease is diagnosed using serology tests, bacterial cultures, and/or PCR of tissue (NOT BLOOD). Blood may be used for PCR in very acute cases, otherwise tissue biopsy is needed. Predictive value influences serological test interpretations—only treat animals with clinical signs suggestive of disease!!!

Rocky Mountain Spotted Fever

Agent: Rickettsia rickettsia

Sometimes placed in “Spotted Fever” disease group

Vector: Dermacentor variabilis

Geographical distribution: Eastern US mainly. Most frequently reported tick borne disease in the eastern US. Animal health: Recent evidence has shown that untreated RMSF may lead to death of the affected animal. Clinical signs include whole body pain and are painful on palpation.

Cytauxzoon felis

Piromplasm of cats. Bobcats are reservoir host that is transmitted by Amblyomma americanum. Clinical signs: fever, dehydration, icterus, lymphadenomegaly, and hepatosplenomegaly. Treatment with atovaquone plus azithromycin. Diagnosis: PCR, blood smear (negative blood smear does not rule out infection) since early stage only see schizonts in macrophages. Prevention: Keep cats indoors!! Use preventative for tick infestation

Anaplasma phagocytophilum

Intracellular rickettsia that causes human granulocytic anaplasmosis

Inflicts granulocytes and leads to bleeding, fever, leukopenia,

Clinical signs/symptoms may be worse with co-infection with Lyme or Babesia

Vectored by Ixodes scapularis so same geographical distribution as Lyme Disease. Can be transmitted by blood transfusion.

Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in granulocytes.

Don’t treat animals that are clinically normal but are only seropositive—potential false positive due to positive predictive value.

Treatment with doxycycline or minocycline

Anaplasma platys

Intracellular rickettsia that causes infectious cyclic thrombocytopenia in dogs

Common clinical signs include bleeding, due to cyclic thrombocytopenia…may be worse with co-infection with Ehrlichia canis, which is transmitted by same tick.

Transmitted by Rhipicephalus sanguineus –worldwide distribution

Diagnosis: clinical signs, PCR (acute cases), serology (chronic). Don’t treat animals that are clinically normal but are only seropositive—potential false positive due to positive predictive value.

Treatment with doxycycline or minocycline

Ehrlichia canis

Intracellular rickettsia that causes canine ehrlichiosis

Infests monocytes and leads to fever, anorexia, lethargy, thrombocytopenia, lymphadenopathy, edema, bone marrow suppression.

The acute stage is mainly due to a vasculitis. E. canis replicates in monocytes. The infected monocytes bind to vascular endothelial cells and leads to a vasculitis

Transmitted by Rhipicephalus sanguineus –worldwide distribution

Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in monocytes

Don’t treat animals that are clinically normal but are only seropositive—potential false positive due to positive predictive value.

Treatment with doxycycline or minocycline
Sniffing Out the Facts: Slow Kill vs. Melarsomine for Canine Heartworm Treatment

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Dirofilaria immitis – heartworm
I.H. – mosquitoes
D.H. - dogs and wild canidae, marine mammals, ferrets, cats

♂ 12-22 cm (6-9 inches)
♀ 25-31 cm (12-14 inches)
Mf 300 - 325 x 6 - 7 µm
PPP 6 months

Life cycle
- Juvenile worm matures to adult over next 3 months in dog.
- Microfilaria produced by young adult worms 6 months post infection (6 month Life Cycle)
- Male worms 6-9 inches, females 12-14 inches
- Lifespan is 5 to 7 years in the dog
- Average infection is 14 worms but can be in excess of 100

Clinical signs
- Cough
- Dyspnea
- Tiring on exercise
- Weight loss
- Classic patient: Active middle-aged dog
- Ascites
- Anemia
- Eosinophilia and thrombocytopenia
- Glomerulonephritis and proteinuria

Reasons for a dog to be AG positive and Knott’s/Filter negative
- 5 month old worms (too young –rem PPP)
- All female worms (single sex)
- Immunological Occult
- Prophylaxis/Drug induced
- Few mf present

Reason for a dog to be MF positive and AG negative
1. Adults dead/mf circulating
2. Ag sequestration/antigen antibody complexes

Time of testing
- The earliest heartworm antigen is detected is 5 months post infection
- With low worm burdens or animals on macrocyclic lactone preventives, antigenemia can be delayed to 9 mos.

What tests are recommended during annual physical exam?
- Serology for heartworm antigen AND
- Microfilariae concentration test

Notes on testing recommendations from AHS
- Antigen testing - most sensitive diagnostic method when screening an asymptomatic dog or seeking verification of a suspected heartworm infection
- But a study conducted on shelter dogs found a 7.1 percent false negative rate because of formation of antigen-antibody complexes.
• AHS now recommends mf testing in tandem with AG to detect dogs that are AG- but mf+

What would you do before treatment?
Evaluate the dog
• Already have results from Knott’s & Ag test

Radiography to assess severity of cardiopulmonary disease
• enlarged, tortuous, and often truncated peripheral intralobar and interlobar branches of the pulmonary arteries, particularly in the diaphragmatic (caudal) lobes
• pulmonary parenchymal disease, right heart enlargement etc

Echocardiography

Stabilize dogs presenting with clinical heartworm disease

Treatment AHS/CAPC -3 immiticide dose regimen
• Safety
• Efficacy
• Decreased possibility of needing further melarsomine treatment
• By initially killing fewer worms and completing the treatment in two stages
  o reduces cumulative impact of worm emboli on severely diseased pulmonary arteries and lungs

Current treatment protocol for positive dogs
First month
• Start macrocyclic lactone (preventive) and continue monthly for life
• Rx Doxycycline 10mg/kg bid for 4 weeks
  o If dog can not tolerate dose, reduce to 5mg/kg
  o (Wolbachia nos will remain low for 3 to 4 mos)
• If dog symptomatic, Rx Prednisone 1mg/kg reducing weekly during 1st month.
• Begin exercise restriction.

Second month
• Give second dose of heartworm preventive.

Third month
• Give third dose of heartworm preventive.
• Give one injection melarsomine (Day 61).
• Rx Prednisone 1mg/kg reducing weekly.
• Decrease activity level even further. Cage rest in more severe cases.

Fourth month
• Give fourth dose of heartworm preventive
• Give second and third melarsomine injections (Day 90 & 91).
• Rx Prednisone 1mg/kg reducing weekly for four additional weeks.
• Continue exercise restriction for 6 to 8 weeks after last melarsomine injections.
• Antigen test in 6 months
• Knott’s test or other test for microfilariae in 6 months
• Any treatment method utilizing only macrocyclic lactones as a slow-kill adulticide is not recommended!!!
• New information about resistance also prompted the AHS to place additional emphasis on the importance of year-round administration of heartworm preventives.
Dirofilaria immitis – heartworm

I.H. – mosquitoes

D.H. - dogs and wild canidae, marine mammals, ferrets, cats

**Reasons for expanding populations of heartworm disease.**

- Decrease in funding for mosquito control
- Movement of infected dogs into areas not traditionally associated with heartworm
- Compliance of heartworm preventatives
- False positive results on antigen test
- Reason why to use Knott’s test on all testing
- Resistance to certain preventative
- Climate change
- Urbanization

Toxoplasmosis

**Hosts/Disease**

- Cats serve as definitive hosts and numerous mammals and birds are the intermediate hosts
  - Most cats in the wild become infected shortly after weaning
  - Mice are the usual intermediate host and a normal predator-prey relationship exists between the cats and mice that enhances transmission

**Epidemiology**

- Sporulated oocysts contain two sporocysts each with 4 sporozoites (2 x 4 architecture)
- In the environment, sporulation occurs in 1 – 5 days; under favorable conditions, sporocysts can survive about 18 mos.; can survive in fresh and salt water
- Seroprevalence of *T. gondii*
  - Serology is not useful in predicting shedding of oocysts by cats → oocysts shed prior to antibody formation
- Infection routes for cats
  - Carnivorism (primary)
  - Transplacental
  - Oocyst ingestion (lowest)
  - Cats can be both definitive and intermediate hosts
    - If intermediate host, usually see lung infections and pneumonia in cats

**Human epidemiology**

- Fecal-oral ingestion of oocysts (primary way humans are infected in US)-most important infection of humans
- Ingestion of tachyzoites and/or bradyzoites in undercooked meat and raw milk (goat’s milk esp., unpasteurized),
- congenital
- Organ transplant
- Blood transfusions (much less common)

**Pathology & pathogenesis**

- Pathology varies with strain of parasite, age of host, organs invaded, immune status of host, species of host
  - Enteritis
  - Hepatitis
  - Pneumonitis
  - Myocarditis
  - Chorioretinitis
  - Encephalitis
  - Placentitis
  - abortion
Clinical signs of congenital infection

- *T. gondii* naïve woman stands a 20-50% probability of passing infection to fetus if infected during pregnancy
- Earlier infection, more damaging to fetus

Control

- Keep cats indoors
- Discourage feral cat colonies and educate owners about *Toxoplasma* risks due to predation of intermediate hosts
- Keep cats away from livestock
- Keep cats away from sand boxes & public parks, and beaches
- Adequately cook meat
- Freeze meats before eating- freezing kills tissue cysts

Trypanosoma cruzi

Epidemiology and distribution

It is estimated that between 6 and 8 million people worldwide are infected and have Chagas disease, with estimates of up to 100 million people at risk for infection. There are 21 countries in the Americas that are documented to be endemic for the disease. With changing climates, the distribution of Chagas is pushing north with greater numbers of cases being documented in the southern United States, especially in hunting dogs and those used for border patrol.

The vector

Triatominine bugs, reduviid bugs, or the “kissing bug”. These insects are part of the family Reduvidae and can be identified by their thin, cone-shaped heads, thin antennae and legs. They can range from brown to black in color, but have orange-colored stripes around the edge of their bodies. These bugs are most active at night when they feed on humans and other mammals and live in aggregate refuges (sheltered environments) during the day. The parasite is transmitted in feces of infected bugs.

Clinical manifestations

**Acute disease**

This phase generally lasts 4-8 weeks and can be asymptomatic or be a self-limiting febrile phase. There may be swelling around the site of inoculation (chagoma), or when the inoculation is conjunctival, periorbital swelling can occur (Romaña’s sign). In rare cases, the infected individual may experience myocarditis or meningoencephalitis (5-10% of cases) and these cases will often result in death. Treatment with appropriate trypanocidal drugs in the acute phase has between 50% and 80% cure rate. In ~90% of cases of acute Chagas, clinical manifestations will resolve spontaneously with or without treatment. Of these, approximately 60-70% will never develop clinically apparent disease, termed intermediate chronic form.

**Chronic disease and complications**

In the intermediate chronic form of the disease, there are no clinical signs of Chagas, but the individual remains infected with the parasite. Treatment at this stage only results in 20-60% cure rate, and many cases stay in this phase, unless they are reactivated.

The determinate form of chronic Chagas becomes apparent an average of 10-30 years after initial infection. 30-40% of chronic cases will develop cardiomyopathy, digestive megasymphdromes (colon or esophagus), or both of these. Treatment at this stage results in variable, but consistently low rates of cure.

**Screwworm (Cochliomyia spp.)**

Screwworm larvae feed on live tissue of vertebrates including humans. Introduction of screwworms into Florida keys caused great concerns for key deer, domestic pets, and human health. Movement of screwworms can occur via movement of infected animals or the larval or adult stages. Larvae have paired tracheal trunks that are conspicuous for this parasite. Any larvae with morphology suggestive of screwworms should be collected and submitted to state or federal authorities.

**Emerging tick populations**

Numerous tick populations are expanding and in addition discovery of invasive tick species on livestock. This includes finding of large numbers of *Haemaphysalis longicornis* on sheep in New Jersey that had no travel outside of the US. This tick infests humans and animals alike and the species is parthenogenetic. It is unknown what effects this tick can have on human or animal health in the US.

Other tick species include the tropical bont tick (*Amblyomma variegatum*) which is found in Caribbean islands and infrequently on Florida Keys. This tick transmits the agent of heartwater disease that can have significant consequences for livestock and wild ungulate health.
The “down dog” can arise (pun intended) from many causes. Some are orthopedic (bilateral CCL rupture) but the majority are neurologic in nature. They can be further broken down into acute and chronic conditions.

Acute down dog examples are IVDD, FCE, tick paralysis, trauma and metabolic conditions. Chronic origin examples are lumbosacral disease, degenerative myelopathy and diskospondylitis.

A through history and physical examination is a requirement to further understand these patients. A minimal data base (CBC/Chem/UA, plus Thyroid or Urine Culture if indicated) should be done on all patients. Survey radiographs can be extremely helpful in providing practical information. Referral for advanced consultation, imaging (CT/MRI) or other studies (nerve conduction) and surgical intervention (if indicated) may also be needed.

The rehabilitation practitioner may encounter these patients in the peracute setting, or after specialist intervention. Regardless of timing, they must be able to provide a baseline of practical and safe care to assist the patient. General practitioners can also provide a standard of care, and should also be aware of referral to a rehabilitation practitioner (CCRP or CCRT) or boarded rehab specialist (ACVSMR).

Establishing a baseline and then proper communication with the owner is vital. These cases can be “down” for 1-2 days or 1-2 months, or permanently. Understanding prognosis, as well as financial and emotional commitment to these patients is critical. The author recommends working in 2 week intervals, and not committing anyone to a 3 month timeframe. Continuous reassessment and communication is vital.

The team approach to these patients is critical. Establishing a relationship with the patient and the owner will allow for better understanding of the case, and of each patient’s needs. Some cases may be managed as outpatient, while some may require inpatient care. With outpatient, it is recommended to have the patient left for the majority of the business day, so as to slowly work with them. Inpatient care should be discussed and the owner made aware of who will monitor the patient overnight, and care for them on weekends.

Fundamental nursing care is essential for these patients. Changing recumbency every 4 hours (and attempting to keep sternal during daytime) is vital for both physical and mental well being. Clean soft bedding, with appropriate padding (to minimize risk for pressure sores) is needed.

Progressing to standing, items such as carts, harnesses or hoists can be quite useful to save the staff members. Working through assisted standing, partial assistance and then eventually standby assisted standing exercises will depend on each individual patient.

In between standing exercises (done 4-6x a day, or more) the patient should rest. Additional modalities to be used when the patient is resting includes, but is not limited to: LASER, passive range of motion, massage, thermotherapy, cryotherapy, and electrical stimulation.

Bladder and bowel expression may be needed. If so, it should be done in a safe manner. The author recommends avoiding urinary catheters (indwelling or temporary) after the initial 72-96 hours, so that determinations can be made on patient progress. Additionally, catheters provide an access point for infection. Urinary medications (phenoxybenzamine, bethanecol) should be used carefully, and after ruling out any infection.

Bowel expression may needed as well. This can be done in a variety of ways, including manual emptying. The important thing to remember is to give the patient TIME (in a harness, hoist, etc.) to void on their OWN. The author has found that many clinicians and especially owners are not patient enough to give these patients the time they need to void on their own.

For bowels, enemas, oral lactulose and movement (walking) can help facilitate proper bowel movements.

Referral from a general practice/ER to either a rehab practitioner or boarded sports medicine-rehabilitationist should be considered early for these challenging cases. The large breed dogs themselves present unique problems that may not be readily fixable in most practice settings.

Progress can be made with these patients slowly and with proper communication to the owner, reasonable goals can be set and attempted to meet.

Selected references

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Abstract: Rehabilitation is becoming a standard of care in veterinary medicine. As this field grows and owners’ expectations for compassionate care and return to function and activity expand, there is a necessity to offer better and faster options. Laser therapy is a vital and practical cog in the wheel of rehabilitative options. Laser therapy can be utilized for a wide variety of cases, making it a feasible and profitable part of practice. Injuries of many types will benefit from laser treatments. With very few exceptions, cases with injuries can be treated with photobiomodulation therapy as opposed to other modalities that may be limited by a patient’s other medical conditions. This nonpharmaceutical options for treatment can be an asset in veterinary medicine.

**Laser in Veterinary Rehabilitation**

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Laser therapy is generally administered with a hand-held probe, with a small beam area that is useful to treat small surfaces; other lasers have several beam areas in the same unit to treat larger areas. Laser energy may be applied with the laser probe in contact with the skin, which eliminates reflection from the skin and minimizes beam divergence, or with the probe not held in contact. With the noncontact method, it is necessary to hold the probe perpendicular to the treatment area to minimize wave reflection and beam divergence. Noncontact application is recommended for wound treatment. The appropriate dosage may be applied to larger areas by administering the calculated dose to each individual site in a grid fashion, or by slowly moving the probe over the entire surface, being certain to evenly distribute the energy to each site. In any case, the probe should be held perpendicular to the skin. A coupling medium is not necessary, as in ultrasound, because the laser beam is not attenuated by air. Lasers are safe but proper integration into the practice is essential. Most companies will provide laser safety training. Goggles (for all those in the room, including the patient) should be worn when using laser therapy. The goggles must be specific to the wavelength of laser light being used.

Veterinary rehabilitation does not focus on one specific problem, but rather sees the patient as a whole. One of the main goals of rehabilitation is a return to normal or near normal function. The main effects of lasers in rehabilitation can be summed up as: reducing...
pain, reducing inflammation and promoting healing. The benefits of laser therapy for rehabilitation patients focus on two main areas: increasing healing times through faster, stronger tissue repair, and the analgesic effect of the laser. These combine to allow the patient as a whole to feel better and lead to the ability to exercise efficiently (when indicated), to gain strength, and to return to function with an improved quality of life.

**Conditions treated with laser**
Any area that is inflamed or painful can benefit from laser (aside from pregnancy or neoplasia).

**Dermal**
Open wounds, post-surgical wounds, otitis externa and lick granulomas are all conditions that could be treated with laser in a veterinary setting.

**Musculoskeletal**
Treatment with laser therapy may be implemented as part of a multimodal treatment program. The specific goals of laser therapy often include decreased pain, inflammation, and improved circulation. Arthritic joints should be thoroughly treated along the joint lines and surrounding area. Recommendations for arthritic joints have ranged from 4 J/cm² up to 30 J/cm², but more appropriate doses may be 8-10 J/cm². The apparatus may either be held directly over the area for the designated number of joules, or used in a sweeping fashion depending on the unit.

Biceps tenosynovitis, supraspinatus tendonitis, patellar tendonitis, and other in inflammatory conditions involving tendons may be treated with laser therapy. The length of the superficial aspect of the tendon should be treated along with the surrounding soft tissues. The biceps tendon communicates with the shoulder joint capsule; therefore, the shoulder joint capsule should be treated as well.

**Neurologic**
A variety of diseases can affect this system including congenital malformation, genetic diseases, metabolic conditions (diabetes mellitus, hypothyroidism), toxicities (botulism, tick paralysis), autoimmune and inflammatory diseases (acute polyradiculoneuropathy, myasthenia gravis), neoplasia, trauma (brachial plexus avulsion), and vascular conditions (feline aortic thromboembolism).

Additionally, traumatic peripheral nerve injuries can also be treated.

Injuries to the spinal cord include those affecting the brain and herniated intervertebral discs. Common conditions affecting the spinal cord include trauma, intervertebral disk disease (IVDD), fibrocartilageneous emboli (FCE), vascular events, cervical spondylomyelopathy (wobbler’s disease), lumbosacral stenosis, discospondylitis, spondylosis deformans, syringomyelia, degenerative myelopathy, and trauma. Trauma, IVDD, and FCE can have acute onset where the spinal cord may have better healing potential than from the more chronic conditions (lumbosacral stenosis, spondylosis deformans, degenerative myelopathy, etc.) Proper diagnosis will aid in determining the prognosis and course of treatment.

**Musculoskeletal evidence**
Osteoarthritis studies with laser therapy in people; a meta-analysis review was conducted on the efficacy of laser therapy on OA in people. Seven trials were included, with 184 patients randomized to laser, and 161 patients to placebo groups, using a variety of lasers and treatment protocols. Treatment duration ranged from 4 to 12 weeks. Pain was assessed in four trials. The pooled estimate of three trials showed no effect on pain measured using a scale, and two demonstrated very beneficial effects with laser. In another trial, with no scale-based pain outcome, significantly more patients reported pain relief (yes/no) with laser. One study found knee ROM was significantly increased. Other outcomes of joint tenderness and strength were not significant. Lower dosages of laser were found to be as effective as higher dosages for reducing pain and improving knee ROM. The authors concluded that for OA, the results are conflicting in different studies and may depend on the method of application and other features of laser application, including wavelength, treatment duration, dosage, and site of application over nerves instead of joints.

Most of the research on the effects of laser therapy on tendon and ligament conditions is done either in experimental models in laboratory animals such as rats or in people. More evidence from studies in dogs, cats, and other animals is needed. Historically, laser therapy has been recommended for tendon and ligament conditions but the clinical efficacy remained controversial. Recent research proves that laser therapy is appropriate for these types of injuries.

One study looked at seven people with bilateral Achilles tendinitis to see if laser therapy has an anti-inflammatory effect. In placebo versus laser treatment the PGE2 levels were reduced for 75-105 minutes after laser therapy, and pain pressure threshold values increased after laser therapy. The authors concluded that laser therapy reduces pain and inflammation in people with acute Achilles tendinitis. A recent review of 25 articles (13 in vitro and 12 animal studies) was done to evaluate the influence of laser therapy on bone healing. All animal studies showed improved bone healing in sites irradiated with laser. It was concluded that laser could accelerate bone healing in extraction sites, bone fracture defects, and distraction osteogenesis.

**Evidence for laser therapy in the neurological system**
A study by Morries *et al.* (2015) looked at using laser therapy to treat traumatic brain injuries. In ten patients with chronic traumatic brain injury given ten treatments over the course of 2 months, using an 810 or a 980 nm laser, symptoms of headache, sleep...
disturbance, cognition, mood dysregulation, anxiety, and irritability improved. Depression scales and a novel patient diary system specifically designed for this study monitored symptoms. They concluded that laser immunomodulates the response to brain damage.

A research group has studied two spinal cord injury models to show 810 nm laser therapy was effective for transected or contused rat spinal cords (Wu et al. 2009). Laser was applied transcutaneously at the lesion site immediately after injury and daily for 14 consecutive days. The daily dosage at the surface of the skin overlying the lesion was 1589 J/cm² (150 mW, 0.3 cm² spot area, 2997 seconds). Mini-ruby was used to label corticospinal tract axons, which were counted and measured from the lesion site distally. Functional recovery was assessed by footprint test for the hemisection model and open field test for the contusion model. The average length of axonal re-growth in the rats in the treated group with hemisection and contusion injuries was significantly longer than the comparable untreated control groups. The total axon number in the treated groups was significantly higher compared to the untreated groups for both injury models. For contusion model rats there was a significant functional recovery in the laser treated groups compared to control.

A pilot study (Rochkind et al. 2007) investigated the effectiveness of 780 nm laser light in the treatment of patients suffering from incomplete peripheral nerve and brachial plexus injuries for 6 months up to several years. It was randomized, double blind, and placebo controlled using 18 patients. Twenty-one consecutive daily sessions of laser or placebo were applied transcutaneously for 3 hours to the injured peripheral nerve and for 2 hours to the corresponding segments of the spinal cord. Clinical and electrophysiological assessments were done at baseline, 21 days, and 3 and 6 months. The laser and placebo groups were in similar clinical conditions at the start of the study. The analysis of motor function during the 6 month follow up period compared to baseline showed significant improvement in the laser treated group compared to the placebo. No significant difference was found in sensory function. Electrophysiological analysis showed statistically significant improvement in recruitment of voluntary muscle activity in the laser treated group.

**Role of laser in injury rehabilitation**

Based on research, case reviews, and anecdotal reports, it can be concluded that it is indicated to use laser therapy in a clinical setting. A practical approach is also needed, accounting for many factors including patient need, patient cooperation, cost, efficacy, and frequency and duration of treatment. Some clients may allow their pet to be treated with the laser daily; some may only be able to so 2-3 times a week. It is critically important for the veterinarian to prescribe a practical and realistic treatment plan for each individual patient. Laser therapy represents only one element of comprehensive rehabilitation. While laser treatment can be safely and effectively combined with other modalities and integrated easily with other treatment approaches, it is important to prioritize the sequence of application with other modalities.

**Laser therapy as a stand-alone therapy**

Examples of diagnoses that could benefit from laser treatment as a sole treatment modality include surgical incisions, wounds, lick granulomas, osteoarthritis, and tail pull injuries. Application of the laser to every surgical incision at the end of the anesthetic period can reduce post-operative pain and swelling. This can be provided either bundled or as an option for all appropriate surgeries in a practice. Wounds can benefit from laser therapy in the late inflammatory or early proliferative phase, and laser therapy provides continued benefit in chronic or slow healing wounds as healing progresses. Lick granulomas can arise from many reasons and become cycles of healing and reoccurrence. Addressing pain relief, improved circulation, and antimicrobial pathways via laser therapy can provide improvement where other treatment modalities have either failed or only address a single potential cause of the granuloma.

Palliative management of chronic conditions can be achieved with laser. End stage otitis externa in cases that are not candidates for surgical resection will benefit from reducing bacterial load and reducing inflammation and pain. In some cases of osteoarthritis, the author has managed patients with stand-alone laser therapy. It is also a useful agent if having to discontinue other osteoarthritis management modalities. An example is the severely arthritic patient on an NSAID that develops renal, hepatic, or gastrointestinal disease, and NSAID therapy has to be rapidly discontinued.

The therapeutic laser can be used to stimulate acupuncture points along meridians for those clinicians who practice traditional Chinese veterinary medicine. The author has found this helpful for anxious patients that may not sit for 15-20 minutes or more with acupuncture needles in place.

Neurological patients often need a comprehensive rehabilitation program that includes laser therapy to achieve favorable results. But, tail pull injuries provide a straight forward injury where laser treatment can be the only modality that is available, practical, and easy to achieve. Additionally, patients suffering chronic pain from tail docking will also benefit from laser therapy.

**Laser therapy in conjunction with a rehabilitation program**

Proper rehabilitation starts with pain management. Regardless of the injury, no patient will be able to return to function through strength training, and maintain that outcome, if they are painful. Laser therapy is, in the author’s opinion, an extremely valuable modality to achieve the goals of the clinician, the client, and the patient. The release of endogenous opioids stimulated by laser therapy has applications throughout both acute and chronic conditions in injury rehabilitation (Hagiwara et al. 2008). Applying laser therapy
to painful muscles, tendons, ligaments, or joints before (and sometimes after) having the patient do therapeutic exercises, such as underwater treadmill workouts, cavaletti rails, or other core exercises, makes sense.

It is important for the clinician to incorporate laser therapy as one part of a complete rehabilitation program. An example is incorporating laser therapy during the first few weeks of rehabilitation for a dog with biceps tendonitis. This aids in reducing pain and stimulating cytokines and growth factors to achieve better tendon tissue healing. Laser therapy may be phased out of the rehabilitation program once initial goals have been achieved, and the patient progresses to strength training and maintenance.

Osteoarthritis may initially require a high frequency of treatment (multiple times per week) through an induction phase. Then, as pain and inflammation are reduced, the frequency of treatments can be reduced through a transition phase, until a maintenance phase protocol is achieved. If an acute on chronic flare up occurs, the induction phase can be easily repeated keeping the patient functional.

Neurological patients, such as those with brachial plexus avulsion or intervertebral disc disease, may benefit from laser therapy throughout their entire rehabilitation process. Early it may be used for management of pain at the insult site or in inflamed muscles. It can be used to attempt to achieve return of neurological function throughout the recovery, and can be used either to prevent or manage neuropathic pain that is a potential outcome in these cases.

**Reassessing response to laser therapy**

Based on the patient, diagnosis, prognosis, and plan of each case, reassessments should be scheduled and documented appropriately by the clinician. Veterinary professionals delivering the treatment should be looking for both subjective and objective indicators of how the patient is doing. Repeated physical examinations and photographs can be helpful in assessment of patient function. Additionally, goniometry, digital thermography, and stance analyzer measurements can be useful for providing objective data in a clinical setting.

**Conclusion**

Veterinary rehabilitation is an expanding field that requires both a practical approach and staying on the cutting edge of available treatment options. While injuries can occur in many body systems and from many different causes, laser therapy is a vital tool in the veterinary professional’s armament. Its variability, ease of administration, and proven benefits allow many types of patients to recover faster and more effectively. Further research is needed to find other types of injuries that may benefit from laser therapy, as well as further verifying treatment settings and timeframes.

**Selected references**


Therapeutic exercises are the real “meat and potatoes” (or tofu and potatoes for you veggie folks) of veterinary rehabilitation. They consist of a variety of focused exercises that are intended to mimic real life work. Consider this to be the occupational therapy aspect of vet rehab.

The goals of these exercises are:

- Improve active pain free ROM
- Improve muscle mass and strength, balance, performance of daily function, aerobic capacity, prevent further injury.
- Reduce weight (when indicated) and lameness
- Important method to best return of function

Remember that prior to starting any exercise, signalment, history and a complete physical examination (including assessment and plan) MUST be done. Certain patients will have limitations that you need to be aware of. This is not a “cookie cutter” approach.

You can be creative in mimicking activities, for example, if you have agility equipment available, then setting up an easier course and having the dog WALK through that is building up exercise. Cavaletti rails, leashes, weave poles, balance boards are just some of the tools that can be used for ther ex.

Remember to use your environment as well.

- Stairs (traction, not scary)
- Couches (cushions)
- Air Mattresses
- Work outside (hills, sand dunes, tall grass, snow)
- Owner limitations? Have them sit at the table

I start with introducing myself to the patient, and not being in a rush. I find it easiest to start with the “down” patient (whether from orthopedic or neurologic issues) and progress through these exercises to the fully ambulatory patient that is need of fine tuning.

For those down patients remember the fundamental aspects of down dog care (clean, dry bedding, changing recumbency, etc.) For completely down dogs we start with assisted standing exercises. Goals here are to: strengthen the patient, aid in proprioception, improve circulation and respiration, give them a chance to eliminate, are good for their mental well-being. Maximal assistance is needed to provide support 75-100% of the patient’s body weight. They cannot independently stand, and require a team effort. Place feet appropriately and use a sling, towel, Help Em Up Harness to achieve a “normal” position. Adjust for tolerance, but start with 20-30 seconds, per stand, 15 reps per set, 2-4x a day. Slowly increase the standing from 30 second to 5 minutes per session, pending how the patient improves.

Active assisted standing exercises follow, they get stronger let them do more, requiring <75% effort from us. Just enough support to maintain standing, physiorolls great for this, as are carts, hoists, etc.

Standby assisted standing exercises follow. Now has strength and motor to support against gravity, but are still ataxic or weak. You are right by their side, only there to prevent a fall. Once they can achieve rising and holding upright on their own, they may be ready for ambulation. Remember that during ALL of the phases of standing exercises you are doing proprioceptive training. That means that EVERY standing exercise the feet are placed appropriately, providing sensory feedback to the CNS.

For those patients that did not lose ambulation, this is where most of them come in. Proprioceptive training starts with the patient standing independently – time to do it right. Exercises here include 1) weight shifting, 2) unloading of a limb, 3) balance board and 4) exercise balls and rolls. Sit to stand and sit to down to stand exercises with good form are a great home exercise at this time. The patient is actively participating in their recovery.

Dynamic ambulation (aka WALKING) comes next. All walks must be on leash, with adult supervision. The pace must be dictated by the patient, but the handler must encourage the pace (not out smelling the roses, think power-walking). The handler may need to adjust their stride, we want walking, not running. This means for small breed dogs that people must walk extremely slowly, allowing the patient to use all four legs. Otherwise they will run o catch up, and not weight bear and strength train on the limb (think about your FHO patients).

As the patient improves, variety can be taken with different exercises, based on the patient’s needs and goals. This can include:

- Egg-crates, Foam Rubber, Air Mattress
- Couch cushions (shifting balance)
- Stairs – 5-7 steps, 2-4x a day for starters (on leash)
- Pole weaving, tunnels, pulling weight
- Ankle weights
• Syringe cap (on contralateral foot)
• Cavaletti rails

Start with 3-4 simple exercises per session, and always introduce it to the patient, then the owner, before having the owner do it at home. If the patient is improving, increase EITHER the time to a particular exercise by 10-20% each week.

If they are painful? Pause, address, start back up slowly. It may be related to their surgery or be a consequence from their lack of ambulation for a prolonged period of time. Objective outcomes are key, so re-measure – girth, stance analyzer, goniometer. Smart phone apps can be used by owners to track how far/fast they are walking the dog.

A practical, multimodal approach to therapeutic exercises will result in a better patient.

Selected references
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The rise in popularity of brachycephalic dogs and obesity in our pets have led to more airway surgeries needed for pets to be able to breath without overheating or ending in a collapsed larynx. Typically airway surgery for BOAS is reserved for 6 months of age or older, due to the changes in facial structure that can happen as they grow. However, if an animal is in distress at a younger age, surgery can be performed, but may need to be touched up as the animal matured and develops more folds or the skull confirmation changes.

The tracheal size should be assessed with a lateral cervical and chest film before any sedation or anesthesia is planned. A trachea the size of a cocktail straw will not be enough for a puppy to recover from a spay and should be discovered prior to sedatives. While brachycephalic dogs have small tracheas, the collapsing lumen or minute size will require referral for the safety of the patient.

The nares are not simply a cosmetic issues. Stenotic nares increase to airway pressure leasing to everted tonsils and saccules. Correction can be performed in several ways but requires not just alar skin resection but nasal cartilage as well. I prefer a deep vertical wedge resection. This is a deep pyramid piece taken out but leaving the inner curve of the nose and includes sinking the scalpel blade into the cartilage. The site is closed with absorbable suture cut short, to be allowed to fall out naturally. Qtips make this much easier to see. Matching the two sides are often the most difficult part. There is an art to making a pretty nose and avoiding the white scar tissue line that can happen with misaligned tissues.

The soft palate can be resected with laser, harmonic scalpel, ligasure and the traditional cut and sew technique. I do not advocate for laser, as this causes insufflation and swelling of the palate, unlike the other options. The palate should always be checked repeated or even marked with a surgery marker to assure the proper amount is removed. The soft palate should reach the caudal edge of the tonsillar crypt and lightly touch the tip of the epiglottis. The palate in BOAS dogs can be 0.5 to more than 2cm long. However, removing too much palate will result in aspiration pneumonia and chronic lung issues. If you do not remove enough, the animal will continue to have breathing and snoring issues. Judging the correct amount to remove is difficult as the jaw it opened widely, the tongue retracted or pulled for better visualization. This makes it more likely to accidentally remove too much. 0.25mg/kg Dexamethozone is given preop or intraop to help with oral swelling. This does can be repeated if the swelling is severe. If the breathing during recovery is too difficult, the patient may need to be reintubated until the swelling can subside.

Tonsils should be removed if they are inflamed and everted. They can be removed easily but starting a suture line at the caudal crypt, excising the tonsil and continuing the suture line to close the crypt with a simple continuous suture pattern with an absorbable suture. Once the crypt is closed the small tonsillar artery is controlled with tamponade pressure. The tonsils can also be removed by the previously mentioned surgical hemostatic instruments. Cautery should not be used in the oral cavity.

The saccules are out pouches that occur by the vocal folds and can occlude the ventral airway. The patient must be extubated for the saccular to be visualized and removed. They are cut out with Metzenbaum scissors with care not to damage the vocal folds or ventral mucosa. Qtips are used to remove any blood clots and the patient is reintubated for recovery. 0.25mg/kg Dexamethozone is given preop or intraop to help with oral swelling.
Wounds are classified as clean, clean-contaminated, contaminate or dirty/infected. Clean wounds are surgically created wounds that do not open a hollow viscous organ such as the respiratory, gastrointestinal or urogenital tracts. These surgical procedures must have maintained an aseptic technique throughout and no infection is encountered. Clean-contaminated wounds are surgical wounds that either enter a hollow viscus or there is a minor break in technique. Contaminated wounds are traumatic wounds or an operative wound with a major break in technique or a hollow viscus is opened with gross spillage. Dirty or infected wounds contain pus, contents of a perforated hollow viscus and have >10^5 organisms per gram of tissue.

The historic perception of a “Golden Rule” or a preferred time from injury to closure is not accurate and should not be used when making clinical decisions. The important thing to use when judging a wound is not the time period but the number of bacteria. This takes into account the severity of contamination, tissue injury and circulatory compromise.

Surgical wounds have a certain level of infection with aseptic technique trying to minimize the incidence of these infections. It is important to remember that the risk of infection doubles with each hour of surgery. Sources of this infection include the operating room, the operating team, surgical instruments, but most commonly the patient’s endogenous flora. The incidence of surgical wound infections are as follows; clean procedures 0-4.4%, clean-contaminated 4.5-9.5%, contaminated 5.8-28.6%, dirty implies infection.

Some specific points to mention include that bite wounds are not clean wounds and often have underlying muscular or vascular injury. Physiologic degloving is not uncommon underneath the traumatized skin. Anatomic degloving injuries are more common on extremities and are often combined with crushing injuries. Sinus or fistulous tracts are chronic walled-off infections that require exploration. An attempt should be made to probe the depth steriley. But the entire tract must be dissected to prevent recurrence. Gunshot wounds have variable amount of trauma that are proportional to the caliber and velocity. Meaning there is greater cavitation as velocity increases. There is skin, hair and debris in the wound making is contaminated, but the bullet itself is sterile and does not require removal in most cases. Thermal burns are classified by depth and the percent of the body involved. These patients are prone to secondary infection and metabolic disturbances. Pain control is paramount in burns as well as any wounded patient.

Skin tension lines are very important when performing a primary or delayed primary or secondary closure of a wound.

Incisions should be made parallel to these lines whenever possible. Proper technique for underlining skin is vital to assure retention of an adequate blood supply. Also, walking sutures, and releasing incisions should be staggered and spaced to allow for lateral blood supply to the primary incision or wound.

Stenting techniques utilizing horizontal or vertical mattress sutures can be used to relief tension off a primary incision closure. Alternatively an adjustable horizontal mattress pattern can be used to gradually close a distal wound.

Several techniques for regional skin movement can also be used including V-to-Y plasty or Z-plasty are extremely handy and fairly ways to move skin. The angle of the Z-plasty arms should be sixty degrees to optimize tissue lengthening, but will still only achieve a 75% growth. Also the central arm of the ‘Z’ should point in the direction of tension, or similarly be perpendicular to the wound you are trying to close.

Several other case examples will be discussed in lecture including suturing methods to close irregularly shaped wounds, and dog-ears. Local flaps and skin grafts may also be discussed.
When an acute respiratory crisis appears, oxygen may not be enough to solve the issue. Often times anesthesia with control of the airway is required. When a tracheostomy, either temporary or permanent is needed, several issues should be addressed. Temporary trach tubes can be used for severe airway swelling or trauma. Once the underlying obstruction has been relieved, the tube can be removed, and the site left to either close on its own or be surgically closed. Temporary anesthesia trach tubes are more comfortable and better able to heal than pharyngostomy tubes. Usually a small approach on the ventral midline to the trachea is used and a transverse or parallel incision to the cartilage rings is made in the upper half of the trachea. Stay sutures help replacement if the tube is accidentally dislodged early. The trach tube with or without a cuff (cuff required for anesthesia) is place and the area is otherwise bandaged. More permanent tracheostomy options involve creating a “U” or “O” or “H” opening into the trachea and suturing the skin to the tracheal rings. A trach tube will also be used but will not have a cuff. Keeping the site clean, open and unplugged from the increase in mucous secretions make these cumbersome for most patients and owner. Permanent trach tubes are used for upper airway obstruction that cannot be relieved, like laryngeal collapse.

Laryngeal paralysis
Laryngeal paralysis, failure of arytenoid cartilage, and vocal fold abduction is commonly seen in older medium to large breed dogs. It results from damage to the vagus nerve, its branches, or the intrinsic muscles of the larynx. Laryngeal paralysis is reported in dogs and cats and can be unilateral or bilateral with male dogs being 2-3 times more affected than females. The cause of paralysis may be idiopathic, congenital, traumatic, neoplastic, or iatrogenic. Recent evidence strongly suggests that dogs with idiopathic laryngeal paralysis may have an underlying chronic progressive polyneuropathy.1 Congenital laryngeal paralysis is most commonly reported in Dalmatian, Siberian husky, Bouviers de Flandres, and bull terriers. Clinical signs with the congenital form usually presents before one year of age. Acquired laryngeal paralysis is most commonly reported in Labradors and Golden Retrievers, Saint Bernards, and Irish Setters. The median age of affected animals is 9 years old. The acquired form has been associated with chronic endocrine, infectious, or immune mediated polyneuropathy.

Clinical signs and diagnosis
Presenting signs for congenital and acquired form are similar and may be acute or chronic. Clinical signs include exercise intolerance, inspiratory stridor, inappropriate inspiratory effect, loss or change in phonation, coughing (mainly after eating or drinking), cyanosis, and collapse. These signs worsen with obesity, exercise, excitement, and high temperatures.2 In cats, the most common clinical sign is tachypnea or dyspnea. Concurrent disease is common in old cats and dogs with acquired laryngeal paralysis.

Thoracic radiographs are performed to rule out other causes of dyspnea, detect underlying etiologies, and look for concurrent pathology such as aspiration pneumonia, non-cardiogenic pulmonary edema, or megaesophagus. Megaesophagus may be present in dogs with laryngeal paralysis, especially if the condition is secondary to polyneuropathy.1

Laryngeal paralysis is diagnosed by performing a laryngeal examination under a light plane of anesthesia. The animal should be anesthetized to the point at which the mouth can be easily opened but a laryngeal reflex is still present. In normal animals, arytenoid cartilages and vocal folds should abduct during inspiration and passively relax during expiration. In animals with laryngeal paralysis, arytenoid cartilages and vocal folds are immobile or drawn toward midline during inspiration. If only one arytenoid abducts, paralysis is unilateral. Edema and erythema of the arytenoid cartilage mucosa are typically present on the dorsal part of the larynx.

Surgical treatment
Surgery is the treatment of choice for animals with moderate or severe clinical signs or a decreased quality of life. The purpose of laryngeal surgery is to decrease airway resistance by removing, repositioning, stabilizing, or bypassing laryngeal cartilages that obstruct the rima glottis during inspiration. Surgical options include unilateral arytenoid cartilage lateralization, transoral partial laryngectomy, ventral laryngotomy for partial laryngectomy, and permanent tracheostomy.

Unilateral arytenoid cartilage lateralization is the considered the standard technique due to its consistent results. Unilateral cricoarytenoid lateralization involves placing suture around the caudal border of the cricoid cartilage and the muscular process of the arytenoid cartilage. Sutures are tied to a point that results in further arytenoid abduction to the final glottis opening will be no larger than that of the animal with the endotracheal tube in place.1 The animal must be extubated and reintubated for observation. The thyroarytenoid lateralization with or without cricoarytenoid lateralization has also been described. The purpose of the thyroarytenoid suture is to achieve lateral displacement of the arytenoid without caudal displacement. In one study, the rima
glottis area was significantly greater after cricoarytenoid lateralization (207%) than thyroarytenoid lateralization (140%) but there was no difference clinically.4

Transoral partial laryngectomy includes unilateral or bilateral vocal cord removal and resection of the corniculate and vocal processes of the arytenoid cartilage. The goal is to resect enough tissue to provide a functional airway without significantly affecting laryngeal function. The procedure is limited to one side of the larynx because this surgery provides an adequate airway opening and minimizes the risk of aspiration and scar tissue webbing across the glottis.3 Partial laryngectomy can also be approached through a ventral laryngotomy. The ventral approach provides better exposure and more operative space and permits primary mucosal closure.

Permanent tracheostomy bypasses upper airway obstruction without modifying the size of the rima glottis. This procedure is most valuable for dogs at high risk for aspiration pneumonia from myopathy, megaesophagus, gastrointestinal disorders, or other conditions.

Complications and prognosis
The long term outcome after surgical treatment is generally good, but patients are at risk for aspiration pneumonia their entire lives. Success rates for unilateral arytenoid lateralization is 90% and partial laryngectomy is 83-90%. Complications include aspiration pneumonia, persistent cough, vomiting, increased respiratory stridor, and exercise intolerance, surgical failure from suture breakage or arytenoid cartilage fragmentation, and laryngeal webbing.1,3,4 Surgery reduces and resolves respiratory signs in most animals, but does not address the underlying cause. In one study, 10 of 32 dogs with idiopathic laryngeal paralysis had generalized neurologic signs at the time of presentation and all dogs developed neurologic signs more than one year after surgery.2 This suggests that idiopathic laryngeal paralysis is a progressive generalized polyneuropathy.

References
The small and delicate structures of the urinary system make proper surgical techniques and instruments more critical. If something goes wrong, the complications can involved infection, urine leakage, hemorrhage and potentially urinary blockage. Have the proper absorbable sutures, surgical loops with a proper focal length and strength, and atraumatic forceps are just the starting point to helping you to success. Other items to consider on your short list would be:

- Sterile q-tips
- Several sizes of red rubber and tomcat catheters
- Foley catheters
- Bladder spoon
- Tru-cut biopsy
- Baby Metzenbaum scissors
- Lots and lots of stay sutures
- Patient warming devices and warm lavage
- Culturettes
- Malleable retractors
- Balfour retractors
- Suction

Lavaging the penis or vulvar vault before surgery should be performed with betadine. Chlorhexidine should not be used on mucous membranes due to excessive irritation. Male and female genitalia should be included in the draped area to assure lavage, lack of obstruction and ease in potentially placing postoperative urinary catheters. Due to obesity, often towels must be placed under females or the table placed flat (versus a “V”) position to allow ease of drapes placed below the vulva but above the anus. In males the red rubber catheter should be passed both normograde and retrograde in the cases of obstruction and stones. But when the penis is not being used for catheter passage, it should be clamped slightly to the side of the surgery to avoid touching suture, drapes or instruments to the prepuce. Also when the male abdomen is closed, the preputial muscle should be reattached laterally and cranially to avoid an abnormal penile orientation postoperatively.

If a kidney it to be removed, remember the right kidney can have more than one artery. Also the kidney is best removed for neoplastic reasons, by elevated it towards midline and dissecting along the dorsal area. The retroperineal surface will have less adhesions and this maneuver will also prevent intestines from blocking your vessel isolation and ligation.
Help! I’m Scared of Surgery!
How to Move Past the Fear and Get Back on the Horse
Sarah Wooten, DVM
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One of the most common procedures performed in veterinary medicine, an ovariohysterectomy, is arguably one of the most difficult to perform. Ask any veterinarian about the horror of overweight adult Labrador spays and he or she will tell you a tale of woe. Add in the pressure to perform these surgeries quickly with fear of failure or fear of being sued, plus the chronic stress that many vets are already under, plus the performance driven, perfectionistic personalities, seen in veterinarians, and you have a ticking time bomb of fear, panic, and anxiety that is easily detonated by adverse surgical events that are not managed mindfully. If a surgeon is not properly trained to manage his or her internal stress response due to adverse surgical events, then a vicious negative feedback loop of autonomic stress responses combined with conscious negative thoughts reinforces the fear and can devastate careers and mental health.

Objectives
This highly interactive session is designed to help attendees suffering with anxiety, panic, or fear of surgery due to a past traumatic event understand they are not alone (the speaker has her own story of surgical trauma!), understand the fear, understand what is going on in their body and mind that reinforces the fear/stress response, process the fear, and learn techniques to move past the fear and develop a healthy mind-body connection that will free them from past traumas and if they want to, allow them to practice surgery again without FEAR. Attendees will also be inspired to learn why it is important to their personal health to process surgical fear - studies from the field of psychology and quantum theory will be discussed to give attendees broadly applicable tools they need to overcome fear of surgery, and the hope is that attendees will learn tools and techniques that will improve their professional and personal lives.
Common Psychiatric Pharmaceutical Poisonings
Renee Schmid, DVM
Pet Poison Hotline
Scribner, NE

Nearly half of the exposures managed by Pet Poison Helpline are to human drugs. Here we will discuss the most common prescription psychiatric exposures: Anti-depressants, ADHD treatments, and more. For each of these, we will review clinical signs of toxicity, diagnostics, and treatment.

Amphetamines and methylphenidate
Prescription stimulants are commonly prescribed for treatment of attention deficit disorder in children and adults, narcolepsy, and sometimes weight loss. Common amphetamine drug brands include Adderall®, Dexamphetamine®, Desoxyn®, Dyanavel®, Dextrostat®, Evekeo®, and Vyvanse®. Methylphenidate (Ritalin® and Concerta®) and dexamphetamine (Focalin®) are also commonly prescribed CNS stimulants. Novel formulations of these drugs are appearing on the market. Adzenys® XR-ODT is an extended release amphetamine in an orally disintegrating tablet designed to make administration easier for children or other people who have difficulty swallowing pills, and Daytrana® is a transdermal patch designed to deliver methylphenidate throughout the day. Fruit-flavored chews and solutions are also available. These stimulant drugs may come in immediate-release or an extended or sustained release formulation, as indicated by the letters XR, SR, ER, LA, or CD in the trade name.

While the range of toxicity varies amongst these drugs, clinical signs typically begin near a dosage of 1 mg/kg in dogs. Immediate-release drugs are rapidly absorbed, and clinical signs can develop 20-30 minutes after ingestion. Sustained-release products and transdermal patches (if swallowed whole) may result in a slower onset of action as well as a prolonged duration of clinical signs. Signs of intoxication involve CNS over-stimulation and excessive sympathomimetic effects such as agitation, vocalization, hyperactivity, hypertension, head bobbing, mydriasis, hyperthermia, tachycardia, tremors, and seizures.

Treatment is primarily symptomatic and supportive. Emesis should only be performed on asymptomatic animals and needs to be done promptly due to the rapid onset of clinical signs. Activated charcoal may be helpful, especially with sustained release products. Ingested patches may need to be retrieved by emesis, gastric lavage, or endoscopy. Maintaining control of agitation, hyperthermia, tachycardia, and tremors are key elements in these cases. Acetaminophen (0.05-0.1+ mg/kg IV, IM, SQ) or chlorpromazine (0.5-1 mg/kg IV or IM) can be successfully used to achieve sedation and can be additionally beneficial in reducing heart rate, temperature, and blood pressure in agitated patients. It is recommended to start at the low end of the dosage range for sedatives and increase as needed. Some animals may require larger dosages than are listed here. Additionally, serotonin syndrome may occur and can be treated with cyproheptadine 1.1 mg/kg in dogs or 2-4 mgs total per cat orally or crushed into a slurry and delivered rectally. Benzodiazepines are typically avoided as they tend to result in paradoxical increased CNS excitement in these patients. Other commonly used interventions include injectable methocarbamol for tremors, injectable beta-blockers for tachycardia refractory to sedation, and IV fluids. Prognosis is generally good with prompt and aggressive treatment, though prolonged care may be needed, especially in large overdoses of extended release drugs.

Guanfacine and clonidine
Guanfacine is a centrally active drug with alpha 2-adrenergic agonist properties used for treatment of ADHD. It is not a stimulant, and the mechanism of action in treating ADHD is unknown. It can be used alone for treatment or in combination with other stimulants. Common brand names for this drug are Intuniv®, an extended release formulation, and Tenex®, an immediate release form of the drug. Clonidine is another similar alpha 2-adrenergic agonist drug sometimes used off-label to treat ADHD, autism, and Tourette’s Syndrome. Both clonidine and guanfacine were originally used as antihypertensive agents, and clonidine is also sometimes prescribed as a sleep aid, especially for children with sleep disturbances associated with ADHD or the stimulants prescribed to treat ADHD. Clonidine is now being used in dogs for certain behavioral conditions including phobias and separation anxiety.

Overdose of guanfacine and clonidine can result in clinical signs including depression, sedation, ataxia, vomiting, bradycardia, hypotension, and potentially seizures and tremors. Signs can develop at low doses, and these drugs have a narrow margin of safety in pets. Signs are expected within 4 hours of exposure and can last 24-72 hours.

Asymptomatic patients may be induced to vomit and then given one dose of activated charcoal. In symptomatic patients, atipamezole, while not a true antidote, can be used to reverse signs of toxicity with these drugs and is typically dosed at 50 mcg/kg IM. Atipamezole will need to be re-dosed frequently as it typically lasts only 2-3 hours, while the effects of clonidine and guanfacine can have a duration of 24 hours or longer. IV fluids are warranted to maintain hydration, increase perfusion, and support blood pressure. Vital signs, especially heart rate and blood pressure, should be monitored frequently. If seizures occur, they can be treated with standard anticonvulsants.

Atomoxetine
Atomoxetine (Strattera®) is a non-stimulant SNRI (selective norepinephrine reuptake inhibitor) used as a second line treatment drug for ADHD. At low doses, signs of anorexia, sedation or agitation have been reported with potential for hypertension, tachycardia, and possibly tremors at higher doses. Signs usually develop within a few hours and can last 12-24 h. Cats and pets with liver disease are thought to be more sensitive to the effects of this drug.
With recent ingestion, induce emesis and then give one dose of activated charcoal. Treatment is symptomatic and supportive if signs develop with anti-emetics for nausea/vomiting, sedation for agitation, beta blockers if persistent tachycardia develops, and methocarbamol for tremors.

**SSRI antidepressants**

Prescription antidepressants drugs routinely rank amongst the most commonly prescribed medications in the US and are increasingly used in veterinary medicine for a variety of behavioral disorders, including separation anxiety, storm phobias, inappropriate urine marking, stereotypic behaviors, and psychogenic alopecia. Common SSRIs include fluoxetine, citalopram (Celexa®), escitalopram (Lexapro®), paroxetine (Paxil®), and sertraline (Zoloft®). These drugs may come as either an immediate release or extended form prepared.

Selective serotonin reuptake inhibitors block the reuptake of serotonin in the presynaptic membrane, which results in an increased concentration of serotonin in the CNS. The range of toxicity varies depending on the drug and species. Small overdoses of SSRIs typically result in sedation or agitation, hypersalivation, vomiting, mydriasis, and tremors. Larger overdoses may cause tremors, seizures, dystagmus, dysphoria, vocalization, agressive behavior, ataxia, and, bradycardia. As the degree of overdose increases, so does the risk for the development of serotonin syndrome, a toxidrome characterized by central nervous, autonomic, and neurobehavioral signs including agitation, vocalization, vomiting, diarrhea, muscle rigidity, increased reflexes, tremors, hyperthermia, hypertension, and transient blindness.

Treatment of SSRI overdoses is largely supportive and symptomatic. Appropriate decontamination and early emesis and activated charcoal is recommended if aspiration risk is low. Cyproheptadine, a serotonin antagonist, is useful in reducing the severity of signs, especially vocalization and dysphoria and is dosed at 1.1 mg/kg in dogs and 2-4 mg total dose per cat q 4-6 hours either orally or crushed into a slurry and delivered rectally. Agitation may be treated with acepromazine (0.05-0.2 mg/kg, IV, IM, or SQ PRN) or chlorpromazine (0.5-1 mg/kg, IV or IM PRN) starting at the low end of the dosage range and increasing as needed. Some animals may require larger dosages than are listed here. Benzodiazepines are typically avoided as they tend to result in paradoxical increased CNS excitement in these patients. Additional treatments include methocarbamol for tremors (55-220 mg/kg, IV slowly and to effect), IV fluids for thermal cooling and to maintain hydration and adequate perfusion, and beta-blockers (e.g., propranolol 0.02-0.06 mg/kg, slowly IV) for tachycardia and hypertension that is not corrected following appropriate sedation.

Overdoses of other antidepressants such as duloxetine (Cymbalta®), a SNRI, and venlafaxine (Effexor®), a bicyclic antidepressant, are clinically similar to SSRI overdoses. Cats seem particularly drawn to Effexor capsules and will readily ingest them. Treatment is similar to SSRI overdoses but more focus on sedation may be needed.

**Tricyclic antidepressants**

Tricyclic antidepressants are another class of antidepressants used in human medicine as well as veterinary medicine for separation anxiety, other behavior conditions, excessive grooming or feather plucking, urinary conditions, pruritus, and neuropathic pain. Common tricyclic antidepressants include amitriptyline, clomipramine (Clomicalm®), nortriptyline, and doxepin.

These drugs have a narrow margin of safety, and anticholinergic effects can develop with overdose. Signs of toxicity may include constipation, urine retention, mydriasis, sedation vs agitation, disorientation, ataxia, arrhythmias, tachycardia, hypertension, vomiting, serotonin signs, and seizures. Treatment is similar to SSRI overdoses with decontamination, IV fluids, sedation if agitation develops, cyproheptadine if serotonin syndrome develops, antiemetics for vomiting and nausea, methocarbamol for tremors, and anticonvulsants if seizures develop. Close monitoring of vital signs, especially cardiovascular monitoring, is warranted Many tricyclic antidepressants are fat soluble, so treatment with intravenous lipids may be helpful in cases of severe toxicity.

**Benzodiazepines and non-benzodiazepine sleep aids**

Benzodiazepines are commonly used as anxiolytics, anticonvulsants, muscle relaxants and sedatives/hypnotics. Non-benzodiazepine hypnotics are typically used as sleep aids in human medicine. Although the two groups have different pharmacological profiles, both exert their effects through the inhibitory neurotransmitter gamma- amino butyric acid (GABA) and have similar clinical effects and treatment regimens. Common benzodiazepines used in veterinary medicine include alprazolam (Xanax®), diazepam (Valium®), lorazepam (Ativan®), midazolam (Versed®), and zolazepam in combination with tiletamine as the dissociative agent (Telazol®). Other benzodiazepines used in human medicine include clonazepam (Klonopin®), oxazepam (Serax®), and temazepam (Restoril®). Common Non-benzodiazepine hypnotics include zolpidem (Ambien®), eszopiclone (Lunesta®), and zaleplon (Sonata®).

Both families of drugs have a relatively wide margin of safety, and fatality is unlikely to occur with acute overdose. Chronic oral use of diazepam in cats, however, can result in hepatic failure and should be avoided. Following ingestion, clinical signs of acute intoxication typically develop rapidly within 30-60 minutes and commonly include sedation vs paradoxical CNS stimulation (agitation), ataxia, confusion, and vomiting.

Treatment of acute ingestions consists of appropriate decontamination and supportive care. If necessary, the reversal agent or antidote, flumazenil, can be used but is rarely needed as these drugs are typically well tolerated. In symptomatic animals, monitor the body temperature and blood pressure and provide thermoregulation. IV crystalloids can be used as needed to maintain perfusion, treat hypotension, and correct dehydration. In cases of paradoxical stimulatory signs, additional benzodiazepines should not be administered as they will exacerbate the clinical signs. Instead, acepromazine (0.05-0.2 mg/kg IV, IM or SQ PRN) or butorphanol (0.1-0.5 mg/kg IV, IM, or SQ PRN) can be used. The reversal agent flumazenil (0.01 mg/kg, IV to effect PRN) is the antidote for benzodiazepine overdoses but is only necessary in rare cases of severe CNS or respiratory depression.
Lithium
Lithium carbonate and lithium citrate are used to treat bipolar disorder and as an adjunct to other antidepressants in humans, and its mechanism of action is not well understood. It has recently been tried as a treatment of anemia and neutropenia in dogs with bone marrow suppression, though with questionable efficacy. Lithium is a cation that competes with sodium, potassium, calcium, and magnesium at cellular sites, so animals with renal disease, dehydration, and sodium depletion can be more sensitive to this drug.

Acute overdoses of this drug are typically well tolerated with only mild vomiting, anorexia, and lethargy expected. Chronic overdose, which occurs rarely in pets, can be more serious, and signs may include lethargy, muscle rigidity, tremors, seizures, hypotension, arrhythmias, and bradycardia.

Emesis may be induced with recent ingestion. Activated charcoal is not effective at binding lithium. IV fluids can increase elimination of lithium, and 0.9% NaCl may be more effective at enhancing renal excretion. Treatment is otherwise symptomatic and supportive with antiemetics for vomiting, anticonvulsants for seizures, and methocarbamol for tremors and muscle rigidity.

Lamotrigine
Lamotrigine (Lamictal®) is a phenyltriazine anticonvulsant that is also used to treat bipolar disorder in humans. Overdose of this drug can result in vomiting, lethargy vs hyperactivity, ataxia, weakness, tremors, seizures, hypokalemia, and acidosis. Arrhythmias, hypotension, and rare hepatotoxicity are also possible. This drug is rapidly absorbed with onset of action in most cases within 4 hours, and signs can last 24-48 hours.

Treatment of acute ingestions consists of appropriate decontamination and supportive care. Intravenous fluid may aid elimination and is also used for hydration and perfusion. Close monitoring of heart rate, EKG, and blood pressure are warranted. Ventricular arrhythmias may be treated with lidocaine or procainamide if needed. Antiemetics are used as needed for vomiting, and diazepam and/or phenobarbital if seizure activity develops. Very depressed or comatose patients may need monitoring of respirations and blood gas, and some dogs require ventilatory support.

Antipsychotics
Antipsychotic drugs are used in human medicine to treat bipolar disorder, schizophrenia, and other psychiatric and neurologic conditions in humans. Common drugs in this class include olanzapine (Zyprexa®), risperidone (Risperdal®), aripiprazole (Abilify®), and ziprasidone (Geodon®). Signs of toxicity with these drugs include agitation or lethargy, hyperesthesia, vomiting, diarrhea, hypotension, tachycardia, ataxia, vocalization and aggression, serotonin syndrome, and arrhythmias. Olanzapine can cause fluctuations between sedation and agitation. Animal studies of risperidone have shown that induction of emesis with apomorphine can be inhibited by this drug and may not be productive. It is also important to note that the “discmelt” version of Abilify® contains xylitol.

These drugs are quickly absorbed with rapid onset of signs, and signs typically last approximately 24 hours in dogs. Treatment of acute ingestions consists of appropriate decontamination with emesis only in asymptomatic patients and activated charcoal only if low risk of aspiration. IV fluids are used for hydration and perfusion. Treatment is supportive with close monitoring of vital signs and blood pressure in symptomatic pets. Sedation is warranted in agitated pets, and if serotonin syndrome develops, cyproheptadine 1.1 mg/kg orally or rectally may be administered every 6-8 hours as needed. Tremors can be treated with methocarbamol and seizures with standard anticonvulsants.

Suggested reading
Peterson and Talcott Small Animal Toxicology 3rd edition.
Decontamination Methods of the Dog and Cat

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The goal of decontamination is to inhibit or minimize toxin absorption and to promote excretion or elimination from the body. It also allows us to remove or dilute topical irritants or corrosives. Consider the proper patient assessment as described above to determine if the benefits of decontamination outweigh the risk and whether the exposure will harm the patient. If decontamination is deemed to be warranted, selecting the appropriate method will help ensure successful management of the patient. Types of decontamination include:

**Ocular**
If an animal has been exposed to a chemical that is considered an irritant or corrosive to the eye, ocular decontamination is warranted. The pH will indicate if it is acidic or alkaline and there are often key words on the bottle such as caution or danger. As ocular decontamination should be started as soon as possible, owners should be encouraged to flush the eye at home with tap water or saline. Eye drops should be avoided. Once in the clinic, a labeled eye wash is ideal, followed by tap water. Saline has not been shown to be beneficial in cases of alkali ocular burns.

If the product is an irritant the eyes should be irrigated for 10-15 minutes at home and monitored for signs of irritation including redness, lacrimation, pawing or rubbing at the eye, squinting or edema. The eyes should be irrigated for 15-20 minutes at home if a corrosive. Corrosive products should also have an additional 15-20 minutes of irrigation performed by a veterinarian followed by a fluorescein stain, topical antibiotic ointment or drops and use of an Elizabethan collar.

**Dermal**
Dermal decontamination is indicated for exposure to corrosives or irritants, glues or adhesives, gasoline/hydrocarbons and systemically absorbed toxins. This will help prevent oral exposure by self-grooming, remove unwanted substances, minimize paresthesia and reduce the risk of burns.

Irritant products generally have a caution statement on the label and result in mild redness and irritation. Rinsing product off or bathing with a degreasing dish soap is generally effective treatment. Vitamin E oil may also provide relief in situations where paresthesia is present. Corrosive products are alkaline or acidic in nature and generally have a danger statement on the label. These products result in chemical burns to the skin. Rinsing product for 15 minutes and bathing with a degreasing dish soap 2-3 times will help to remove product. Burn/wound management should be used as needed.

Glues and adhesives are typically non-toxic. They adhere to the eyelid, teeth, skin and fur. Certain types can be loosened with oil. The affected fur can be clipped if the animal is bothered to help avoid self-mutilation. Otherwise, if the affected area is not problematic, no therapy is generally necessary, and the product should wear off with time.

Gasoline and hydrocarbons are typically not seriously toxic. They may cause defatting of the skin resulting in cracking, secondary infections and chemical irritant contact dermatitis. Bathing 2-3 times with a degreasing dish soap is generally adequate therapy. There is a small risk for aspiration if oral exposure occurs and if inhaled, CNS depression may develop.

Systemically absorbed toxins do not generally cause dermal damage, however, result in systemic signs. These include tea tree oil, topical pain creams, estrogen creams, pyrethrin products (cats), psoriasis cream and 5-FU. Bathing 2-3 times in a degreasing dish soap will help to minimize absorption depending on the timing since exposure.

**Inhalation**
Toxins that may require respiratory decontamination include concentrates or corrosives, including bleach and ammonia mixtures, as well as smoke inhalation and carbon monoxide. A simple yet important aspect of inhalation decontamination is to remove the animal from the source of exposure. For minor irritants, fresh air is generally sufficient treatment. Animals with underlying respiratory disease may require more intensive treatment. Oxygen therapy is often required for smoke inhalation, carbon monoxide, and cyanide toxicity.

Birds are very sensitive to inhalants, and fragrances, Teflon, and regular respiratory irritants may cause significant concern. The animal should be removed from the source, be given humidified oxygen, and offered heat support and fluids.

**Gastrointestinal**

**Emesis**
Emesis is by far the most common method of gastrointestinal decontamination. Approximately 49% (range of 9-75%) of stomach contents are recovered less than 30 minutes after ingestion. 17-62% is recovered 1 hour after ingestion. These ranges often make emesis success or failure difficult to assess, particularly when the ingestion is suspected but not known or when the number of items ingested is unknown as is often the case when an animal chews up a bottle of medication or eats a handful of raisins. If emesis is unproductive, it does not guarantee that the ingestion did not happen as emesis does not fully empty the stomach of its contents.

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In many cases, there is a window of opportunity of only 1-2 hours for a positive return on emesis. However, there are certain toxins that can have successful emesis for up to 6 hours post ingestion. These include grapes, raisins, chocolate, xylitol containing gum, bezoars, massive ingestions and drugs that decrease gastric emptying (opioids, salicylates, anticholinergics and tricyclic antidepressants).

Inducing emesis in dogs is performed by using apomorphine or 3% hydrogen peroxide. Apomorphine is a dopaminergic receptor agonist drug that stimulates dopamine-2 receptors in the CRTZ and can be given at 0.03mg/kg IV, 0.04mg/kg IM or by crushing ½ tablet for small dogs and 1 tablet for large dogs and placing in the conjunctival sac. Naloxone may be used if excessive sedation occurs without reversing vomiting. 3% hydrogen peroxide is given orally at a dose of 1-2ml/kg (1-2 tsp per 10 pounds). This should be fresh, bubbly and non-expired for effectiveness. Hydrogen peroxide is a gastric irritant and exceeding recommended amounts may result in gastritis with gastric bleeding.

Inducing emesis in cats is best performed by using an a-2 adrenergic receptor agonist drug such as xylazine at 0.44mg/kg IM or dexmedetomidine at 7mcg/kg IM or IV. The sedative effects can be reversed with yohimbine or atipamezole. These drugs are approx. 50% effective in cats, may cause excessive sedation and, in rare cases, cause cardiovascular collapse. This is not generally recommended for older or disease compromised cats. Cats are more sensitive to developing hemorrhagic gastritis with hydrogen peroxide and is often not effective, therefore, not recommended. Apomorphine is also not very effective in cats as the cat CRTZ is mediated by alpha receptors and not dopamine receptors. Apomorphine may cause dysphoria and agitation (“morphine mania”) in cats.

Products used in the past that should NOT be used to induce emesis include salt, syrup of ipecac, digital manipulation, liquid dish soap, raw eggs, Tabasco, or mustard. Salt toxicity, gastric irritation, nerve damage or aspiration may occur when other methods are used.

Emesis should not be induced in symptomatic animals, those that have already vomited to bile/clear, or those with a history of aspiration pneumonia or at risk for such due to laryngeal paralysis or megaesophagus. Examples of toxicity ingestions that should not have emesis induced include sharp/dangerous objects that may cause more trauma to the esophagus or enter the soft palate, corrosive agents (alkaline batteries, disc batteries, alkaline substances with a pH >11, acidic substances with a pH < 3) that may cause chemical burns to the esophagus and GIT, or hydrocarbons (gasoline, kerosene, motor oil) that present a moderate aspiration risk.

Caution should be taken if inducing emesis in brachycephalic breeds, young animals (less than 3 months of age), geriatric pets (greater than 10-12 years of age), animals with a history of heart disease, seizures, recent surgery or those that have a non-toxic ingestion. Species that do not vomit include rabbits, ruminants (sheep, cattle, llama, goat), horses, birds and several rodents including chinchillas, rats and gerbils. Other decontamination methods will be needed for these species.

**Gastric lavage**

Gastric lavage may or may not be more effective at removing gastric contents. Often the more forceful contractions of the gastric muscles during emesis are more effective at removing contents than passive flow from lavage. This is a viable option for those species that do not vomit, symptomatic patients with a large ingestion, a large amount of stomach contents or where emesis was unsuccessful. It also may be helpful with potentially fatal ingestions including calcium channel blockers, beta blockers, buprinofen, and metaldehyde.

Safe performance of gastric lavage requires sedation, intubation, and endotracheal insufflation. The animal should be in right lateral recumbency with the head tilted down at an approximately 20-degree angle. The stomach tube should be measured to the last rib, passed in to the stomach, and flushed with 5-10ml/kg warm water. The stomach should be agitated and then aspirated or allow for gravity to drain stomach contents. Once adequate removal of stomach contents is achieved, activated charcoal can be given. Caution should be used, however, as it is not uncommon for regurgitation to occur and the risk of aspiration is high. If activated charcoal is given, an anti-emetic, head elevation and continued intubation for as long as animal will tolerate until they can protect their airway should also be done.

Risks that are associated with gastric lavage include aspiration pneumonia, the need for general anesthesia, esophageal or gastric rupture and electrolyte imbalances. There are numerous contraindications to performing gastric lavage. These include hydrocarbon ingestions due to the high aspiration risk, corrosives, recent surgery (pending location), unstable patients, and those at a risk for bleeding or injury.

**Activated charcoal and cathartics**

Activated charcoal binds to many toxins in the GI tract by physical contact and weak covalent forces. Charcoal is a black powder composed of partially decomposed cellulose of soft wood. Activated charcoal is produced by heating common charcoal in the presence of a gas which creates numerous internal pores to trap chemicals within the activated charcoal. This process results in a highly porous material with an enormous surface area relative to its weight. The adsorptive capacity of activated charcoal is a function of its binding surface area. There is limited data regarding the benefit of activated charcoal with many toxins and one must weigh the risk vs. benefit when considering its use.

Benefits of activated charcoal include that it is readily available, relatively inexpensive, decreased absorption of 25-30% when administration is delayed, and beneficial use a wide number of toxins. Activated charcoal can be given with food to aid in
administration without decreasing effectiveness. Drawbacks of activated charcoal use include difficulty of administration, potential vomiting after administration, potential diarrhea, binding to therapeutic medications, the unknown benefit with many toxins, and most importantly, the risk of hypernatremia. Hypernatremia may occur with any dose of activated charcoal, with an increasing risk as the number of doses increase.

There are numerous situations where activated charcoal use should be avoided based on the status of the animal. These include animals that are symptomatic, particularly with neurologic signs as aspiration risks are increased, animals with dehydration, current hyperthermia, hypovolemic shock, decreased GI motility/ileus, recent GI surgery and protracted vomiting. Activated charcoal should be avoided in instances where the risk of aspiration pneumonia is higher, including an unprotected airway, decreased level of consciousness, excessive sedation or agitation and when having to force feed. Activated charcoal should also be avoided in situations where endoscopy or abdominal surgery of the GI tract may be needed, concerns of a gastric or intestinal obstruction and ingestions where there is an increased risk of aspiration pneumonia, such as with caustic substances and hydrocarbons. Contraindications to activated charcoal use in general include exposures that occurred > 2 hours after ingestion unless enterohepatic recirculation occurs, or extended release formula medication was ingested, alcohols (ethanol and ethylene glycol), xylitol, heavy metals, salt, paintball and non-toxic ingestions.

Cathartics are helpful at gastrointestinal decontamination for numerous reasons including accelerated speed of drug transit through the GIT, decreased time for toxin absorption, and decreased time for desorption of toxin from the activated charcoal. Sorbitol, a hexahydric sugar alcohol, is frequently combined in activated charcoal formulations at a dose of 3ml/kg PO of a 70% solution. Magnesium based cathartics (magnesium hydroxide, magnesium oxide, magnesium sulfate) should be used with caution in cats due to their increased risk of serum and brain magnesium levels. Hypermagnesemia displayed as hypotonia, ECG changes, altered mental status and respiratory failure may occur. It is also recommended to avoid magnesium-based cathartics in bromethalin toxicities due to potential similar clinical signs if hypermagnesemia were to occur. Magnesium hydroxide is often used in cases of mild iron toxicity due to its ability to precipitate binding of iron in the GIT to insoluble iron hydroxide.

The standard dosing of activated charcoal is 1-2g/kg, with 1g/kg being ideal in most situations. A cathartic is recommended to be given with the initial dose to help increase the rate of intestinal evacuation. Repeated doses of activated charcoal without sorbitol is valid for products that undergo enterohepatic recirculation or medications that have extended release properties. Doses are typically repeated every 6-8 hours for up to 24 hours, depending on the toxin. Sodium levels should be monitored and IV or SQ fluids given to minimize the risk of hypernatremia.

**Whole bowel irrigation**

Whole bowel irrigation (WBI) is rarely used in veterinary medicine. Situations when WBI may be helpful include enteral coated medications, iron ingestion, sustained or extended release medications and ingestions of packets of medications. WBI is performed using a nasoesophageal or nasogastric tube and administering 25-40ml/kg PEG-ES solution orally followed by a continuous oral infusion of 0.5ml/kg per hour until there is radiographic clearance or clear feces are present. Contraindications for WBI are like that of activated charcoal administration.

**Endoscopy and surgical removal**

Endoscopy may be indicated for ingestions of objects in situations where emesis would not be safe either due to the object size/shape or risk of oral/esophageal injury, such as ingestions of coins, non-leaking batteries, patches (fentanyl, lidocaine), bottles/plastic and metals. Endoscopy may also be warranted in evaluating injury to the esophagus and stomach. Negative aspects to endoscopy include the status of the animal if symptomatic, cost, equipment access and the need for general anesthesia.

When an animal is unable to vomit or if an object is not able to be removed endoscopically, surgery may be necessary for a successful outcome. Examples of this include leaking batteries, bread dough, a large number of objects and medication bezoars. Occasionally, surgery is required for removal of substances that do not pose a toxicity concern, however, a foreign body/obstruction concern. Sharp objects and large foreign bodies may require surgical removal. Gorilla Glue® has expansive properties and while toxicity is not seen, can form a hard, rock-like substance that encompasses the diameter of the stomach.

**Suggested reading**


Peterson and Talcott Small Animal Toxicology 3rd edition.


Chocolate and caffeine supplements
Methylxanthines are comprised of caffeine and theobromine, the toxic component found in chocolate. Methylxanthines cause an increase in cAMP due to inhibition of cellular phosphodiesterase, stimulate the release of catecholamines from the adrenal medulla, cause competitive inhibition of adenosine and inhibition or calcium sequestration within the sarcoplasmic reticulum and an increase in calcium entry into cardiac and skeletal muscle cells. While caffeine is rapidly absorbed and reaches peak plasma levels within 30-60 minutes, theobromine is slowly absorbed in dogs and may take up to 10 hours before peak plasma levels are reached. The half-life between caffeine and theobromine is 4 hours and 17.5 hours, respectively. Caffeine toxicity will result in mild signs at 15mg/kg moderate at 25mg/kg and severe at 5mg/kg. Theobromine toxicity will result in mild signs at 20mg/kg, moderate at 40mg/kg and severe at 60mg/kg. Signs of toxicity include vomiting, diarrhea, agitation, hyperactivity, tremors, tachycardia, hypertension, arrhythmias and seizures. Hypokalemia may develop with caffeine toxicity. The LD50 or caffeine in dogs is 140mg/kg and 80-150mg/kg for cats. The LD50 for theobromine is 200-500mg/kg in dogs and 200mg/kg in cats. Treatment consists of symptomatic and supportive care. Potassium should be monitored in cases of caffeine toxicity. Due to enterohepatic recirculation and significant renal excretion, multiple doses of activated charcoal and IV fluids are useful components of therapy.

Mouse and rat poisons
Anticoagulants
Anticoagulant rodenticides are Vitamin K antagonists and interfere with the activation of Vitamin K dependent clotting factors (2, 7, 9 and 10). The coagulation system generally continues to function well until about 24 to 36 hours after ingestion when the natural decay of clotting factors occurs. Anticoagulant rodenticides are well absorbed orally, with peak plasma levels occurring within 12 hours. Warfarin, the most common short-acting anticoagulant has a half-life of 12-18 hours. Due to widespread resistance amongst rats, warfarin is rarely used as a rodenticide in the US and more commonly seen are long-acting anticoagulants (LAACs). LAACs have a much longer half-life of days to weeks. The margin of safety for anticoagulant rodenticides for many species varies based on the active ingredient. Cats are significantly more resistant to the effects of these products than most species, especially dogs, and rarely suffer poisoning. The most common signs of poisoning in dogs and cats are generalized abnormalities such as lethargy, exercise intolerance, inappetence, pallor, and dyspnea or wheezing. These signs typically begin 3-5 days following exposure. For acute exposures or asymptomatic animals, a baseline PT as well as a PT performed 48-72 hours post-ingestion may be performed in lieu of empirical antidotal treatment with vitamin K1. This allows enough time for the currently present clotting factors to decay but does not (typically) provide enough time for the onset of clinical signs.

Bromethalin
Bromethalin is a neurotoxic rodenticide which has experienced increasing commercial use in the USA since 2012. Ultimately, toxic effects are a result of white matter vacuolization in the CNS due to myelin edema. Clinical effects are dose dependent and include CNS depression, ataxia, tremors, seizures, paresis/paralysis, hyperthermia, abnormal PLRs, anisocoria, nystagmus, coma and more. The minimum lethal dose in cats is reported to be ~0.25 mg/kg rendering them much more sensitive than canines, whose minimum lethal dose ranges from 0.95-1.05mg/kg. The LD50 for cats is 0.54-1.8mg/kg and in dogs is 2.38-3.65mg/kg. In general, signs of toxicity are seen within 2-24 hours after ingestion, however, can be delayed by 2-5 days with ingestions that are below the LD50 but above 1/10 of the LD50. There is no antidote available for bromethalin, leaving aggressive decontamination crucial for successful outcomes. Treatment includes prompt GI decontamination, supportive and symptomatic care including mannitol for cerebral edema and methocarbamol for tremors. Intralipid emulsion has shown a positive response in many cases and is reserved for symptomatic patients.

Cholecalciferol
See Vitamin D3 discussion.

Acetaminophen
Acetaminophen (APAP), a cyclooxygenase (COX)-3 inhibitor, is a popular over-the-counter (OTC) analgesic and antipyretic medication used frequently in humans. It is not considered a true NSAID as it lacks anti-inflammatory properties. APAP is metabolized into non-toxic conjugates primarily via glucuronidation and sulfation, two metabolic pathways in which cats are relatively deficient. As a result, cats are more sensitive to APAP compared to dogs or people. The toxic dose of APAP in dogs is 100-150mg/kg and in cats is 10-60 mg/kg. In dogs, clinical signs may develop within a few hours with vomiting and up to 24-48 hours with hepatic damage or failure and higher doses causing a risk of methemoglobinemia. For cats, clinical signs may develop within hours of ingestion and typically include cyanosis, methemoglobinemia, lethargy, anorexia, respiratory distress, facial/paw edema,
hypothermia and vomiting. Icterus may also occur and is usually due to hemolysis. Hepatotoxicity can occur in cats but is not as common as with canines. Treatment includes prompt decontamination via the induction of emesis and administration of one dose of activated charcoal with a cathartic. Antidotal therapy with N-acetylcysteine provides a source of glutathione precursors. Intravenous dosing is recommended in cats due to potential poor oral bioavailability (Burr et al.), however, IV or oral routes in dogs can be used. Additional therapies such as SAMe can be helpful. In addition to hepatoprotectants, IV crystalloids should be provided for hemodynamic support. Packed red cells can be given if needed. Methylene blue can be used to treat methemoglobinemia but may also cause Heinz bodies so should be used cautiously in cats.

**Ibuprofen**

Ibuprofen, a non-selective COX-1 and COX-2 inhibitor, is one of the most common over-the-counter and prescribed NSAID. Its effective analgesic and anti-pyretic properties make it a popular medication for humans. Ibuprofen has complete absorption in dogs, is metabolized in the liver to inactive metabolites and is highly protein bound. Peak plasma levels are reached in 30 minutes – 3 hours. Ibuprofen has primarily renal excretion at 70%, with the remainder excreted in feces. The toxic dose of ibuprofen in dogs may be seen with doses as low as 8mg/kg, with GI signs (vomiting, inappetence) possible. A higher risk of GI ulceration occurs with doses >25mg/kg. ARF may be seen in adult, healthy dogs at 100mg/kg. CNS signs (lethargy, ataxia, tremors, coma) may occur with doses >400mg/kg and death is possible even with aggressive therapy for ingestions >600mg/kg. Young, geriatric or disease compromised dogs are potentially at risk for renal toxicity with doses as low as 50mg/kg. As with other NSAIDs, cats are more sensitive to toxicity, with toxic doses considered to be half that of dogs. Clinical signs include vomiting, inappetence, diarrhea, GI ulceration, pu/pd, dysuria and renal failure. Initial signs may be seen within 2 hours of ingestion, while ARF generally occurs within 24-48 hours, it may be delayed by several days. Treatment includes prompt decontamination via the induction of emesis and administration of one dose of activated charcoal with a cathartic. For ingestions at risk of GI ulceration only, GI protectants for 5-7 days should be initiated. Additional therapy is required for ingestions with a potential for renal toxicity. Ibuprofen undergoes enterohepatic circulation, making repeated doses of activated charcoal q 6-8 hours for the first 24 hours after ingestion beneficial for ingestions >100mg/kg. GI protectants should be initiated and continued for 7-10 days. Baseline labwork including pcv/tp, serum chemistry and u/a is recommended for ingestions at risk for renal toxicity. Starting IV crystalloids for 48 hours will help provide renal protection and hydration support. If renal values remain normal after 48 hours, fluids can be tapered and discontinued. Renal values, along with pcv/tp, should be evaluated q 24 hours for 48 hours and again 24 hours after discharge of patient.

**Xylitol**

Xylitol is a sweet, sugar alcohol naturally occurring in small amounts in many fruits and vegetables. Due to its sweet taste, low glycemic index, and dental plaque fighting properties, it is widely used in oral care products (e.g., chewing gum, toothpaste, oral rinses, etc.). Recently, xylitol has been found in an increasing array of prescription and OTC medications such as nasal sprays due to its cooling sensation, OTC sleep aids, multivitamins (especially gummy or chewable formulations), antacids, children’s liquid stool softeners, and smoking cessation gums. It is also present in prescription products such as sedatives, NSAIDs, anti-diabetic agents, etc. In addition to its flavor, xylitol is also a humectant (retains moisture) so is often added deodorants and lotions. Due to its use as a sweetener, xylitol is most likely to be found in medications formulated as chewable or gummy pills, oral solutions or suspensions, or orally disintegrating tablets. Determining the amount of xylitol in OTC and drug products can be difficult. Unlike food labels in the USA, which must list ingredients in descending order of predominance by weight, the regulations regarding the labeling of ingredients in drugs and dietary supplements in the USA are considerably different. Xylitol is often considered an “inactive” or “other” ingredient, neither of which is required to be listed in order of predominance. Often, inactive/other ingredients are listed in alphabetical order, which may result in a misinterpretation that there is a very low concentration of xylitol in the product as it appears as near the end of an ingredient list. To obtain the concentration of xylitol in a drug, contact the product manufacturer or an animal poison control center.

**Vitamin D3 products**

**Vitamin D3 supplements and cholecalciferol**

Vitamin D3 ingestions may result in severe hypercalcemia and hyperphosphatemia. Soft tissue mineralization including the renal tubules results in ARF development. Vitamin D3 is found in rodenticides and over the counter products and is available in much higher prescription strengths. The toxic dose for vitamin D3 is 0.1mg/kg with a potential likelihood for tissue mineralization with doses >0.5mg/kg. Signs often begin with vomiting, inappetence and lethargy. Within 2-3 days of ingestion, polyuria and polydipsia, more significant lethargy, weakness and continued vomiting are seen. Labwork typically shows hypercalcemia, hyperphosphatemia and azotemia. Decontamination includes emesis and multi-dose activated charcoal. Cholestyramine makes vitamin D bound bile acids insoluble and unable to be re-absorbed, so may be helpful. While there is successful data in humans, there is limited data for its use in animals. Treatment involves IV fluid therapy to promote calciuresis using 0.9%NaCl. In cases where hypercalcemia and hyperphosphatemia are evident in healthy adult dogs and cats, prednisone and/or furosemide may be needed to increase calciuresis. Caution should be taken in interpreting results for puppies and kittens, as their “normal” calcium and phosphorus values are often
higher due to bone development. Phosphate binders are used to aide in hyperphosphatemia therapy. Bisphosphonates such as pamidronate disodium are used to decrease calcium absorption. Serum calcium is expected to decrease within 24-48 hours. If no response is seen within 3-4 days, repeat dosing may be needed. Symptomatic animals often require weeks to months of laboratory monitoring and medication adjustment.

**Calcipotriene/calcipotriol**
Calcipotriene/calcipotriol is a synthetic vitamin D analogue commonly used for the treatment of human psoriasis. Intoxication from calcipotriene results in life-threatening hypercalcemia and hyperphosphatemia leading to metastatic mineralization and subsequent renal failure. Death from cardiac failure secondary to hypercalcemia may also occur but is less likely. The minimum acute toxic dosage in dogs is 10mcg/kg with an acute lethal dosage of 65mcg/kg. These values have not been reported in cats but is well established that they are more sensitive to the effects of calcipotriene than dogs are. Signs of toxicity, including electrolyte abnormalities, typically begin only a few hours after ingestion, PU/PD, anorexia, vomiting, lethargy, melena, frank bloody diarrhea, hematuria and death. While the renal system is often most acutely affected, eventually the cardiac, pulmonary, GI and CNS systems may be as well. Treatment is similar to that of vitamin D₃ toxicity.

**Antidepressant medications**

**Selective serotonin reuptake inhibitors (SSRIs)**
SSRIs block the reuptake of serotonin in the presynaptic membrane, which results in an increased concentration of serotonin in the CNS. Cats, as compared to dogs, are more sensitive to SSRIs. Small overdoses of SSRIs typically result in sedation or agitation, vomiting, mydriasis, tachycardia and hypertension. Larger overdoses may cause tremors, seizures, dysphoria, vocalization, aggressive behavior and ataxia. As the degree of overdose increases, so does the risk for the development of serotonin syndrome, characterized by central nervous, autonomic, and neurobehavioral signs. Signs may include muscle rigidity, increased reflexes, tremors, hyperthermia and hypertension. Treatment of SSRI overdoses is largely supportive and symptomatic; no specific antidote is available. Appropriate decontamination is recommended. Cyproheptadine, a serotonin antagonist, is useful in reducing the severity of signs, especially vocalization and dysphoria. Agitation may be treated with acemannone or chlorpromazine. For seizures, benzodiazepines may exacerbate neurologic signs, so barbiturates and levetiracetam can be considered instead. Additional treatments include methocarbamol for tremors, IV fluids for thermal cooling and to maintain adequate perfusion, beta-blockers for tachycardia and hypertension if this is not corrected following appropriate sedation.

**Other antidepressants**
Overdoses of other antidepressants such as duloxetine, a SSNRI, and venlafaxine, a bicyclic antidepressant, are clinically similar to SSRI overdoses. Due to their mechanism of action, these agents have an added element of increased presynaptic concentration of norepinephrine and dopamine (venlafaxine). This may lead to sympathomimetic signs such as mydriasis, tachycardia, hyperthermia, hypertension, etc. Treatment is similar to SSRI overdoses but more focus on sedation may be needed.

**Fertilizers**
In general, ready to use fertilizers have a low degree of toxicity. GI upset including emesis, diarrhea and possible inappetence are generally the limited signs seen. Exposure to industrial or agricultural fertilizers may result in corrosive injury if direct ingestion occurs. Once a product has been applied and either dried or watered in, toxicity concerns are minimal. Nitrogen concentrations are not generally a concern with fertilizer ingestions in dogs and cats. Small animals have deficient numbers of microorganisms to convert nitrates to nitrites.

**Blood meal and bone meal**
Blood meal is dried, ground and flash-frozen blood that contains a large amount of nitrogen. Ingestions generally result in acute emesis and diarrhea. Larger ingestions may result on pancreatitis as well. Bone meal is made up of defatted, dried and flash-frozen animal bones that are ground in to a powder. While toxicity is not expected, GI upset including emesis and diarrhea may occur. Large ingestions may cause a bezoar formation and FBO, though rare. Decontamination with emesis will help to avoid potential complications.

**Iron**
Iron is commonly added to fertilizers. With the exception of fertilizers with >5% iron concentration, most ingestions will result in GI upset. The amount of iron included is rarely listed in its elemental form and must be converted to elemental iron when calculating for potential toxicity. Most exposures result in vomiting, hematemesis, diarrhea and possibly hematochezia.

**Grapes and raisins**
Grapes, raisins, sultanas (pale, green grapes popular in Europe) and Vitus spp. currants (marketed as Zante currants) have been shown to cause ARF to develop. Ribus spp. currants are in the gooseberry family and do not cause renal toxicity. The toxicity risk to cats and ferrets is unknown. While the mechanism of toxicity is unknown, it is thought that there is individual susceptibility to metabolize components of the fruit or presence of salicylate-like chemicals in the fruit. Grape seed extract appears to be safe and not cause renal
injury. Wine, grape juice and raisin paste risks are unknown and are generally cautiously treated as potential toxins. There are many reports of a “toxic dose” for grape/raisin ingestions. Due to the unknown toxic component and the potential for individual sensitivity it is not recommended to rely on ingested doses to determine an animal’s toxicity risk. Signs of toxicity include vomiting and 24-48 hours after ingestion, signs progress to lethargy, continued vomiting, inappetence and possible abdominal pain. Azotemia may occur within 24 hours of ingestion. Decontamination is a key factor and emesis can be initiated up to 6 hours after ingestion followed by one dose of activated charcoal. Therapy includes labwork to monitor renal function, anti-emetics and IV fluids.

**Amphetamines**
Amphetamines are sympathomimetic compounds. Although structurally related to norepinephrine, amphetamines are more potent. They stimulate the release of norepinephrine from stores in adrenergic nerve terminals. They also directly stimulate alpha and beta adrenergic receptors. While the range of toxicity varies amongst these drugs, clinical signs typically begin at a dosage of 1 mg/kg in dogs. These drugs are rapidly absorbed and clinical signs often occur 20-30 minutes after ingestion. Sustained-release products and transdermal patches (if swallowed whole) may result in a slower onset of action as well as a prolonged duration of clinical signs. Signs of intoxication involve CNS over-stimulation and excessive sympathomimetic effects such as agitation, vocalization, hyperactivity, hypertension, head bobbing, mydriasis, hyperthermia, tachycardia, tremors, and seizures. Treatment is primarily symptomatic and supportive, and similar to that of SSRIs.

**Suggested reading**
Peterson and Talcott Small Animal Toxicology 3rd edition.
5-Fluorouracil or 5-FU

5-fluorouracil is an anti-neoplastic agent used topically, in human medicine, for treatment of skin cancers like actinic keratosis and superficial basal cell carcinomas. Common topical preparations have a 0.5-5% concentration, and brand names include Efudex®, Carac®, Adrucil®, and Fluoroplex®. It is also used parenterally in both human and veterinary medicine for GI, mammary, and ovarian cancers. Dogs chewing into tubes of the topical product represent the majority of pet intoxications. The minimum canine toxic dose is approximately 5 mg/kg (PO or IV). Experimentally, the minimum lethal dose is approximately 20 mg/kg with 40 mg/kg considered uniformly fatal, though dogs have survived 43, 46, and, shockingly, 330 mg/kg dosages. Cats are more sensitive than canines and just a couple of licks of topical preparations may prove toxic.

The onset of clinical signs is rapid with signs often developing within an hour of ingestion, and death can occur in less than 24 hours. The most common signs on presentation include: vomiting, grand-mal seizures, tremors, dyspnea, and cyanosis. Seizures are often refractory to diazepam. Additional acute signs include ataxia, depression, hypersalivation, diarrhea. Death occurring in the first 24 hours is usually a result of poorly controlled seizures leading to non-cardiogenic pulmonary edema with subsequent cardiopulmonary arrest. If the patient survives the initial phase of intoxication, then cytotoxic effects such as bone marrow suppression and GI sloughing due to the loss of intestinal crypt cells may develop and can also be life-threatening.

Treatment for 5-FU intoxication is primarily supportive as the antidote currently utilized in human medicine does not appear effective in dogs and is usually cost prohibitive. In many cases, GI decontamination is not feasible due to rapid absorption of the drug from the stomach and due to rapid onset of clinical signs. However, if the animal is asymptomatic, prompt GI decontamination is advised. Treatment includes anti-convulsant therapy, anti-emetic therapy, IV fluids to maintain perfusion to both the GI and CNS, temperature regulation, antibiotic therapy to help prevent sepsis from severe leukopenia or bacterial translocation, monitoring of baseline blood work to evaluate bone marrow and organ function [e.g., complete blood count (CBC), chemistry, venous blood gas], and symptomatic and supportive care. Achieving control of seizures is notoriously difficult and highlights the need for multi-modal therapies. There is some evidence to suggest that agents which act on the GABA A receptor, such as diazepam and phenobarbital, are less effective than other anti-seizure agents in 5-FU cases. Due to the critical nature of 5-FU intoxications, treatment in an ICU setting is always advised so that the patient may receive intensive monitoring and aggressive round the clock care. Overall prognosis is guarded to poor in symptomatic patients with a survival rate estimated at 20-25%.

Calcipotriene/Calcipotriol

Calcipotriene is potent synthetic analog of vitamin D3 that is used commonly in human medicine as a treatment for psoriasis. This drug is called calcipotriene in the US, and it is known as calcipotriol in Canada and internationally. Dovonex® 0.005% cream, ointment, or solution and Taclonex® solution (calcipotriene 0.005% with betamethasone 0.064%) are common brand names.

Intoxication from calcipotriene can result in life-threatening hypercalcemia and hyperphosphatemia leading to mineralization of tissues and subsequent renal failure. Exposure in pets typically occurs when they chew into a tube of topical cream/ointment. The minimum acute toxic dosage in dogs is 10 mcg/kg with an acute lethal dosage of 65 mcg/kg.

Signs of poisoning, including electrolyte abnormalities, which typically occur within 24 hours of ingestion. Signs may include PU/PD, anorexia, vomiting, lethargy, melena, frank bloody diarrhea, hematuria, and death. While the renal system is often most acutely affected, eventually the cardiac, pulmonary, GI and CNS systems may be as well.

Treatment is focused on controlling hypercalcemia and includes the frequent monitoring of electrolyte levels, especially calcium and phosphorus. In addition to standard treatment for hypercalcemia (IV fluids, prednisone, furosemide, saline diuresis, etc.), bisphosphonates, such as pamidronate, may be helpful and recently have become more readily available and cost effective for pet owners. Pamidronate is dosed at 1.3-2 mg/kg IV diluted in saline over 2 hours and can be repeated in 3-7 days if needed.

Zinc oxide ointment

Topical zinc oxide is found in diaper rash creams (such as Desitin®) and some forms of sunscreen. Zinc oxide can be found over-the-counter (OTC) in concentrations ranging from 5-61%. In acute ingestions, elemental zinc toxicity with hemolysis is unlikely to occur. Zinc oxide is a potent gastric irritant, and most animals will experience spontaneous and self-limiting vomiting following ingestion resulting in self-decontamination. Symptomatic and supportive care may be warranted if vomiting or diarrhea becomes severe or prolonged.

Nicotine transdermal patches
Nicotine transdermal patches may contain substantial amounts of nicotine. When an animal chews on a patch and compromises the rate-controlling drug-release structure, the majority of the nicotine may be quickly released leading to a rapid onset of clinical signs. Individual patches contain anywhere from 7-114 mg of nicotine per patch, which is greater than the amount it is designed to release. Even used patches still contain nicotine and pose a poisoning risk. Clinical signs in cats/dogs have been reported at 1 mg/kg and the oral LD₅₀ for dogs is 9-12 mg/kg. Lethal dose data has not been reported in cats. That said, it is common to find that dogs can tolerate significantly higher doses than this without fatality, especially when treated appropriately.

Nicotine affects multiple organ systems. Soon after ingestion, salivation and vomiting typically occur due to stimulation of the CRTZ. Other signs may include agitation vs lethargy, mydriasis, tachycardia, hyper- or hypotension, tremors, and with higher doses seizures may occur. Mild cases of nicotine toxicity may be over within several hours, but more severe cases can last up to 18-24 hours.

Due to the rapid release and absorption of nicotine from chewed or licked but not fully ingested patches, treatment is typically focused on managing clinical signs. However, if the patch remains intact when ingested, the release of nicotine could be very slow over many hours thus warranting removal via emesis, gastric lavage, or endoscopy. Treatment is symptomatic and supportive as no antidote for nicotine toxicity is available. Heart rate and blood pressures should be monitored closely and treated as needed. Beta-blockers may be used for tachycardia. Severe CNS stimulation or seizures may be treated with diazepam, barbiturates, or phenothiazines. Intravenous fluid administration may increase the rate of nicotine excretion and can be helpful for purposes of normalizing hydration and perfusion. Antiemetics can be used to control vomiting and nausea, but antacids are not recommended as the may increase nicotine absorption from the stomach.

**Fentanyl transdermal patches**

Fentanyl is a synthetic opioid analgesic which binds to μ receptors and is considered 50-100 times more potent than morphine. The transdermal patch is designed to release a constant amount of drug over a fixed time period, but once the internal structure of the patch is disturbed (e.g., chewed on), a large amount of drug may be quickly released. Fentanyl patches contain significantly more fentanyl than is designed to be released during therapeutic transdermal absorption. For example, Duragesic® 50 (Ortho-McNeil-Janssen Pharmaceuticals, Inc) patches are designed to release 50 micrograms of fentanyl per hour for a duration of 72 hours. However, this same patch holds a total of 5 milligrams of drug. Used patches still retain up to 84% of their fentanyl and may pose risk of serious toxicity if ingested.

Signs of toxicity vary between the species. Dogs tend to exhibit more classic opioid effects such as sedation and central nervous system depression, depression of cardiac and respiratory rates, miosis, and hypothermia. Cats are more likely to exhibit paradoxical central nervous stimulation and mydriasis.

Decontamination of pets ingesting fentanyl patches needs to be managed with care. Licked patches typically do not require gastrointestinal decontamination. In a symptomatic patient, naloxone could be administered at the clinic to reverse signs and then emesis could be induced once the pet is more stable. If this is not feasible, in some cases the patch may need to be removed via surgery or endoscopy. Because these patches are designed to release small amounts of fentanyl over a period of days, patch removal is an important part of case management. Once removed, signs of toxicity should begin to resolve within 2-6 hours.

Naloxone may be administered for the reversal of respiratory and CNS depression and may need to be re-dosed as often as every 30-60 minutes or as needed until the pet can support itself. Additional treatments such as intravenous fluids to support the cardiovascular system, thermal support, and mechanical ventilation may be necessary.

**Tea tree (melaleuca) oil**

Tea tree oil, also known as melaleuca oil, is an extract of the leaves of the Australian tea tree (*Melaleuca alternifolia*). This oil has antibacterial and antifungal properties and is typically found in low concentrations <5% in numerous personal-care products such as face washes, shampoos, lotions, and ointments, as well as in shampoos and topical medications marketed for pets. Exposures to these diluted products do not typically result in poisoning. Tea tree oil is also touted as an insect repellent and antiparasitic compound, though with questionable efficacy. Poisoning most often occurs when well-intentioned but uninformed pet owners apply 100% oil as a means of parasite control or for the at-home treatment of wounds or other dermatological conditions.

Tea tree oil is rapidly absorbed from both the skin and gastrointestinal tract with clinical signs beginning within 2-12 hours after exposure. Signs may include lethargy, vomiting, hypothermia, ataxia, weakness, CNS depression, tremors, and bradycardia. Hepatotoxicity has most commonly been reported in cats, but elevated AST, ALT and ALP have been reported in both dogs and cats. Most signs will resolve within 1-2 days or sometimes sooner with minor exposures.

An antidote is not available, and treatment consists mainly of decontamination and supportive care. Bathing the animal with a degreasing soap such as liquid hand dishwashing soap will aid in the removal of the oil from the skin. As tea tree oil undergoes enterohepatic recirculation, the use of multiple doses of activated charcoal may be warranted, even with dermal exposures, provided the patient has an appropriate gag reflex and is not at risk for aspiration. In severely affected patients, the use of IV fluids can be helpful to maintain hydration and tissue perfusion but does not enhance excretion. Temperature support may be necessary, along with nursing care. Baseline biochemistry testing is warranted in symptomatic patients, and liver enzymes should be rechecked until clinical
signs resolve or liver enzymes return to normal. Hepatoprotectants (e.g., SAMe) can be useful if indicated based on biochemical
testing, especially in exposed cats.

**Topical NSAIDs**

Topical NSAIDs are being used more frequently in human medicine, especially for treatment of localized osteoarthritis, such as knee
and hand pain. Diclofenac is the most common topical NSAID in the US and is available as a patch, cream, gel, and solution. Topical
diclofenac 1% cream is also available as an equine product under the brand name Surpass. The FDA recently issued a warning to pet
owners about the potential for toxicity from topical flurbiprofen after cats became ill when humans in the home used these products on
themselves, and it was not determined specifically how the cats were exposed.

Both diclofenac and flurbiprofen are potent NSAIDs with a narrow margin of safety in dogs and cats with potential for gastric
ulcers and renal toxicity at low doses. Treatment involves gastric decontamination including retrieval of ingested patches, bathing
dermally applied products from the skin, activated charcoal, gastroprotectants, and intravenous fluid therapy while monitoring renal
function and for signs of GI bleeding.

**Topical antibiotic ointments and creams**

Topical antibiotic ointments and creams, such as Neosporin and other over the counter generic triple antibiotic combinations, are low
order toxicity when ingested due to poor gastrointestinal absorption. However, there is some potential to kill some good bacteria in the
gut, which could result in mild and self-limiting GI upset. Treatment is symptomatic and supportive if persistent GI signs develop, and
treatment with a probiotic may be helpful for antibiotic-associated diarrhea.

**Topical corticosteroid ointments and creams**

Topical corticosteroid creams and ointments, especially over the counter products, are also well tolerated in most cases. Ingestion of
topical corticosteroids may result in mild GI upset and self-limiting PU/PD, polyphagia, and increased panting. Concomitant ingestion
with NSAIDs can increase risk of GI ulceration. Treatment is symptomatic and supportive if persistent GI signs develop with
antiemetics, fluids, and gastroprotectants as needed.

**Soaps, shampoos, lotions, and cosmetics**

Commercial soaps, shampoos, lotions, and cosmetics are typically low risk for toxicity when ingested by pets. Stomach upset can
occur with ingestion and is usually mild and self-limiting. It is best to check product ingredients as occasionally products might
contain other toxins such as xylitol or salicylates, which can result in more serious toxicity. Gastrointestinal foreign body obstruction
may be a risk if packaging is ingested by pets.

**Suggested reading**

Peterson and Talcott Small Animal Toxicology 3rd edition.
Fry MM, Forman MA: 5-fluorouracil toxicity with severe bone marrow suppression in a dog. Vet Hum Toxicol 46:178, 2004
13:472, 1999

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Xylitol, a pentahydroxy sugar-alcohol, with a sweetness index similar to sucrose, was first discovered by a German scientist named Emil Fisher in 1891. It occurs naturally in many fruits and vegetables, such as strawberries, raspberries, plums, and lettuce and is manufactured by extracting xylan (a polysaccharide found in hardwoods), hydrolyzing xylan to monosaccharide units (D-xylose), and then hydrogenating the D-xylose to produce xylitol. Corn cob remnants from ethanol plants are also now being used for xylitol production. Xylitol has been shown to be antiangiogenic and have antimicrobial properties against common oral bacteria, which makes it a popular addition to gums and candies. It has a low glycemic index and requires few carbohydrates for metabolism, making it an ideal sweetener for diabetics. However, xylitol use is not limited to its effectiveness as a sweetener. Research continues to be done in humans, showing the unique advantages of xylitol to human health. Also, due to its humectant properties, xylitol is used in many non-food items, including deodorants and skin gels. These benefits have led to the expansion of xylitol use over the past several decades, which increases the potential for exposure and resulting toxicity in dogs.

Xylitol is a very safe sugar substitute in humans and many other animals, however, the toxicity to dogs is high, where hypoglycemia may be seen with ingestions of 0.1g/kg body weight and hepatic necrosis at 0.5g/kg. Inadvertent ingestion of xylitol containing gum and candies are the most common causes of inadvertent toxicity. The use of human prescription medications in dogs may pose a toxicity risk as xylitol is frequently used to increase palatability, particularly those that are in a liquid or chewable form. More recently, the addition of xylitol to food products such as sugar-free peanut butter has become a topic of concern in the veterinary field.

Xylitol is incompletely absorbed in the stomach and upper gastrointestinal tract. Absorption and peak plasma levels occur rapidly within 30 minutes in dogs. Xylitol is primarily metabolized in the liver (70-80%) by oxidation to D-xylulose. D-Xylulose is phosphorylated to an intermediate in the pentose phosphate pathway which is then converted to glyceraldehyde-6-phosphate or fructose-6-phosphate, ultimately forming glucose, glycogen or lactate. The majority of xylitol is converted to glucose, with a small amount being converted to lactate. In dogs, xylitol stimulates pancreatic insulin secretion leading to profound hypoglycemia. Dogs develop a dose-dependent increase in plasma insulin concentration after ingesting xylitol. The LD<sub>50</sub> in dogs has not yet been established.

In dogs, vomiting due to the development of hypoglycemia 30-60 minutes post ingestion is commonly seen as the first sign of xylitol toxicosis, followed by lethargy, weakness and ataxia. Elevations of serum hepatic enzyme concentrations, predominantly ALT and T. bilirubin, may occur as early as a few hours after ingestion or be delayed by 24-48 hours. Most commonly, ALT levels rise within 12 hours of toxic insult and peak in one to two days. Coagulopathies that develop in dogs with xylitol toxicosis are likely secondary to acute hepatic failure, disseminated intravascular coagulopathy, or both. The cause of hepatic necrosis in canine xylitol toxicosis is unknown, but two theories have been proposed. One thought is that depletion of adenosine triphosphate (ATP) may result in the inability of liver cells to perform necessary cellular functions, including protein synthesis and maintenance of membrane integrity, which results in cellular necrosis. Another proposed mechanism is that the metabolism of xylitol results in high concentrations of cellular nicotinamide adenine dinucleotide, which produces reactive oxygen species that can damage cellular membranes and macromolecules, leading to decreased viability of hepatocytes.

Treatment includes decontamination with emesis if patient is stable and blood glucose is within normal limits, stabilization of blood glucose levels and providing hepatic support. Due to low binding properties, activated charcoal is not typically used. Monitoring blood glucose closely and providing dextrose supplementation when needed is critical in the early stages of management for xylitol toxicosis. Intravenous fluids with 2-2.5% dextrose supplementation and monitoring blood glucose levels is generally needed for 12-24 hours in dogs that become hypoglycemic. For dogs that develop significant hepatic enzyme elevations, dextrose supplementation may also be beneficial in providing hepatic support. It is important to note that hepatic necrosis may occur without the development of hypoglycemia. Use of hepatic protectants such as S-Adenosyl-L-Methionine and N-Acetylcysteine, monitoring hepatic parameters for a minimum of 2-3 days and providing supportive care, are necessary for a positive outcome in overdose situations. NAC is recommended for dogs with evidence of severe hepatic enzyme elevations. In addition to its mucolytic properties, it is effective at restoring glutathione synthesis and reducing oxidative stress in the liver. Glutathione is essential in cell detoxification and many metabolic processes. SAMe is generally recommended to be given for 2 weeks in dogs with mild hepatic enzyme elevations and continued for 4 weeks in dogs that develop hepatic necrosis. SAMe is present in three major biochemical pathways important to the liver. Through these pathways, SAMe plays a role in liver mass regeneration, cell membrane structure, fluidity and function, cell detoxification and is converted to glutathione. The liver normally produces SAMe, however, in a diseased state.
endogenous SAMe conversion is decreased, thus the administration of exogenous SAMe may increase liver glutathione levels and prevent its depletion.

Coagulopathy is a common sequela to hepatic necrosis leading to an increase in PT and PTT. All coagulation factors, with the exception of factor VIII are synthesized in the liver which is the site for vitamin K dependent activation of factors II, VII, IX and X. Vitamin K deficiency may be seen due to altered enterohepatic circulation of bile acids, leading to malabsorption. Fresh frozen plasma treatment is generally successful in restoring PT and PTT values as it provides all of the coagulation factors in their active form.

There is no antidote for xylitol toxicosis and general treatment guidelines include decreasing absorption, treating emesis, stabilizing blood glucose, protecting the liver, addressing coagulopathies, and providing further care as needed.

Suggested reading
Peterson and Talcott Small Animal Toxicology 3rd edition, Ch. 83.
I Just Don’t Want Him to Suffer—Pain and Death
Dani McVety, DVM
Lap of Love
Lutz, FL

It is a common statement, “animals hide their pain.” But do they really? And is pain the same as suffering? Do our animals not want us to see their pain or are they simply genetically programmed to not emotionally respond to it the same way we are? All veterinary professionals know that different species (and even different breeds within species) react differently to stress and physical discomfort. Understanding these differences and how to think about pain and death from a pet’s point of view helps us better communicate these nuances with clients. This results in a more comprehensive and understandable explanation to the owner of the patient’s perception of discomfort and the capacity for suffering during the end of life experience.

We all understand the broad concept of “pain” (i.e., nociception). Whether or not animals can feel pain is luckily not debated any more. (However, we don’t think insects feel pain since they do not “pain guard” (Eisemann et al 1984), such as limping on a broken leg. The fruit fly is the only known exception to this, they have been shown to pain guard when injured (Tracey et al 2003)). So what is the difference between pain and suffering? After practicing emergency medicine followed by thousands of veterinary hospice cases, I have come to define suffering as the inability to both think about anything else AND the inability to physically do anything else other than address the pain (be it mental or physical) that an individual is experiencing. This would hold true for both the cat in congestive heart failure that is struggling to breathe and the dog with severe thunderstorm anxiety; they are both suffering.

Think about how your dog reacts when you step on his toe and you realize that he has no problem communicating his discomfort to you. But then think about the female Labrador that was spayed a few hours ago and now must be restrained from roughhousing; is she really hiding her pain? Why are there differences in these outward signs of discomfort when we know both of these examples are painful (although much different types of pain)? The first step is coming to an agreement on the term “hide.” When clients tell us they are worried about properly identifying pain in Fluffy because she hides it, does that mean Fluffy doesn’t want her owner to see her pain and therefore displays these outward signs of discomfort in private? Or does it mean that Fluffy is biologically programed to not show pain at all in order to protect her standing in the pack or to avoid predation (like prey animals)? Or perhaps Fluffy simply doesn’t care about her pain (although fully feels it) in the same way humans do? Does the difference between these concepts really matter? The answer is probably somewhere in the middle but without the ability to speak “dog,” we will never fully know for sure. But any veterinary professional will tell you that even when an animal is alone, they will usually (not always though), act as if their injury or illness does not hurt them as much as we believe it would hurt a person (research is still torn on this subject, however).

So do animals simply experience pain differently than humans? Again, we know there are species differences, but are there major anatomical differences that would help us conceptualize this? As humans, we are considered “higher beings” due to the more developed frontal lobes in our brains. This is what allows us to make music and contemplate our own existence, to name a few. Since animals do not have as much grey matter as we do (and theoretically less “consciousness”), is their experience of pain different than ours? We know that different species can use different parts of the brain for different functions. The connection between the frontal lobes and pain (mainly chronic pain) has been studied for years (Lorenz et al 2003) and was the idea between the controversial and strange practice of leucotomies in the 1940’s and 1950’s. Before antipsychotic medications or therapists this was how society began dealing with the mentally impaired. Basically, the procedure involved cutting the connections to and from the prefrontal cortex, the anterior part of the frontal lobes of the brain (Achary et al 2004). Besides some pretty awful side effects, there were interesting post-surgical developments in those patients experiencing life-limiting and debilitating pain before the procedure. Patients that were completely nonfunctional due to extreme physical suffering (probably akin to fibromyalgia in modern day) were up and about, playing card games and conversing just days later. They appeared to be better. One patient, after being asked how he was doing, responded, “the pains are the same, but I feel fine now, thank you” (Demasio 1994). There are numerous accounts describing this phenomenon. After the procedure, it is said that the patients stopped caring about their pain; Dr. Demasio noted that they “kept their pain but lost their suffering.” These patients still asked for painkillers but were satisfied with aspirin, no longer needing morphine. It’s clear that they still felt pain because when poked with a pin they shrieked; in fact, they shrieked louder than a normal person, probably due to lower impulse control from the disconnected frontal lobes. These patients were most likely feeling what normal humans consider mild pain; that which still exists and causes discomfort but can be ignored and does not ruin your life by consuming your mental thoughts. (This procedure was all but extinct by the 1970’s due to a myriad of undesirable side effects such as loss of initiative, inhibition, and decreased cognition to name a few.)

Perhaps animals lie somewhere in the middle of a leucotomy patient and a normal human being. To me, there are many similarities, although certainly not the same, between the leucotomy patients and dogs in how they emotionally react to pain. I am not inferring that animals don’t physically feel as much pain as normal humans do, simply that they don’t emotionally interpret, respond, and react to it the same way a normal human with intact frontal lobes would. For example, a few years ago my rat terrier jumped out
of a friend’s arms, completely fracturing her radius (complete mid-diaphyseal fracture). She did not whine, cry out, or even hide (although other dogs might have done these things). She simply jumped on the couch and sat there looking at me with her bright eyes, holding up her mangled leg. I knew she was hurting just from the look on her face, but she honestly reacted the same as if I had stepped on her foot. Of course I was a mess; I knew this meant my little girl would have to go to the veterinary hospital (which she hated), put under anesthesia, surgery, recovery, cage rest, and so on. I took on the emotional component while she experienced the pure and unadulterated physicality of pain, seemingly void of interpretation to what that pain meant. And yet the benefit of my understanding was that I knew her pain would eventually end. Animals, on the other hand, cannot perceive an ending to their state of pain, making our job of pain identification and treatment incredibly important.

If it were me that broke my arm, I would be anxious about the impending surgery, recovery, loss of time with my children, and so on. I would generate negative emotions that lead to amplification of my physical pain. The bright side, however, is that I know that with some medical attention I will be out of pain in the future. Animals may not experience this in the same exact way that I would, but watch a fearful dog walk into a veterinary clinic, or a thunderstorm completely debilitating an animal and you will see a pure form of suffering. Temple Grandin, PhD. says in her book Animals in Translation, “the single worst thing you can do to an animal emotionally is to make it feel afraid… fear is so bad for animals I think it’s worse than pain.” Herein lies the most important part of managing end of life cases in our hospice practice; address physical pain but most importantly address any stress, anxiety, and fear that our pets are experiencing as a result of either their physical or mental discomfort. Many of our arthritic or immobile pets appear more agitated by their inability to stand up rather than the pain that standing up elicits. These dogs may not understand why they cannot ambulate, leading to excessive panting, whining, crying, and additional physical pain through their attempts to move. Much of the time these symptoms are alleviated simply by the owner’s presence, but this is not always possible. Many times, the mental battle is bigger than the physical one with our patients.

These are concepts I discuss with families on a daily basis. Veterinary hospice care, by striving to maintain quality of life versus quantity of life, is centered on addressing pain AND any other mental stressors that may be present. To this extent, the owner becomes our greatest source of early identification of new developments with their pet’s condition. They generally feel that their bond is so strong that they can sense the discomfort, and with a little retraining and education on how an animal may react and perceive pain AND anxiety differently than we do, we can become partners in the journey of making the end of life period as pain-free, anxiety-free, and fear-free for both the pet and yes, for the owner as well. There needs to be a clear understanding of the differences between discomfort (something we will all have when we’re 95 years old!), pain (something that should always be addressed), and suffering (a mental state that should be avoided at all costs). Euthanasia is not just about ending suffering that is occurring at that moment, but rather about preventing it from occurring in the first place. And with a better understanding of mental and physical pain and/or suffering, clients feel better equipped to make that important decision with the guidance of their veterinarian.

**Helping families identify pain in their pets**

As veterinarians, we have many resources available to assist in pain identification in animals. The International Veterinary Academy of Pain Management is a wealth of information. Below are some additional helpful tips that we have found particularly useful when talking with clients during the hospice period.

- Does your pet act overly concerned when you approach him? Does he seem to shy away from your caresses? This may indicate anticipatory pain. Your pet may be anticipating discomfort that is elicited when being touched or moved. Humans in hospice care show a similar phenomenon of not wanting to be touched when the body is nearing the end (usually days to a few weeks before death).
- Is your pet appearing more hunched back or grumpy, especially after waking up?
- Pay special attention to the way your dog lies down. Hiding a certain paw can indicate even mild pain in a related part of the body. Taking a few pictures of him throughout the day may illustrate mild changes.
- Many dogs blink the moment they feel pain. If you see it, try to replicate the movement again or note how and why the increased blinking occurred. Along those same lines, follow your dog’s eyes. Avoidance of eye contact or looking away can indicate pain.

**Resources**


Integrating Veterinary Hospice
Dani McVety, DVM
Lap of Love
Lutz, FL

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. So, when do you make that decision? This presentation will give attendees tools to help guide owners through the decision process 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 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:

- 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.”)
- What do you hope the life expectancy of your pet will be? What do you think it will be?
- What is the ideal situation you wish for your pet’s end of life experience? (at home, pass away in her sleep, etc.)
- Do you hold any stress or anxiety about any of these issues?
- Pet suffering; 2.) Desire to perform nursing care for pet; 3.) Ability to perform nursing care for 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)

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 (an “imminent” condition).

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 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. 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 the 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)

**Social functions**
1.) Desire to be with the family has not changed; 2.) Interacts normally with family or other pets (i.e., no increased aggression or other changes).

**Natural functions**
1.) Appetite has stayed the same; 2.) Drinking has stayed the same; 3.) Normal urination habits; 4.) Normal bowel movement habits; 5.) Ability to ambulate (walk around) has stayed the same.

**Mental health**
1.) Enjoys normal play activities; 2.) Still dislikes the same things. (i.e., still hates the mailman = 0, or doesn’t bark at the mailman anymore = 2); 3.) No outward signs of stress or anxiety; 4.) Does not seem confused or apathetic; 5.) Nighttime activity is normal, no changes seen.

**Physical health**
1.) No changes in breathing or panting patterns; 2.) No outward signs of pain. (See Resources Below); 3.) No pacing around the house; 4.) My pet’s overall condition has not changed recently.

**Results**
- **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.
- **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.
- **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.

**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
- 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.
- 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.
- 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 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 HHHHHMM 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

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

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.

Resources
When it comes to ethical-border-line euthanasia requests, we have a very important decision to make as veterinarians, but we need to ask the right questions from the start. Instead of deciding whether or not you are comfortable euthanizing that pet, the question should be “what are the alternatives for this pet.” By requesting euthanasia in the first place, the family is communicating to you that the human animal bond is broken. We can either help change the situation for them (remove the pet from their care via adoption or euthanasia), or do nothing by sending them home because “I just can’t do it.” And in my opinion, doing nothing is professional suicide; you’ve now ruined any rapport you had with that family, a small loss that does not create societal trust and respect for our profession. Helping a family, in whatever way, is far preferable than sending them home with a broken human-animal bond.

Remember, medicine is not our product in the veterinary world, the human-animal bond is. Without that bond, they are not coming into our clinics. When euthanasia is requested, the family is telling us that there’s something wrong with that bond and they care enough to tell you about it instead of letting the dog or cat go on the side of the road.

So what should be done in these extreme cases of uncomfortable euthanasia requests? Allow me to push the boundaries a bit; in my opinion, we must take responsibility for the pet in some way. As a house call hospice veterinarian, if I am at a home of a pet that I do not feel comfortable euthanizing, and with an owner that simply cannot go on, the pet will come home with me. Yes, it’s happened. And have I euthanized animals that I may not have euthanized if they were mine? Absolutely. Have I euthanized animals that other veterinarians have refused to euthanize? Absolutely. Have I euthanized animals whose owners were completely at a lost, unable to go on for many reasons, and with tears in everyone’s eyes (including mine), we knew it was a difficult but good decision? Absolutely. And when those families hug me, knowing that I did not judge them for that tough choice we made together, that I did not force an altruistic or idealistic view on them, and that I partnered with them in opting for the best alternative option for their pet, a new level of respect is earned.

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.
- Non-Medical & Convenience Euthanasia Rules
- 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 the pet and the greater good.
- 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.
Never Have I Ever...Have You?
Tame Conflict and Fear- and Let Well-Being Run Wild
Caitlin DeWilde, DVM
The Social DVM
Webster Groves, MO

Kimberly Ann Therrien, DVM
Banfield Pet Hospital
Palm Harbor, FL

Sarah Wooten, DVM, CVJ
West Ridge Animal Hospital
Greeley, CO

Never Have I Ever is a party game, but in this presentation, we will utilize the game to teach three important tools that are necessary for leadership success. This presentation promises to keep session attendees laughing while simultaneously navigating the career obstacles our profession, and often our gender, may encounter.

As professional women, we’ve got so much to do and so little time that the idea of spending time doing anything unrelated to the to-do list actually creates stress. We convince ourselves that caring for our minds, emotions, and bodies is a waste of precious time. We even convince ourselves that rest is a waste of time. In the interest of caring for our careers and everybody else in our lives, we have pushed aside respecting our own emotional, mental, and physical health. In order to survive, we’ve got to lighten up, take care of ourselves, deal with conflict head on, examine negative thought patterns and habits that have lead to a crisis of depression and anxiety within the profession, and develop new tools and strategies to not only change the trajectory of our professional careers and raise our effectiveness as leaders, but our own pursuits of happiness and fulfillment.

Did you raise your paddle because you frequently shy away from conflict and perhaps allow yourself to be sucked into drama that you really wish you were not a part of? Many of us, I’m sure, would love the opportunity to work in an environment where conflict rarely arises.

Unfortunately, the profession we have chosen is filled with conflict - conflict with clients, conflict with peers, conflict with managers! Confidence with conflict resolution and managing through difficult situations can absolutely be achieved. Let me show you how!

Application: Books like “Crucial Conversation” and “Influencer” offer great tips and tricks on how to be more confident with conflict.

Did you raise your paddle because you are scared of something that you have really wanted to try? One of the major blocks to our success is our response to fear. Many of us are conditioned to shy away from what scares us professionally and personally. When it comes to fear, however, we have more control than we think. We can’t control what happens in our professional and personal lives, but we can control our interpretation of it. This is called ‘reframing’. Take one possible belief or worldview, discard it, and reach for a more positive one. What could have been interpreted as a negative situation could now be interpreted as positive.

Application: Take an active look at how you are interpreting external events. Once you recognize it, challenge that view. Try to reframe any fearful, negative views into more positive ones. Reach for any thought that gives you a sense of relief or positivity.

Many of us could “raise the paddle” on working way too many hours, missing lunch, allowing clients to treat us poorly, and so on and so on. But these shouldn’t be things that we are proud of, or sacrifices to wear as a badge of honor. Just because we can doesn’t mean we SHOULD. Reverse the trend. For instance, make taking your lunch hour the norm, not the exception- the paperwork can wait. We all know we’re going to have to miss one or two to see that emergency,

but the majority of the time, that lunch break can help us re-energize, quiet the storm in our brains (and maybe in our clinics), and provide a much needed downtime. In many cases, our productivity will increase when we’ve allowed ourselves the proper breaks.

Outline for yourself the qualities, time and goals you need to feel balanced and happy. Start working toward these goals within your practice, and don’t be ashamed to keep your paddle down.
We Don’t Go Into Vet Med for the Money—Clearly!
Real-Life Strategies to Enrich Our Money Mindset
Caitlin DeWilde, DVM
The Social DVM
Webster Groves, MO
Kimberly Ann Therrien, DVM
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Palm Harbor, FL
Sarah Wooten, DVM, CVJ
West Ridge Animal Hospital
Greeley, CO

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, it’s 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 AVMÀ’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 “give 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, let’s 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
How to Stop Blaming Yourself and Take Responsibility for Your Mistakes

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Two issues we have with mistakes
We are either fearful of making them or the fear of making a mistake stops us from even trying in the first place. Both forms will hold you back from reaching your full potential. This lecture will dive deep into strategies we can use to face our fears.

Because it feels like the end of the world
It’s happened, you made a mistake. Maybe it’s just a small mistake, maybe it’s a colossal one. Maybe it’s a mistake you never thought you would make, and scoffed at others who did. Maybe you thought you had prepared, and were in full control, when the unexpected happened. Whatever the reason, making a professional mistake can be soul crushing and utterly devastating. If you’re anything like the majority of veterinary professionals, regardless of how it happened, you WILL, I repeat, you WILL beat yourself up over it. And no matter what anybody tells you, it will not soothe your aching soul, at least not in the initial aftermath it won’t.

Driven by fear
I don’t know about the rest of you, but I can speak for my veterinary class at least, and I will tell you, that our professors put the fear of God in us when it came to making a mistake. Obviously this was coming from a place of wanting us to learn from others mistakes, and to be prepared, yet it was also quite damaging. The truth is we are all going to make mistakes. Yes it is in our best interest, to be as prepared as we can be, to train as hard as we can, and to minimize the likelihood of it happening. Come to think of it, most of my success in veterinary school was driven by fear of failure. Fear can be a great motivator, but it can also be a handicap. It’s up to us to discern when fear is holding us back.

We are scientists and we want to predict and control outcomes
We are dealing with biological systems here, and as much as we try to predict and control these systems, there is plenty that is not in our control. This is a fact of life. And we have to find a way to come to terms with this otherwise we will lead ourselves down a path of self destruction.

Balance is key
This does not mean you get to go around making mistakes left and right, and say; “Well that’s just life!” or “I’m learning to let go of my attachment to outcomes.” It’s about learning a new skill, or Do not let the pendulum swing all the way in the opposite direction or extreme. Sometimes for instance when you first learn a new word you have never used before, you start using it everywhere, even in sentences it doesn’t make sense in. Discernment is key, requires effort and daily practice.

Trust your inner voice
I worked in emergency and general practice for some time, and there were many nights I was at the clinic by myself, stressing over if I should try and get a urine sample one more time from the dying kidney patient, or if I should scan the belly one more time to see if there is free fluid. Did I give enough pain med? Did I give too much? Are they panting because of pain? Or are they in metabolic acidosis? Did I do enough? When is enough? Should I have pulled the plug sooner? Am I causing this patient to suffer more? The list of questions goes on and on. These questions are important to ask, and you should be asking yourself these questions. However, there comes a point in the day, or night, where you have to decide that you have done everything you can for that patient and that (you have to be willing to let go. Trusting yourself is a skill that needs to be honed, and sometimes this skill can only be perfected through making mistakes. Take a bathroom break and take a few breaths when you need to get in touch with your inner voice. That’s why the bathrooms need to be the most Zen place so you can have some privacy and get away for a few minutes. (Soft cushy toilet seat, diffuser for aromatherapy, timer with soothing sounds, a nice poster of your happy place.)

The four agreements
In the lonely hours of the night that I sat in the clinic looking for comfort, and answers, I would often turn to “The Four agreements” by Don Miguel Ruiz. I would use this as a checklist for my day. Will give one example of each and ask audience for interactive sharing.

1. Be impeccable with your word
Speak with integrity. Say only what you mean. Avoid using the word to speak against yourself or to gossip about others. Use the power of your word in the direction of truth and love.
2. Don’t take anything personally
Nothing others do is because of you. What others say and do is a projection of their own reality, their own dream. When you are immune to the opinions and actions of others, you won’t be the victim of needless suffering.

3. Don’t make assumptions
Find the courage to ask questions and to express what you really want. Communicate with others as clearly as you can to avoid misunderstandings, sadness and drama. With just this one agreement, you can completely transform your life.

4. Always do your best
Your best is going to change from moment to moment; it will be different when you are healthy as opposed to sick. Under any circumstance, simply do your best, and you will avoid self-judgment, self-abuse and regret.

Empowering yourself and letting go of self abuse
This stems from what we learned in childhood. We were often told to be good girls or good boys. We would try to lie to cover up when we made a mistake. Think about a time when one of these scenarios may have happened at work. What was your role in it? How did you respond?

- Vaccine mix ups
- Drug mix ups
- Drugs left out
- Drugs not logged
- Mixed up blood tubes
- Surgical mistakes

In order to get over all this you have to do the inner work. Being honest in your career will also help you with all other areas of your life. Being able to admit when you were wrong is a very important human quality. It starts with being honest with yourself.

Sharing stories of a time you made a mistake and how you took responsibility for it
My chemo story…and stories shared from attendees.
Coping with Anesthetic Loss
Hilal Dogan, BVSc
Veterinary Confessional Project
Maui, HI

Tasha McNerney, BS, CVT, CVPP, VTS (Anesthesia/Analgesia)
Veterinary Anesthesia Nerds
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It starts with understanding traumatic stress
Traumatic stress manifests in almost all of the Medical professions. Although widely recognized in first responders, caregivers and emergency physicians in the human medical field, only since the early 2000’s has it been gaining more awareness in the veterinary profession. Reasons for this may be multi-factorial. One such reason might be due to the fact that in the past animals were not viewed to be sentient. ‘Sentience’ is defined as the ability to have perceptions and sensations. A ‘sentient animal’ is an animal that is aware of his/her surroundings and of what happens to him/her and is capable of feeling pain and pleasure, at the least.

What took us so long to see the cause?
Compassion fatigue has been defined as “the emotional burden that health care providers may experience as a result of overexposure to traumatic events that patients are experiencing” (Schwam, 1998). Therefore if animals were not previously thought to have the ability to experience pain per say, then perhaps it was not as easily recognized as with humans. An earlier term used to describe this phenomenon was secondary victimisation (Figley, 1982). Carla Joinson is generally credited with introducing the term “compassion fatigue” into the literature (Joinson, 1992) in an article about a special type of burnout being experienced by nurses in an emergency department. (Huggard & Huggard, 2008). If animals were almost akin to an inert object, then it makes sense that this key concept, that we can experience traumatic stress, and more specifically vicarious trauma (compassion fatigue/secondary traumatic stress) has gone under recognized in the veterinary profession.

Anesthetic losses are a form of secondary traumatic stress
Indeed. Yet there are no practices in place in most clinics, even schools to help one deal with this when it occurs. In my opinion, this is negligent on behalf of the practice owner, institution or even us, associates and technicians. It’s like sending someone to battle with no armor, or basically being ignorant.

This is your brain on secondary trauma
Remember that commercial “This is your brain on drugs”? Where they crack and egg open onto a frying pan and it cooks? Same deal. However back then we didn’t have the knowledge of Neuroplasticity that we do now. Therefore the plus side of this information now is that we know the effects can be reversed, greatly reduced or even prevented (in certain circumstances, but not all).

Healing from trauma and honing your resiliency skills
There are countless ways now to heal from trauma. The most important step in recognition, then healing can start! Some methods we will demonstrate during this session include: meditation and deep breathing exercises, self inspired-dialogues, fear inventories and peer based connection.

References
Firing Employees- and What to Learn From It
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Terminating an employee is a difficult and painful process for both the employee being let go and the practice employees having to do the firing and then having to pick up the pieces afterwards. However, every business will need to terminate employees at one point or another. Knowing how to get rid of someone professionally and how to learn from the experience can make the process easier.

Preparation
Pre-termination preparation is critical. The following steps should be taken:

- Understand your legal position
  - Even in an “at will” state where you can generally fire anyone for any reason, there are exceptions. For example, you can’t fire someone because you don’t like their religion or a woman has decided to have a baby.
  - Remember that written documents (offer letters, contracts, and employee handbooks) and oral statements can also impact the employer’s rights in this regard.
  - Anyone can sue anyone for any reason; you don’t want to deal with this even if you win in the end. It takes time and costs money. Handling the termination well can help prevent this.

- If this is an out-of-the-ordinary firing or you have any concerns that the situation could deteriorate badly, talk to an attorney before you terminate the employee. This is particularly important to consider with
  - Employees who talk a lot about their rights and have some knowledge of them
  - Sensitive issues such as sexual harassment
  - Members of a protected class
  - Employees who’ve complained about poor treatment before or filed some sort of claim
  - Employees on medical, military, or other protected leave
  - Contractual rights

- Identify the specific behavior that is leading to the desire to terminate the employee. Vague statements such as “he’s just not a good fit” or “he doesn’t seem to catch on well” aren’t good enough. It is important for you to know why so you can prevent a similar situation from happening in the future, and so you can explain to the employee why you are letting him/her go. And while you generally shouldn’t discuss the firing of one employee with another, employees know when a company handles these situations professionally and fairly and when they don’t. Even if you have the legal right to fire for any reason, consider the effect on other employees if the reason is frivolous.

- If the firing is going to be done because of one specific action by the employee, make sure you have fully investigated the situation including having talked to the employee involved.

- If the firing is based on an ongoing pattern of behavior, make sure the practice has proper and escalating documentation and, ideally, a specific warning to the employee that their job is in jeopardy. Even in an “at will” state, this will protect the practice if there are any unexpected legal consequences.

- Think through whether firing is the best choice—is the employee behavior so bad that this is the only realistic choice? Is the employee being treated consistently with how other employees have been treated? Is firing consistent with other actions of the practice related to this employee? (For example, was the employee recently promoted or given a raise that would seem to be at odds with the decision to fire him/her?) If you’re being harsher on this employee for the stated offenses than with other employees, search for the real reason you want to get rid of this person.

- Consider whether the practice provided the employee with the tools and resources needed to do their job and treated the employee fairly and professionally. Were expectations communicated appropriately?

Meeting with the employee

- Select a private location and an appropriate time. Usually the best time is at the end of a workday when you won’t be interrupted and the employee won’t have an opportunity to be disruptive nor be embarrassed when leaving. If at all possible, have a witness to the meeting.

- Review your documentation and the history of the situation immediately before the termination meeting so it is clear in your mind.

- State the actual and true reasons for the employee’s discharge in clear, understandable language. There is no benefit to being mean and hurtful nor to sugar coat the truth. Stick to the facts of the situation and the employee’s behavior and not your interpretation of the facts or a description of the employee’s personality traits. For example, say: “You have been late 19 out of 22 workdays this month” instead of saying “You show no dedication to this job.”
• If appropriate, give the employee the option to resign.
• Don’t bargain or negotiate with the employee about the decision to discharge him or her. The decision to terminate someone should have been made after all measures were already tried to improve the employee’s performance.
• Act in a professional manner—do not become upset or defensive. Remember that this is very hard for the employee and treat them with respect and courtesy and as you would like to be treated in a similar situation.
• Take notes during the meeting.
• Explain the compensation and benefit situation to the employee—make sure you are in accordance with state and federal law in how you handle this.
• Use a checklist to insure all important items are covered in the meeting; this includes points of discussion as well as actions that need to be taken such as a return of keys.
• Allow the employee to gather their personal items and escort them from the building—do not leave them time to do damage to the practice or have an opportunity to vent hostility on other employees or clients.

After the meeting
• Take your notes and convert them into a report that includes what was said and significant things that were not said (for example, the employee didn't dispute the reason for discharge.) Stick to facts in this document; no opinions nor any comments that could be perceived as violating relevant law. Witnesses who attended the meeting should review the report, add comments if needed and sign the report along with the original author.
• Announce to the rest of the team that the employee has left the practice and how scheduling will be handled until a replacement can be found. Do not discuss the reasons for the termination outside of the management team members who need to know. Remember that, realistically, employees generally already know!

Learning from your mistakes
Anytime an employee is terminated, there is something the practice could have done better. Whether this involves hiring, training or performance management, learn from these mistakes. Areas to review include:
• Job descriptions
• Performance evaluations
• Training
• Day-to-day coaching
• Employee manual
• Contracts
• The termination meeting itself

It is common for practices to put off dealing with problem employees until it is too late to realistically improve performance. Be open and honest in dealings with employees from the beginning—if they are not doing well, deal with it sooner rather than later. Make sure the practice has clearly set expectations for the employee and is aware of any issues on the employee’s side that may be contributing to the problem. Train all employees in your practice who supervise employees to do the same.

Review your system for dealing with employees who aren’t meeting expectations—typical steps include a verbal warning, another verbal warning documentation in the employee’s file of this warning, a written warning, a formal improvement plan and a last chance warning. The last chance warning should be very specific: “We have talked multiple times about the fact that you are tardy several times every week and it has not improved. If this happens again, you will be let go.”

Of course, as a part of this process, the practice needs to look at whether they have done their part in making it possible for the employee to succeed. While the above steps are used with most employee performance issues, there will, of course, be some offenses that warrant firing immediately.

Have a plan for replacement
Before you terminate someone (assuming it doesn’t have to be done immediately), have a plan in place for replacing this employee:
• Consider whether some current employees would be interested in OT
• Review the schedule to see how it could be revised
• Review your applicant file
• Call colleagues for castoff applicants
• Start networking for applicants

While terminating an employee is never fun, the practice will generally be better off because of it. As Harvey Mackay (“Swim with the Sharks”) says: “It’s not the people you fire who make your life miserable, it’s those you don’t.”
Selling a veterinary practice can be difficult logistically, financially and emotionally and the hardest part is often just knowing where to start. One of the most important areas to focus on concerns the amount you (as a seller) want to get for the practice. The sooner you start to consider this figure, the more opportunity you will have to actually get the price you want.

The profitability of the practice is almost always the most important driver of practice value. Whether you are 2 years or 30 years away from selling your practice, this is a critical metric to understand and yet most practices don’t know what that number is. Improving your profitability will improve your practice’s value at the time of sale and increase your cash flow in the years before. This is true whether you sell to another veterinarian or to a corporate consolidator.

Profitability
Historically, practice owners have assumed (and with good reason) that when they decided to sell their practices there would be buyers ready to purchase them and willing to pay a good price. In other words, they have assumed there was value in these businesses that could be transferred to someone else. Of course, there have always been a few practices for which this assumption didn’t hold true. A buyer couldn’t be found or what buyers wanted to pay wasn’t remotely what the seller thought the practice was worth. Typically these practices have been easy to identify and had several traits in common. They tended to be smaller practices with owners who had not focused much on the business side of things. Often the facility and equipment were old and the doctors hadn’t kept up with the changes in medicine as much as perhaps they should have. These practices had little profit in them and, because the bulk of practice value is determined by profitability, the practices had little value. Fortunately there weren’t too many of these practices.

However, in the last fifteen years, the number of practices with no or little value has been increasing—to the point where the Veterinary Valuation Council of VetPartners coined the term “No-LoSM practice” to describe these practices. More and more practices, when appraised, did not have the value that owners wanted them to have. And, in almost all cases, the owners of these practices were totally unaware of the problem. Some of these practices had traits in common with the practices that have historically had little or no value. They were small practices with a low level of profitability and couldn’t keep up with changing client demands regarding service, quality of medicine, advanced technology and improved facilities. The other practices with no or little value, however, were a surprising group. On the surface, these practices would appear to be doing very well. They are located in very attractive facilities, practice good medicine, have all the latest equipment and a large support staff, offer comparatively high compensation and benefits to their employees and, in the owners’ eyes, cash flow is strong. However, practice value is largely based on profits and the very factors that make these practices look attractive on the surface are those that are reducing profitability.

Calculating the true operating profits of a practice is, however, not a simple task. None of the standard financial or management reports a practice usually gets includes this figure. Neither the taxable income from the tax return nor the net income from the profit and loss statement represents true profitability. This doesn’t mean those reports are improperly prepared; it simply means the reports required by the IRS or accounting standards for small businesses weren’t designed to determine profitability. No one report will give a practice all of the financial information it needs to make intelligent operating decisions; unfortunately, the report that seems to be prepared least often is the one that calculates true practice profitability. Because practice owners and managers aren’t used to getting this kind of information, they generally don’t know what the true profitability of their practice is. The first time many owners realize their true profitability is when their appraiser talks to them about it.

The operating profit is the difference between the operating revenues and expenses of a practice. Operating revenue and expenses include only items normally and necessarily seen in the day-to-day operations of the practice such as fees for professional services and drugs and medical supplies expense. These items should be stated at fair market value rates. For ease of comparison with other practices, the profit margin is generally stated as a percentage—this is calculated as practice profits divided by gross revenue. Some of the items that must be calculated differently to determine operating profit versus taxable income or net income include: practice owner payments, facility and equipment rent if these items are owned by the practice owner and leased to the practice, services provided by family members to the practice, depreciation, interest on debt and perks.

How is the operating profit calculated?
The following steps are generally what is needed to get to this figure although there can be some variations in individual practices. Taxable income per the tax return is usually the starting point. Various adjustments are made from there.

- Add back: depreciation, amortization, equipment lease payments treated as an expense in the tax return and interest on debt
• Deduct the estimated average amount spent on equipment per year—purchasing equipment is a true operating expense of the practice but depreciation as determined by tax law is not the best estimate. A reasonable estimate in many practices is 1.5% of gross revenue.
• Determine how much the owner was paid in compensation and rent (if the practice owner owns the practice facility as well) during the year.
• Adjust owner compensation to represent a fair compensation for medical/surgical work—20% of personal production is a good average in a small animal practice.
• Adjust owner compensation for management work—management expense generally averages 3-5% of gross revenues—if the practice has a practice or office manager, the owner should get less than this amount as management compensation—1.5% is often used to represent owner management compensation when the practice has a manager.
• Adjust payroll taxes for any compensation changes.
• Adjust rent expense to fair market value if paid to owner at a rate greater or less than fair market value.
• Determine the $ amount of personal perks paid by the practice and remove this expense—perks would be items not necessary to the operation of the practice but paid by the practice generally to gain a tax advantage (examples include excess meals and entertainment, excess auto costs, swimming pool payments, personal furniture, trips to Tahiti, etc.).
• Deduct the cost associated with free services provided to the practice—family members may provide bookkeeping or other services to the practice at no charge—if the practice had to hire someone to do this work, there would be a cost involved and this should be included as an expense.
• Add back any compensation and benefits paid to family members that don’t provide equivalent services to the practice.
• Remove any true non-recurring income or expenses such as one-time insurance proceeds or expenses related to a natural disaster.
• Remove interest and dividend income.
• Remove interest expense.
• Recalculate net income.
• Divide the new net income by gross revenue to express profitability as a percentage.

The resulting percentage is the true operating profit of the practice—how does it compare to other investments you have? And to other practices? 20% or above would be considered superior, 12-13% average and less than 6% poor.

The above may sound a little daunting but there are resources available to help. If you work with a veterinary financial advisor, this person should be able to calculate your profitability. VetPartners also has “The No-Lo Practice” available for download at www.VetPartners.org. Within this document is a worksheet to help guide you through this calculation process.

If the practice’s profits aren’t at the desired level, what can be done about it? A lack of profitability either comes from revenues that are too low, expenses that are too high, or a combination of the two. Understanding not only the profitability of the practice but the kinds of factors that lead to this state is critical. Until the practice has an idea of the root causes of the problem, it is difficult to determine what the correct solution is. Working with a financial advisor or practice consultant may help in not only gaining a greater understanding of the issues impacting profitability but in identifying and implementing solutions.
Fire Power:
When Will YOU Terminate the Problem Employee?

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In my career, I have managed practices ranging in size from 2 doctors to 13. I have also been a regional director of five practices totaling more than 300 employees. The decision to terminate and the process of termination have always been difficult for me. In the aseptic security of an HR seminar, who to terminate and how to terminate always seemed clear, but invariably when I returned to the practice, the waters muddied again. ‘How can I terminate Dr. Gafferty? She’s one of our top producers and the clients love her…yet she is consistently stirring a pot of gossip. Keeping her on means more drama, yet if I let her go, it will take me months to replace her.’

In hindsight, I wish that I had had stronger convictions about the value of culture and the practice’s mission; that I would have stood against any person that challenged these important foundation stones. The waters of termination would still have been muddy, but I would have been better at coaching the employee as they edged towards termination; I would have agonized less over my decision to terminate; and I would have handled the termination process better.

In our lecture, we’ll give you some ideas that may make your decision to fire a marginal employee easier, if anything like termination can ever be called ‘easy’. Mostly, I know what it’s like to make these decisions within the lonely confines of y our office where you have no one to act as a sounding board for your thoughts. This lecture will give you more confidence when deciding if you should terminate an employee.

It’s about what you are about
Practices that have a very strong sense of who they are and how they should operate are more likely to have team members who fall in line with those beliefs. Practices where the leadership has a clear vision of the practice’s brand:

- Have strong role models for all members of the practice team to emulate.
- Are more likely to correct aberrant behavior in its earliest stages.
- Are more likely to have a culture of high expectations and self-autonomy, rather than a ‘when the cat’s away, the mice will play’ culture.
- Actively and passively demonstrate to employees how to behave every day.

Written mission statements are designed to help a practice more clearly articulate what they are all about, but the real illumination of a practice’s identity comes in the day-to-day activity of its employees, especially its leaders, and most importantly, the business’s owners. You may have no idea where your written mission statement is, or even if you have one, yet many of you have great cultures. That’s because you and the rest of your team have figured out a way to live your mission statement. You live it in the way you interact with one another, and the way that you interact with clients and pets. More power to you, but you shouldn’t discredit the discovery and learning potential of writing a mission. Trying to articulate, in writing, what you believe to be the meaning of your actions, can be an extremely impactful exercise.

Leaders that fully understand and are committed to the practice’s mission are better at coaching and more clear when they give direction. They spend less time meeting with employees talking about trivial policy infractions.

Hold out for the right hire
A big reason why we have problem employees is because we’ve hired problem people. Managers need to drill down into generic job descriptions and ask themselves if the documents really define the person that the practice needs to hire. When building hiring screening tools, ask questions that filter for the people that will be right for your culture, not just people who have the skills to work in a veterinary practice. Take your questions digital to ensure a more consistent interview process and to reduce the elimination of bias.

Don’t sweat the small stuff
In my experience, most new hires don’t blow us away with their first few weeks of employment. Where we saw only wonderful qualities during the interview (probably because that’s what we wanted to see), we now see only gaping performance holes.

In the many years that I have been hiring, coaching, and working with people, what is most striking is how different we are in the way that we solve problems, approach our jobs, understand ‘work ethic’, and undertake our work. It’s been a frustrating ride. ‘What are they doing? Why are they taking so long? Why can’t they just do it like I asked them to do it??’

It’s likely that you have been exposed to a ‘communication style’ class in which you were taught that individuals can be categorized by how they express themselves, but these tools aren’t just Rosetta stones for how people communicate; they also teach us that individuals have different ways of thinking, feeling, problem solving, and reacting to the world. Everyone should expose themselves to a class where communication styles are discussed, not just so that you can communicate more effectively, but so that
you can be more accepting of the different ways people undertake learning, doing work, prioritizing work, handling workload, and so forth.

That’s not to say that I’m advocating for a freewheeling, do-what–you-feel-like workplace. There can be no debate over certain medical protocols, but like all things, balance is key. Beneath blanket statements like ‘Millennials don’t work the way I was taught’, or ‘He just doesn’t have the same work ethic as me’, one should look for shared core values and a strong interest in moving the company forward as the true test for whether an individual should or should not remain in your employ.

**Creative management**

Once, I was at a conference discussing hiring and firing. An attendee raised his hand and instructed the entire room of 200 how HR should be handled. Where he worked, there was none of this namby-pamby business of coddling marginal performers. If you infractions the employee manual, HR called you into the office and directed you to draft your own Personal Improvement Plan orPIP. You were given a week to draft it, asked to return to HR to discuss it, and then expected to live up to what it said. Employees were allowed three loops on this circuit before they were terminated.

When the man finished his story, the rest of us in the room sat in a kind of shamed silence. How could it be that this person’s workplace was so together while the rest of ours was awash in exceptions-to-the-rule, kooks, personality quirks, and so forth? Haltingly I asked him to tell me the name of his hospital and he replied, “Oh I don’t work in veterinary medicine. I’m telling you how they did it when I worked for Sunoco.” The room burst into laughter.

I think it’s interesting that we laughed. Were we somehow exonerated because this guy didn’t work in veterinary medicine? Did our laughter acknowledge that we believe we can’t have an orderly system for hiring, firing, and managing employees, because collectively, we believe that veterinary professionals are too odd a group to be managed so rigidly?

I think part of our problems is that we have adopted a classic approach to discipline from companies that do not have as wide of a spectrum of employees working for them as we do. At our practices, we have 60-year olds sitting next to 20-year olds; high school students in management positions presumably having authority over doctors of veterinary medicine; formally trained nurses working with others who have never before set foot in a veterinary practice; and volunteers. In short, are there other companies that you can think of where such a broad range of educational backgrounds, emotional intelligence levels, socio-economic backgrounds, training, and experience all converge under one roof to get a job done together?

We’re also very short on applicants. If you are hired to work for Apple and you don’t meet their expectations within the first few weeks, you are gone. Same for great human hospitals like New York Presbyterian and the Mayo Clinic. They can afford rigidly enforced discipline programs because they have a long file of applicants waiting to fill vacant positions, but at our practices, we keep less-than-perfect hires, because we have told ourselves that they’re the best we that we can find… and I think on some level, there’s truth to that statement. I believe managing employees at veterinary hospitals requires more creativity than one might otherwise employ if one were managing at other businesses because we’re managing a wider spectrum of ages, personalities, education levels, and degrees of experience and because we have a major shortage of qualified candidates.

To be frank, I have sometimes tolerated employee infractions against our office policies. I have tolerated tardiness, call outs, and gossip to some degree because opposing it wasn’t worth the effort. I picked my battles because too often my one-size-fits all disciplinary policy still wasn’t big enough to accommodate the wide variety of individuals that worked for me. It was just not always possible to square reality with policy. I got creative. Instead of discipline, I often defaulted to coaching… with varying degrees of success. In the short term, I had mixed luck, but in the long term, I built trust with my employees and got many of them to give me a great day’s work… on a sufficient number of days. If there was anything I was firm about, it was our culture and our mission. On those points, I drew a strong line in the sand and the tactic of holding employees really accountable for just two, very important goals, gave these goals real resonance. A strong conviction about what we had to be, both as a practice and as a team, helped me to coach more effectively and to create a grave enough threat of termination that kept people respectful of one another and the quality of our work good despite our many differences.

**Revisit your employee manual**

Think hard about what your employee manual says about corrective actions and grounds for termination. I advocate for strong language against overt hostility or unkindness to clients, coworkers and patients, but ensure that your team members are equipped to handle all the problems that can arise in this area. For example, what if two team members have had a simmering hostility towards one another for weeks, months or years? Not everyone gets along and changing one’s negative feelings towards another isn’t easy. A hospital policy of ‘work it out between yourselves’ sounds great on paper, but how many of us have taken it upon ourselves to tap our enemy on the shoulder, invite them to the nearby donut shop, and work things out over coffee? Animosity can have deep roots. A young team member may simply not have the life experience that equips her with how to manage workplace animosity by herself. Help your employees to stay out of trouble, by keeping an eye out for how everyone interacts and then intercede before things get out of hand.
**Terminate effectively**

Before making the decision to terminate, talk to your management peers about your decision. Make sure that you have a list of things you’ll need from the employee upon termination. It’s also a good idea to have a written handout that lists any generic information you want to provide them with respect to final paycheck, COBRA and unemployment benefits. Have your attorney review this for accuracy.

Termination should never be a surprise (unless, of course, the employee was caught on camera raiding the drug box or the cash register). You should be compassionate, but strong and convicted. Keep the discussion short and firm. “Bash, we’ve had a number of conversations about your performance. It’s not working out and I’ve decided to terminate your employment.” Always have a second party on hand during the termination process and write a description of what happened in your employee journal.

Give the employee time to talk if needed. Listen compassionately, but do not engage in a debate. Listen, acknowledge and then repeat, “I’m sorry Bash, but the decision has been made and it’s not reversible.”

Be cognizant of the shame that Bash will feel. Choosing a time to terminate him when there are fewer employees in the building is better. Escort him to his locker whilst he cleans it out. Terminating employees on a Monday is better than at the end of the week, because the employee can immediately begin applying for work the following day. Pursuing new employment will help the employee move past the shame, disappointment and anger over termination. Terminating an employee on a Friday means that they’ll have the whole weekend to stew on it and to grow more upset.

Don’t be a jerk. While no one likes to get terminated, nearly everyone who believes they were treated fairly during the process will accept termination. Those that believe they were treated poorly are more likely to challenge their termination legally.
Workshop: How to Get 5-Star Yelp (and Google and Facebook and...) Reviews
Sarah Wooten, DVM
Sheep Draw Veterinary Hospital
Greeley, CO

The Numbers On Review Influence: Check out the following results from different market studies on how consumers rely on reviews for making purchasing decisions:

- 67% of consumers are influenced by reviews found online.
- 54.7% consider online reviews in their purchase decision.
- 59.2% is the risk level of losing a customer if 3 negative reviews appear.
- Today’s consumer trusts peer reviews more than traditional advertising.

The data is in and the results are definitive. Reviews are the driving force behind purchasing decisions. The take-away from the data is that reviews matter, and are the driving force behind purchasing decisions. Now more than ever, keeping reviews positive and developing a marketing strategy to minimize the damage of negative reviews is critical to business success. So now what?

If you haven’t been paying much attention to the reputation you’ve built online, there is no time like the present to start. While we know that the majority of our clients are satisfied with our service, there are always the few that are not, and if a disgruntled client feels like you are not approachable in resolving their concerns, then they tend to vent on social media and ruin all the good reviews that you have worked so hard to attain. How good do you look online? This workshop is designed to give you tools you need to take control of your online image.

Objectives
- Review results of 3 areas of client service needs from the BIAH survey from 2017
- Attain a broad awareness of the top ten reasons why veterinary hospitals receive one star yelp reviews
- Develop a strategy to address the reasons behind 1 star reviews
- Attain a broad awareness of the top ten reasons why veterinary hospitals receive 5 star reviews
- Develop strategies to implement these reasons for 5 star reviews in your practice
20 Lessons Learned from the Veterinary Confessionals Project

Hilal Dogan, DVM, CCTP
The Veterinary Confessionals Project
Pala, HI

1. The qualm of People vs. Animals (Pet parents vs. human parents)
2. People piss us off. Especially clients.
3. Millennials vs. GenXers, Baby Boomers and every other generational currently inhabiting earth.
4. Workplace bullies, we don’t need them anymore.
5. The disillusionment of the veterinary dream. One of the biggest fallacies is that veterinarians are well loved and getting into vet school is the hardest part.
6. Finding your voice and standing your ground: Just give me the antibiotics lady! Can’t I just get the steroid shot?
7. The last 30 minutes of a shift "when everybody wants to go home" and the “I don't wannaa work anymore!” tantrums that compromise patient care and create hostility between coworkers.
8. The student loan debt struggle is real. Can we find a way out?
9. Love and throat punches; Practice managers and bosses: It is your circus and these are your monkeys.
10. Why are you really waiting to have children? Exploring fear based motivations versus false beliefs.
11. Why can’t we just get home on time? Is the work life balance a myth?
12. Who is your hero? Did James Herriot let you down? Time to be your own hero.
13. We aren’t skilled at setting boundaries, instead we put up walls. Finding your way starts by being honest with yourself.
14. Crazy stories from the trenches, and you thought you had heard them all.
15. Suicide, or idealization of suicide; can you find your way back to health?
16. Building Resilience ...this is not about being optimistic. Optimism will kill you. Build community and tell the truth.
17. What is your fudge factor? Ethics and ethical principles in vet med. How much can you cheat, break rules and think you're still a good person? Big cheaters vs. small cheaters. Self deception likes to ride the optimism bus.
18. Passion is not a plan, it’s a feeling and feelings change. Having to have everything you do fit into this passion mindset is unrealistic and elitist.
19. Are you a victim or are you just playing a role? How to face the truth of the matter.
20. Locus of control, do we know how to use it to our advantage?

1 secret to represent each topic and one solution per topic. Audience participation requested but not required.
How to Recognize You’re Toxic and Other Signs
You’re Your Own Worst Enemy

Hilal Dogan, DVM, CCTP
The Veterinary Confessionals Project
Pala, HI

Self-awareness is key
If you have done any reading on emotional intelligence you will find that the first step is developing your sense of self-awareness.

If you are wondering if you may be toxic at work…here are some important signs to consider, do a self-check and then ask other co-workers for an evaluation to check against your answers.

1. Do you have difficulty taking responsibility for your mistakes? The cardinal sign of a toxic person is they blame others even though they were responsible for why things went wrong, or at least partially responsible.
2. Do you have trouble stopping yourself from gossiping, especially when it involves putting other people down? Especially, if it’s a new employee or a client you don’t agree with. Especially with clients, do you scurry to the back to talk smack about them?
3. Do have an intense need to be right, even if that means resorting to cruelty? Do you slam drawers and doors, or slam equipment down when you don’t get your way, even resort to throwing instruments?
4. Do you tend to focus on and dwell in the negative. Are constantly complaining? For instance if someone didn’t put something back where it belongs you go around announcing it out loud to everyone, and then saying “If people just put things back where they belong, my life would be so much easier.”
5. Do you dominate the conversations and speak over others even when your opinion has not been asked? Do people feel more at peace and that it is quieter when you are not at work?
6. Do you take everything as a personal attack on you, or think everything is about you? Are you professional? When it comes to resolving conflict between you and a fellow co-worker or do you resort to scare tactics and aggression?
7. Do you treat yourself like a victim in every situation without trying to see the other persons perspective?
8. Do you overreact emotionally when you feel defensive?

During this session we will explore the concepts that arise in these questions. The key to understanding if you or someone else is toxic is to discern between what is a healthy response in a situation vs. what is unhealthy (toxic). Also there are times where all of us have acted like this, especially before we learned how to manage our emotions and communicate effectively. Just because you have some toxic behaviors does not mean you are doomed. The good news is that it can be turned around. However, it starts with recognizing where your weaknesses are, where your toxic or self-sabotaging behaviors are, and then working on it daily. There will be some days you may resort back to old behaviors, and fall off the band wagon, but like a muscle that needs resistance, it will only make you stronger and more resilient in the long run.
Attracting and keeping clients is always a challenge. Focusing on giving them what they want vs. driving them crazy with what they don’t want makes the difference in whether they go or stay.

While the overall growth in revenue and visits in 2016 and 2017 was good to see, there are still some concerning trends. The first is the slowing rate of growth in client visits in 2017 reported in the VHMA Insiders’ Insights report; this data is pulled directly from the PIMS of the ~600 practices involved in the study. Related to this is a decline in both cat and dog visits over the period 2011-2016 reported by the AVMA; this information comes from a pet owner study performed by the organization. The second issue and perhaps the most alarming one, has to do with new clients—according to the Insiders’ Insights survey, new client numbers have declined in almost every month during the last three years and by significant amounts.

What do clients want?
There have also been a number of studies done in the last few years that provide some insights into what clients want; perhaps one of the most intriguing was released several years ago by Banfield. In the past few years Banfield has harnessed the power of their incredibly large data base and published several reports documenting a decline in pet health. In 2015, they did something a little different; they focused on what pet owners are saying about their pets and their related needs. But instead of doing this via a traditional survey of pet owners, they took to the internet and spent over a year reviewing 2 million plus online conversations posted to blogs, forums and other social communities with the idea that in this kind of environment, pet owners would be more likely to talk about what they really think about their relationship with their veterinarian and their needs related to pet care.

Several themes bubbled to the surface in this analysis including:

- Preventive care to a veterinarian means vaccines, spay/neuter and parasite control whereas preventive care to pet owners includes diet, exercise, play and emotional well-being
- Interactions with veterinarians aren’t focused on the overall wellbeing and health of the pet and are “transactional” in nature—they just focus on specific services
- Because pet owners don’t get everything they want from veterinarians, they turn to others for this information—daycare providers, groomers, boarders, breeders, and trainers
- Areas pet owners most commonly look for more information in are behavior, breeds and genetics, food/nutrition/diet and health concerns

A practice’s relationship with clients is built step by step each time a pet owner visits and each time a pet owner talks about that visit to others whether in person or online. The practices with the strongest reputations and those that do best from a business standpoint don’t just provide services and sell products to clients; they also focus on building real relationships between clients, veterinarians and team members.

What do practice teams need to focus on to build and reinforce the bond with clients? And what do we need to stop doing that drives clients crazy?

Driving clients crazy mistake #1: Ignoring the cost of veterinary care
The cost of owning a pet and the cost of veterinary care are recurring themes in studies of pet owners and one of the most important issues practices need to focus on. Clients are dealing with the increasing costs of veterinary care resulting from the availability of more sophisticated medical options, the extended life span of pets which results in more routine care spending as well as an increased likelihood of the pet developing a serious and/or chronic disease and fee increases well above the rate of inflation. Even clients who are fully committed to providing quality care are pushing back at cost and looking for alternatives. It’s not reasonable to think that we can just give veterinary care away; running a veterinary hospital isn’t cheap and veterinarians and their staff have the same need to earn a good living as anyone else. But there are things we can do to make it easier for clients to take care of their pets; these include:

- Use a well thought out price-setting process that focuses on value to the consumer
- Understand who your customers are, what their financial position is and what they want from their veterinary practice
- Better educate pet owners about payment alternatives offered in the practice
Driving clients crazy mistake #2: Not giving pet owners the information they want
Research has shown that the information that pet owners want and the items the veterinary team want to discuss are often different. If pet owners don’t get the information they need and want from the veterinary team in your practice, they will go elsewhere. What is one thing everyone can do to make sure pet owners get their questions answered? Ask! The exact words don’t matter; any of those below will work. Asking, at multiple times during the visit, is what is important:

- What else is going on with Fluffy that you have questions about?
- Did you have any questions about what we have discussed?
- What other concerns do you have?
- Are there any other questions you have?
- Can I do anything else for you?

There is also research that shows that clients will change their behavior more readily if the team focuses on the client’s ability to cope with the new recommendation the veterinary team is making, rather than focus on the severity (fear) of the risk driving the recommendation. In other words, statistical probability of risk, while important to know, is usually not enough to motivate clients to change from the known and familiar course of action. Clients need to know that the recommendation made by the veterinary team will work; that they can do their part (i.e. give a pill if that is required); and that the recommendation is affordably priced or that it can be financed through a third party.

Driving clients crazy mistake #3: Making generic recommendations
Pet owners want care recommendations that are personalized to their situation. They don’t just have “a” dog as a companion; they have “this” dog that lives “this” lifestyle and they want their conversation with the veterinary team to recognize that. While many of the recommendations the practice team makes may be the same for most dogs or cats, there are some that should be different based on breed and pet lifestyle considerations. And even if the recommendations aren’t different, the pet owner still wants to know that you have thought through what is best for this particular pet. Actions you can take to personalize the discussion:

- Collect information about the pet’s lifestyle via questionnaire before meeting with the pet and pet owner
- Reference the pet’s breed and age when discussing your recommendations
- Discuss potential problems a pet of this breed or this age may encounter
- Provide breed and age related information on your website and in handouts
- Use breed and age related guidelines to customize your recommendations and standards in the practice
- Ask the client what questions and concerns they have and give them a chance to think and respond

Driving clients crazy mistake #4: Too many nonsensical policies

- “It would be better if you didn’t visit while Fluffy is in the hospital. We’ll see you on the day after tomorrow when Fluffy’s ready to go home.”
- “We’d prefer you don’t leave Fluffy’s blanket and toy mouse here. We don’t want to lose them.”
- “It’s not our policy to release boarders on Sunday.”
- “You’ll have to come by and pick up that prescription; I know its way out of your way but we can’t mail it to you.”

Every one of the above comments tells clients what you won’t do for them. And each of these client requests is very reasonable compared to what they see in other well-run businesses. Practices need to focus on providing to pet owners what pet owners want, not what we want them to want.

Driving clients crazy mistake #5: Poor client service
Client service is a joke in many businesses these days. Customers put up with it because sometimes they don’t have many options but they silently (or not so silently) hate it and as soon as they have an alternative, they vote with their wallet. In veterinary medicine these days, clients have many alternatives.

Pet owners, like everyone else these days, are busy and stressed. They want their experience at the veterinary practice to be easy, relatively quick, be reasonably priced in comparison to the value and not leave them cross and annoyed at the end because of some expectation that went unfulfilled.

Client service is particularly important because pet owners generally can’t judge the quality of the medical care so service is not only something they judge on its own but it influences how they feel about the quality of the medicine. A very interesting article (“Clueing in Customers”—Berry & Bendapudi-2003) in the Harvard Business Review about the Mayo Clinic reiterates this point. The authors said: “When a company’s offerings are hard to judge, customers look for subtle indicators of quality.” The Mayo Clinic is probably the most recognized brand in the world for quality medicine with very little spent on traditional marketing and advertising. They got there not only by providing great medicine but also by focusing on the client experience, not just the medicine. They recognize that medical care is frightening and complicated and do everything they can to ease the patients concerns. Scheduling is
done around the client’s needs, not the hospital’s needs, the hospitals are attractive and soothing in design and decor, and everyone (janitors, nurses, receptionists, etc.) are trained to understand how the smallest thing they do impacts the patient.

Client service is also about how the pet gets treated and how the pet owner perceives that treatment. Most cats and a surprising number of dogs get very stressed when they visit a veterinary practice. Stressed pets lead to stressed owners and a decline in the number of visits and the care these pets get. Practices need to focus on things that can be done to de-stress the visit both for pets and their owners; some examples include: separate entrances or waiting areas for cat owners and dog owners, a calming environment, “cat only” appointment times, house-calls, a greater use of sedatives before and during the visit and gentle control techniques.

It’s not enough to just do these things, however; you also need to communicate them to the pet’s owner. Unless you explain it in words of one syllable, the client may not understand or appreciate the efforts. When the receptionist immediately moves Fluffy (a cat) and her owner into an exam room, say “We’re going to put you in an exam room straight away so Fluffy won’t be frightened by the other pets in the reception area. This room is nice and quiet and especially designed for our cat patients.”
Good Discounts vs. Bad Discounts:
Know the Difference
Karen Felsted, DVM, MS, CVPM, CPA
PantheraT Veterinary Management Consulting
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It is not uncommon for 5-10% or even more of total revenue to be lost via discounts or missed charges in a typical practice. Periodic review of discount programs and ongoing medical record audits are essential to managing both discounts and missed charges.

“Discounting” is the deliberate reduction of fees charged to clients from what is stated in the fee schedule. It may be a partial discount or a 100% discount - either way the practice owner, the doctor on the case or a staff member is consciously deciding to reduce the fee for the services that the client received. This isn’t always a bad thing; for example a marketing discount that entices more clients to the practice can more than pay for itself if those clients wouldn’t have visited otherwise. Random discounts, however, are another story—these are the discounts doctors and team members give for no reasonable purpose.

Missed charges are not deliberate—these are fees that are accidently not charged to clients, generally because the practice doesn’t have appropriate systems in place to catch all the charges or someone just got busy and made a mistake.

While the bottom line impact on profitability is the same with either discounts or missed charges, it is important to understand what is causing the revenue drain because the corrective action is different for each category.

Why is this topic so important? First of all because both discounts and missed charges can have a huge impact on practice profitability. Secondly it takes a disproportionate number of additional appointments to make up for the profits lost through either a discount or a charge that is not entered into a client invoice. And, of course, profits are what allow a practice to invest in new equipment and improved facilities, offer raises and additional benefits to team members, and provide better pet care.

According to the most recent versions of Benchmarks: A Study of Well-Managed Practices and the AAHA Financial and Productivity Pulsepoints, discounts run about 2% in a practice. This may not seem to be such a large number but it is critical to remember that the total amount of discounts in many practices is often significantly higher because most discounts are not entered as such on the client invoice and thus are not tracked by the software system.

For example, if a doctor gives a free nail trim to a client and doesn’t show it on the invoice, the software system won’t pick it up. Only if the service and the related discount are listed on the invoice as shown below, will the discount figures from the practice management system be accurate:

<table>
<thead>
<tr>
<th>Service</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBC</td>
<td>$50</td>
</tr>
<tr>
<td>Discount</td>
<td>$25</td>
</tr>
<tr>
<td>Net fee</td>
<td>$25</td>
</tr>
</tbody>
</table>

If the fee for this service was just changed in the computer for this particular invoice from $50 to $25 or entered on the invoice as a miscellaneous or onetime charge of $25, no discount will be tracked by the system. And, of course, if a charge is accidently left off the invoice entirely, the software system can’t capture it as a missed charge.

In reality, the amount of discounts and missed charges in a typical practice is often much higher than the 2.0% figure discussed above.

A medical record audit is the only way to insure a practice finds all discounts not recorded in the invoicing software and all missed charges. To start, pull the medical records for 40 cases per doctor—this should be a mix of initial appointments, rechecks, and hospitalized cases. Compare the services provided to the client as documented in the medical record with what was charged on the invoice. Capture the results of the audit on a chart or a spreadsheet with the following items included: patient name, date and time of appointment, amount of invoice, type of appointment (surgery, hospitalization, outpatient), doctor, date, nurse (if known), receptionist (if known) and information about the discounted or missed charges (procedure performed, correct fee, actual fee charged, amount missed or discounted and whether or not a discount was part of a formal discount program the practice offers). Once the amounts discounted or lost are calculated for this sample, they can be extrapolated to the total practice revenue for an estimate of the total lost in a month or a year. For example, assume that the medical record audit reveals discounts and missed charges to be the following percent of each doctor’s invoice sample:

- Dr. A 10%
- Dr. B 19%
- Dr. C 2%
- Dr. D 3%
- Dr. E 10%
Multiplying these percentages by each doctor’s total personal production results in an estimate of the total discounts and missed charges:

<table>
<thead>
<tr>
<th>Doctor</th>
<th>Discount Percentage</th>
<th>Estimated Discount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dr. A</td>
<td>10%</td>
<td>$23,100</td>
</tr>
<tr>
<td>Dr. B</td>
<td>19%</td>
<td>$47,500</td>
</tr>
<tr>
<td>Dr. C</td>
<td>2%</td>
<td>$4,900</td>
</tr>
<tr>
<td>Dr. D</td>
<td>3%</td>
<td>$5,940</td>
</tr>
<tr>
<td>Dr. E</td>
<td>10%</td>
<td>$21,100</td>
</tr>
</tbody>
</table>

The total amount lost is $102,540 or 9% of total revenue.

After calculating the total amounts lost, discounts and missed charges should be separated for further analysis.

If the amount lost was a discount, what kind of discount was it? Veterinary practices generally have three kinds of discount programs: employee benefits, marketing programs, and charitable contributions to the community. In addition, they often have a large amount of random, unplanned discounts that doctors or staff give simply because they are uncomfortable with the fee structure or are just nice people. It is critical that the practice periodically review all discounts and make sure they are still accomplishing the intended goal, if there was a particular goal in the first place.

The marketing discounts should first be reviewed. As noted above, discounting is not automatically a bad thing. Many businesses effectively use discounts to bring in clients for a particular service or during a slow time of the day or year. Start with listing the kinds of discount programs being used in the practice. Common ones include those for senior citizens, multiple pets, breeders, and bundled services. Then take a look at each program individually; for example, if the practice gives a free exam to everyone who purchases a puppy at a local pet store in order to build its client base, is this really working? Is the practice actually getting and keeping these people as clients? These clients should be tracked over time to see if they remain with the practice and they brought in enough revenue to warrant the discount. Similar analysis should be made for all other marketing programs. Discounts to clients who would have come in anyways and paid the full amount of the fee obviously don’t make sense as a marketing strategy. However, discounts that bring in clients who wouldn’t have come in anyways or entice them to buy services they wouldn’t have bought otherwise can be very beneficial. All formal discounts offered by the practice should be reviewed periodically and tracked over time to see if they are effective in bringing in new clients and/or enticing old clients to come more frequently or buy more services. If, after a trial period, the discount isn’t accomplishing its goal, it should be phased out.

The next discount category to be reviewed is those given to employees. Employee discounts are a benefit used to attract and keep good quality staff. The questions to be asked when reviewing these discounts are:

- Are the benefits competitive with other practices?
- Are employees abusing the benefit?
- Are employee debtor balances too high?

Whether or not to continue offering these discounts is a question to be considered in a similar fashion to the offering of any other employee benefit.

Some practices give charitable discounts to animal welfare organizations or individuals who provide care to stray animals. The amount of charitable contributions given by a practice is at the discretion of the practice owner. The owners are truly the only people in the practice with the right to determine if those discounts are too much. Owners, of course, must understand the financial impact of these discounts when making that decision.

The last kind of discount is the random discount; that given for no particular reason. These are clearly the BAD discounts. Most practices find a certain number of discounts in the medical record audit that aren’t part of any particular program. Once the random discounts have been identified, it is important to search for patterns. Is the same doctor involved in all of the transactions? The same receptionist? Do these things happen on the same day of the week? Same time of day? Same type of appointment? Patterns will help identify the root of the problem. For example, is one doctor primarily responsible for most of the discounts? Are there certain kinds of fees that are regularly discounted? Once the root cause has been identified, steps can be taken to correct the problem. The corrective action depends on the problem identified.

And of course, the last category in the medical record audit is the missed charges; those items that weren’t deliberately given away but still the client was not charged for them.

What can be done to reduce the amount of random discounts and missed charges?

First of all, review the practice’s policy on the charging of fees and the use of discounts; if there isn’t a policy, one should be written. The policy should very clearly state that employees are expected to charge the fees set by the practice and list the discounts that can be given and who can authorize them as well as any limitations. Practice owners and managers need to share the policy with all team members regularly and make it clear to employees that part of their job is to charge appropriate fees.

Review missed charges and discounts given per doctor or other team member on a regular basis. Use this information to identify holes in the system that need to be plugged or individuals who need to be counseled about too many discounts given.
Often doctors are more inclined to give discounts than non-doctors. If so, designate non-doctors to enter charges into the computer system. Train these employees in the importance of accurate invoices and how to appropriately use the fee schedule.

Simplify the fee schedule so there are fewer choices by reducing the number of service categories and the number of individual services. Use the same prices for services that are essentially the same; for example a short in-patient hospital exam and a short medical recheck exam. If one is priced less than the other, it will inevitably be selected more frequently.

Review miscellaneous charges and overrides on a frequent basis; these may be used to give away services at a price less than that which should be charged.

Use production pay so the doctor’s pay is impacted by any discounts given.

Give associates a discount fund to use at their discretion for needy pets. Any discounts above that amount are paid 100% by the associate.

Educate employees about the importance of profitability and the impact of inappropriate discounts. The ‘business’ of veterinary medicine should be talked about regularly at staff meetings; discuss whether revenue is growing or declining, what the daily breakeven cost is, what issues are impacting the profession and the impact of discounts on the practice’s financial health and the ability of the practice to invest in equipment, raises and employee benefits.

Doctors and other team members often give discounts when they can’t justify to themselves the cost or the value of the services the practice provides. Leadership is critical in helping employees be comfortable with both. It is more difficult to justify high fees if the practice in question has fees that are much higher than others in the community or it is not readily apparent what is better about this practice compared to others. Non-owners are also more accepting of the level of fees charged in the practice if the overall practice is run fairly; for example:

- Employees are paid well and treated well
- Clients are treated well
- The value and quality of the services are apparent
- Employee pay is tied to performance

Owners need to set an example as well. Violating the practices discount policy themselves or talking about the hard times the practice is going through after showing off a brand new expensive car will not gain respect and compliance from employees.

As discussed above missed charges are generally a function of a system breakdown rather than an intent to give away a service. They generally occur because of:

- Staff shortages—either real shortages or poor scheduling
- Lack of systems
- Bad systems
- Incompetent or non-caring employees

Many different kinds of invoicing systems will work well in capturing all fees but each step in the system must have one person specifically accountable for accuracy. Most systems have both manual and electronic steps and everyone, including doctors, must follow the agreed upon rules for medical record documentation and client invoicing. There needs to be a designated person to do a final review and sign-off on each invoice before a client leaves. Some practices designate an employee to review all records within 24-48 hours of the visit and compare actual services performed to those invoiced. Generally, the salary paid to these individuals is more than made up for by the missed charges they identify. When appropriate, clients should be called and told about the mistake and any holes in the system that allowed the missed charge to occur should be reviewed and fixed.

Discounts and missed charges can be a very large drain on the practice’s revenue. Many practices have some systems, policies and rules in place to deter this from happening however, few have done all they need to do. The goal is to see the right thing occur for “every client, every patient, every record, every time.”
What to Do With Out-of-Control Inventory Costs
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PantheraT Veterinary Management Consulting
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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 your profitability and is actually one of the easier things to do well in a practice.

Before discussing why inventory costs might be high, it is important to understand the goals of an effective inventory system. Controlling costs is just one of these goals.

- Maintenance of the smallest quantity of drugs and supplies needed by the practice, procured at the lowest possible cost while providing the practice with everything needed to provide the highest quality care and without incurring stock-outs
- Systems and controls are in place to keep theft and other shrinkage to a minimum, to insure accurate records are kept and that drugs and supplies are available when needed
- Accurate records are readily available in order to evaluate the efficiency of the system and to improve upon it
- System is simple for all to use
- Inventory is well organized within the facility, easy to locate and not vulnerable to theft or misplacement
- Vendor numbers are kept to a minimum
- Vendors selected are reputable, interested in the practice’s success and the success of the profession and provide products necessary within the practice, as well as good service and fair prices
- All medications and products sold to clients are included on their invoice and appropriately charged for
- Inventory is sold to clients before the bill has to be paid (there will be some exceptions to this when good deals present themselves); generally this means inventory needs to turnover once a month
- A reasonable profit is realized on sales

How to know if inventory costs are too high
In order to improve your inventory control, you first must get a handle on the current situation by answering the following questions:

- What is the current or most recent dollar value of inventory? As of what date?
- Does this information come from your balance sheet or the practice information management system (PIMS)? Are either or both of these reports accurate?
- What was the total dollar value of inventory purchases for the past fiscal year and as a percent of gross revenue?
  - Drugs and medical supplies
  - Food
- What was the inventory turnover for the past year? This should be calculated for pharmacy items, food and all inventory combined

Most practices use a cash basis of accounting for internal purposes. This means that when the bill is paid, the expense gets recorded in the financial statements. In order to know what the cost of inventory really is, the expense in the income statement needs to represent what was sold to clients or used in the hospital during the month, quarter or year under review, NOT what was purchased. This means an accrual system of accounting needs to be used and the figure for total inventory shown on the balance sheet MUST equal what is really on the shelves and not some number that is purposefully or accidentally wrong. If the balance sheet is wrong and the Practice Information Management System (PIMS) inventory reports are wrong, then the practice really has no idea what their costs are.

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></th>
<th>Apr</th>
<th>May</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs &amp; medical supplies expense</td>
<td>$30,000</td>
<td>$25,000</td>
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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. The timing of the bill paying must always be considered in expense analysis. A review of the outstanding accounts payable will help. The practice should consider using some of the accrual accounting features available in most accounting software or doing the five or six journal entries necessary to convert cash to accrual every quarter or every year. 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.

**What causes inventory costs to be too high?**

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

Paying too much for product isn’t usually the biggest issue in a practice although those doing the ordering should regularly check product costs across vendors. There must be a balance between the time it takes to price-shop every product and the cost savings. There is also an advantage to working regularly with just 1-2 vendors as long as, on average, their costs are competitive or the practice is getting some other added value that is worth paying more for.

The owners and managers in practices should try to carry as few choices as possible in each product category. It may be necessary to have one or more doctor meetings to talk through these choices and reach a consensus that is a balance between carrying a minimal number of products and insuring the practice has the drugs and supplies necessary to provide optimal care. It is often better to write a prescription for products which are rarely used rather than stock them.

The vast majority of practices can get almost all products within just a few days of ordering; therefore there is no reason to keep vast quantities on the shelves (unless a genuinely good deal was available.) And yet, many practices have several months’ worth of products on the shelves instead of just a couple of weeks. Keeping too much on the shelves as well as product theft and accidental dispensing of product without charging the client are the most common reasons for high costs. All of these problems can be mitigated with better inventory control.

**Good inventory control**

Good inventory control is easy to obtain but it takes a system and the right people administering that system for this to happen. Now it is necessary to define the critical steps in the process and see if these are being done in your hospital at all times.

- Who is in charge of the inventory system?
- Who normally orders the inventory?
- How does the practice determine what is needed?
- How many distributors does the practice regularly order from?
- What, if any, group purchasing arrangements does the practice participate in?
- How frequently are prices checked amongst vendors? Who does this?
- Are purchase orders used to order inventory? If not, is a written list of each item ordered kept?
- Have reorder points been determined? Has the practice determined how much of various products are used in a given month to aid in determining how to order? (This can vary by time of year.)
- When the order is received, is the list of items ordered compared to the items received?
  - Who does this?
  - Is the order list initialed by the person doing the comparison?
  - Who follows up on discrepancies?
  - Are back ordered items tracked?
  - How?
- When supplies are delivered to the practice, is the packing slip or invoice included in the box checked against the items actually received?
  - Who does this?
  - Who follows up on discrepancies?
- Where is the packing slip or invoice placed after it is checked?
- Are the quantities received and item prices entered into the computer after the order is received?
  - When?
  - By whom?
  - Where is the packing slip or invoice put after computer entry?
- Are the prices charged to clients changed when the clinic cost changes?
- Are items included in group codes or packages checked each time an order of the item is received?

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• Who tracks short dated or out of date product?
  o How is this done?
  o Who arranges for the return of product to the vendor?
  o How is this information communicated to the bookkeeper?

• Are packing slips compared to invoices?
  o Who does this?
  o Who follows up on discrepancies?

• Are invoices compared to statements?
  o Who does this?
  o Who follows up on discrepancies?

• Who writes the checks?
• Are the invoices reviewed by the check signer when signing the check
• Who has the authority to sign checks?
• Is there a limit to the amount?
  o What is it?

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. 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.

Good physical control of the inventory is important for several reasons:

- Helps insure inventory is properly stored based on its physical requirements; i.e. temperature and light
- Inventory that is well organized and easy to find makes it easier for you to assess how much is on hand, facilitates keeping track of short-dated product and allows for quicker and more accurate physical counts
- Proper organization and storage is a deterrent against theft and makes it easier to keep track of in-house usage
- Sensible organization facilitates good record-keeping

In general, good physical control requires:

- A locked central storage area with limited access—even here only small quantities of product should be kept
- Small quantities of products kept in exam rooms, pharmacy, lab area and other areas easily accessible to employees
- Empty boxes displayed in public areas

Other inventory control issues to be considered include:

- Frequency with which inventory prices are reviewed
- What is the dispensing charge added to each product sale?
- Is there a minimum charge for each product sale?
- What is the markup for food? Flea & tick products? Heartworm preventative? Other prescription drugs?
- If product is purchased at a special price, is this passed through to the client?
- Are inventories adequately insured?
- How frequently are medical record audits done?
- Are inventory costs as a % of gross revenue reviewed from period to period?
- For what kinds of inventory items are more than one similar product carried?
- Are employee accounts reviewed periodically to assess the reasonableness of purchase quantities?
- Do practice owners take inventory items from the practice without paying for them?
- Are the following reports used from the practice’s inventory computer module? Usage, order history, inventory quantities, reorder points

While setting up and administering this system can seem daunting at first, good inventory is actually one of the easiest things to achieve in a practice.
A Manager’s Guide to Common Client Complaints
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Use client complaints to empower your team members, to bond more closely with clients, and to improve your business’s operations. Manage complaints well and you’ll avoid bad reviews, client loss, and potential lawsuits.

Empathy works
Whether they are thinking clearly or not, angry clients are hurting clients. When dealing with angry clients or clients that have a complaint, never focus on who’s right. Instead, stand in the client’s shoes, understand their frustration, and let them know that you understand how they feel. By agreeing on one thing, in this case that the client’s frustration is understandable, you set a stage where all parties involved can calm down and start to work out differences.

Serve the client, not the computer
Before diving into the details of a client’s concerns, you need to get the facts, but be sure not to put the client in a kind-of virtual holding pattern while you cycle through collecting information like, name, address, phone number and so forth. Making the client serve your needs, before their needs are met, is a client turn off. Get the data, but let the client know that he or she is what really matters. “Mr. Smith, my name is Bash. I don’t believe I’ve ever met you before, but I’m going to solve your problem today. Before we start, may I get some information from you so I have everything I need in place to address your problem?”

Try using the client’s first name
It’s the custom in our industry to address clients by their surname, but the habit puts a formal distance between the client and us. Consider adopting a policy where you ask the client how they would like to be addressed. “Mr. Halow, it’s great to have you at our practice! Some of our clients like when we address them by their first name. Do you prefer Bash or Mr. Halow?” Keep a record of their response by capitalizing their preference in the software.

If both parties use first names during the complaint process, both are inclined to behave more reasonably and respectfully with one another. It humanizes things for both sides.

Invite team members to handle client complaints
It’s a mistake to only allow supervisors to handle client mistakes. Team members, with sufficient tenure and experience, should be given the power to handle client complaints as they see fit. It will make them more mindful of customer service and solid advocates for great service within their peer group. Often, practices don’t allow lay staff to handle client complaints because they are afraid that the team member will botch the response; but if leaders are on hand to assist with the process, team members emerge more empowered and feeling valued. Addressing client complaints helps individuals learn first hand the value of great service and the consequences of when it falters.

Hold meetings about positive client interactions
We have a tendency to spend valuable meeting time talking about client service failures as opposed to successes. Regularly drawing your group’s attention to their best service acts is a positive way of discussing the value of client service, of singling out the team members responsible for the great service, of training new team members, and of creating a sense that the group is responsible for genuinely outstanding accomplishments.

Discuss client complaint procedures in the employee manual
Address how client complaints should be handled in your employee manual. Discuss how you expect team members to behave in the face of potential or direct physical threats or violence. Agree on how one is supposed to alert all staff members of a menacing individual in the practice or of a situation about to go awry.

Three steps to handling all client complaints

Listen
Invite the client to talk to you about their concern. Demonstrate that you are actively listening by making eye contact and nodding your head appropriately. Explain that you would like to take some notes, a sign that you are really taking their concerns seriously.

Acknowledge
Validate their concerns by sharing a story that relates to theirs. “Bash, I completely understand what you mean. I have been in the same situation in the past and I know how you feel. I’m really sorry that this happened. Let’s see how we can fix this.”
Solve the problem
Drawing the client into a pact to resolve the issue with you is the best way to finalize the client complaint process. Lead off with your thoughts on how to set things right. “Bash, I completely understand that you are upset about the blood test that Dr. Cunningham added onto your bill, but as I see by the notes, Dr. Cunningham felt that the test was vital to managing Rye’s diabetes successfully. Can I schedule a time for you to speak to Dr. Cunningham about his decision? If you still have concerns after that discussion, I’ll do whatever I need to do in order to satisfy you with our work.”

“Do whatever I need to do?” Yes. Veterinary practices that are advertising online spend anywhere from 14 to 70 dollars per client lead. The life-time value of a client, when you consider the referrals they provide your practice and the number of pets they own in their lives, has been calculated to be somewhere between 14 and 60 thousand dollars. The average veterinary practice requires 3 to 7 dollars of income per minute just to break even. Will I eat the cost of a lab test to keep a client? Very likely. And if the decision keeps me up too late at night, I’ll call the lab and ask them to help with the cost.

That’s not to say that I won’t look for other ways, besides a refund, to assuage the client’s concerns. Most clients simply want validation that their concern is legitimate. Discounting services, giving back money, or other forms of remuneration can cheapen the complaint process on all kinds of levels. Consider this response to another client’s concerns. “Henry, Dr. Gittelman, our practice owner, is going to hear about this. What you went through with your cat, Oleander, is the last thing that he wants for any of the clients that come to our practice. I can personally assure you that he will hear about your concerns and I’m sure they will be addressed at a company-wide meeting. All of us work hard to do our very best. In your case, we failed you. I am honestly very sorry about that.”

Dealing with escalation
When clients threaten to write a bad online review
None of us like to be strong-armed with the threat of a bad online review. Here are some ways to handle the threat.

Invite the client to your office to discuss their complaint.
A face-to-face meeting can humanize things for both parties and pull the client’s finger off the online review button. Additionally, setting a meeting time delays the discussion and gives everyone a chance to cool down. “Mr. Ludwig, this is Bash, I’ve met you several times and last June, I personally cared for your dog, Salamander, when he was sick. I’m really sorry we failed you. I would like my co-manager, Linda, to hear your story so we can make sure that this sort of thing never happens again. We’ll also be able to figure out what we can do to satisfy all of your concerns. Are you free tomorrow at 4pm?”

Reason with them
While writing a terrible review about a practice feels good in the moment, it does nothing for either party in the long term. Both end up unhappy and with a lingering, unpleasant lack of resolution. Try sharing such reasoning with the client. “I hear that you’re frustrated and that you want to vent online, but I’m here to help you with your problem now. I want to do what I can to help you feel better, get the satisfaction you deserve, and put this behind us.

Know your rights
There are federal laws that prevent online threats and certain kinds of harassment. Individuals can’t write a bad review and then ask for money in order to remove it and they can’t anonymously use the Internet to threaten or harass you. If you are concerned that someone is menacing you or trying to extort you online, consult an attorney. Try this response to anyone who threatens to write something malicious. “Bash, I know that you’re angry and I want to help you, but there are federal laws against certain kinds of online threats and harassment. Our attorney takes any such posts extremely seriously and she will contact legal authorities if she believes the law has been broken. I’m here to help you now. What have we done to upset you can be corrected. Let’s fix it together.”

Bury bad reviews
Sometimes people are just going to vent online and there is nothing you can do to stop them. If you’ve reached out to them, but failed to resolve their issue, let them know you are sorry and ask if they would like to be inactivated in the software. If so, wish them well and let them know that you’ll be happy to transfer their pet’s records whenever they need them. Be courteous and take the high road throughout the conversation. It will serve you in the future. Reach out to your most trusted clients and let them know what happened. Ask them to respond to the review by writing an excellent one, but don’t ask them to get into an online shouting match with the disgruntled client. With luck, the great reviews will bury the bad.

Cultivate an active online community
One of the great things about an active online community through Facebook or other social media channels is that your most beloved clients can come to your rescue whenever someone says something negative. Scroll through the Bangor Humane Society Facebook page for examples of how this group allows client complaints to evolve into resounding endorsements for their practice.

This too shall pass
It is rare (but not unheard of) for bad online reviews to do long term damage. At this point, all of us have lots of experience reading online reviews and we can filter out what does or does not sound reasonable. If an angry client refuses your overtures to make amends and proceeds with drafting a negative review, so be it. You’ll be all right. The review will eventually sink beneath the horizon and you’ll live another day to provide the great service you and your team are so eager to provide.
I'm going to call my attorney
When faced with this threat, reiterate that you are present now to address the client’s concerns and are willing to work things out without further cost to their valuable time and emotions. As mentioned above, try inviting them to a face-to-face meeting on the following day. The added time will allow everyone a chance to cool down. Still, if they persist in this threat, you will be forced to notify your attorney what has happened. Unfortunately, once the attorney card has been played, the fight goes above your pay grade and is now in your lawyer’s hands.

Concerned about a firearm or other avenues of violence
Do not hesitate to call the police if anyone threatens you with bodily harm. Do not be afraid to lock the door and to notify the authorities if a menacing client says that they are on their way to your practice to confront you. Do not expect your staff to defend themselves in the face of a client that threatens to get physical. If you suspect that a client may be on their way to your practice and you are unclear whether or not they will be violent, move more team members to the front of the practice where they can be on hand should another member of your practice team need help.
It’s true. Stress can kill you. I’ve seen it firsthand; but what’s more likely to happen is that stress with kidnap you and steal you away from happiness and fulfillment.

**Primary stress**

There are two kinds of stress: primary and secondary. Primary stress is stress from past or upcoming events. When the event is especially traumatic, one can suffer from Acute Stress Disorder. This is stress that occurs immediately after a traumatic event and can last for weeks. When one’s stress lingers for months or years after the traumatic event, one is described as having Post Traumatic Stress Disorder.

**Secondary stress**

Secondary stress occurs when one is on the sidelines of a stressful occurrence. Compassion Fatigue fits into this category. It occurs when one is placed in environments where he or she is witness to primary trauma on a regular basis and slowly becomes desensitized to it over time.

**Signs of stress**

There are physical, psychological, and behavioral signs that accompany both primary and secondary stress. They are:

- **Physical**
  
  - Exhaustion
  - Somatic symptoms
  - Sleep disorders
  - Decreased sex drive

- **Psychological**
  
  - Emotional exhaustion
  - Decreased empathy and compassion
  - Increased interpersonal issues
  - Increased self doubt
  - Depression
  - Decreased job satisfaction
  - Changes in sensitivity to emotionally charged circumstances
  - The feeling that making a difference is elusive, if not impossible

- **Behavioral**
  
  - Increased social distancing
  - Decreased motivation
  - Increased difficulty making decisions
  - Increased call outs
  - Increased cases of compromised care
  - Being a caretaker in your personal life

**Resilience**

Resilience is when stress teaches us how to think better, to be more accepting of our circumstances, and to live our lives more fully. Resilience is embodied in the expression ‘what doesn’t kill us, makes us stronger’. Obviously, using stressful situations as a pathway to greater happiness and success is ideal.

**Get help managing your stress**

If you believe that you are suffering from any of the above-mentioned signs of stress, don’t try to self diagnose. Seek out the opinion of an experienced psychological counselor for a professional diagnosis and a treatment plan.

**Reduce opportunities for stress in the workplace**

In my role, I get to visit dozens of veterinary practices every year. What is most striking to me is how little opportunity our business model provides us to succeed. In too many veterinary offices, workers are trying to be all things to all clients at all times. In the interest of best customer service, of trying to demonstrate that we care, of trying to underline our value, and of growing the business and paying the bills, employees are pushed to throw themselves headlong into customer service without benefit of adequate planning.
tools, support, and training. In our line of work, we’ll never free ourselves of the death, suffering, and heartbreak we are witness to, but there are changes that we can make to the way that we work that can reduce other sources of stress.

**Review workflow**

Meet as a team to discuss how clients and patients move through the practice. Challenge workflow paradigms that regularly fail like the surgery admission process and patient pick up crunch times. Ask yourself whether a solution of ‘work harder and faster’ is really a solution or merely avoiding the question.

**Huddle**

Help employees function as a team by reviewing the daily appointment schedule together and sketching out a game plan. Practices where employees huddle in the morning and afternoon prior to appointments provide better customer service, are more likely to succeed at providing great service, have higher sales, make fewer mistakes, and enjoy their work more than their counterparts who do not. Huddles are ten minutes of planning that provide for 4 hours or more of great performance.

**Ensure that team members have a chance to connect with people and pets**

Remember that your team members are in this line of work to serve, not sell. Ask yourselves whether doctors, nurses, and client care representatives have the time that they need to do what matters most: connect with clients. Look at workflow practices that pull team members away from client interaction and bury them in administrative work. Ensure that the technology you use serves client interaction, not stands in between it. Is there anything more frustrating than waiting for the computer, the app, or the machine to allow for the completion of a transaction? No team member wants to apologize for mechanics that don’t work and no client wants to wait until such things are fixed.

**Allow for qualitative socialization**

Provide a clean, comfortable place for your team members to sit, talk, eat and socialize together. A recent visit to a practice introduced me to the concept of a ‘Walton’s table’. Named after the 70’s television show, not the retail storeowners, the table allows for all team members to sit together, eat, and talk. The discussion is freewheeling and steers toward practice-problem-solving as much as it does towards children, hobbies, gardening and current events. Common spaces in practices allow disparate individuals to become a community.

**Focus on the wins, not the losses**

Think about the feedback we provide one another on a daily basis and ask if it’s hurting or harming the situation. In my experience we meet when something goes wrong, rehash it, and try to find a way to stop it from happening in the future. I believe these meetings are demoralizing for everyone involved and seldom provide any lasting way to end errors. The best way to limit errors is to inspire vigilance in all team members and to get them to think like owners. To this end, empower team members to publicly share their stories of success. Review the reasons why you succeeded, not failed. Train your employees like you would train any animal in your care: with support, trust, and positive reinforcement.

**Keep the workplace safe**

Be a steward of a safe workplace culture. Take a strong, immovable stand against behavior that is threatening, undermining, belittling, unkind, harassing and so forth. Early in my career, I turned a blind eye to the bad behavior from some very valuable, hard-to-replace techs and doctors because I believed that if I corrected them, they would quit. That was a mistake.

**Help employees to be objective about things**

Lastly help your employees discover flaws in the way they process information. In general we tend to fear the future, when in fact we have a lifetime of evidence that we cope just fine with the unknowns that lie ahead. We have a tendency to believe all or nothing, to think in absolutes, when the world is actually a variety of greys. We perceive ourselves as isolated: in our failures, our fears, our frustrations, and in our perception of things; but on a bigger scale, we mostly feel, think and hope the same as everybody else.

Be someone who listens. Be a levelheaded, respectful and honest friend. Sometimes all it takes to help another individual cope with their present situation is to let them know that you earnestly care.
Manage to the Future: How Stagnation Kills a Hospital
Bash Halow, CVT, CVPM
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Stagnation drags on office morale, risks the business’s long-term viability, leads to a less healthy work force, decreases the quality of patient and client care, increases the risk of injury on the job, and whacks job satisfaction in the knees. Identify stagnation in your office and in your employees and learn what you can do as a leader to avoid or emerge from it.

Signs of stagnation in the organization

Disproportionate focus on profit
Practices that regularly benchmark success by measuring financial growth risk alienating the team members from what they really care about at work: interacting happily with co-workers, connecting with clients, feeling magnanimous, and comforting and healing pets. Whenever I do surveys with practice teams to find out what makes them happy, no one ever lists ‘make more money for the practice’. Invariably the things that motivate team members are the things that I listed above: feel like one is part of a team, feel singled out as great at what one does, accomplishing one’s work thoughtfully without excess distraction, and connecting with clients. Use financial benchmarks only as a starting point for conversations on how all of us can do what is most meaningful.

Fewer group meetings and new initiatives
Groups that are invested in changing enjoy getting together and talking about their progress and success. Practices that don’t have regular, purposeful meetings are probably suffering from some degree of stagnation.

Leaders interact less
All members of leadership should enjoy working together to advance the practice. They should look forward to it! Practices in stagnation often have leaders that are compartmentalized into their own microcosms of responsibilities and duties.

Clutter
Clutter is an outward sign of a decrease in morale and focus within the team, two big symptoms of stagnation. Sometimes organizing a hospital-wide clean up initiative is sufficient to shock the practice into life again, but the cause of stagnation is much deeper than office cleanliness and until the bigger issue is addressed, the office will soon refill with mess.

Increase tardiness, absences, missed deadlines, and a decrease in follow-through
Stagnation weighs on your team’s spirit. More employees arrive late to work or call out because work is no longer as rewarding and enjoyable as it once was. Leaders don’t follow through and miss deadlines because leading stagnant practices is like trying to fan a fire that won’t light.

Decrease in new initiatives
Active practices are regularly exploring new techniques, revisiting old protocols, and engaged in ongoing discussions about what’s working and what is not. Stagnant practices are often doing the same things that they have been doing for years. Recent changes in the way people find practices, shop for their veterinary services, pay for their services, and learn about animal health are so radically different from what they were 5 years ago, that it would be impossible to run a business successfully that hasn’t undertaken some new approach in this area…unless of course, the business is stagnant.

Individual signs of stagnation
Stagnant individuals in prominent positions within the practice can sink the whole business into stagnation. Look for these signs of stagnation in either yourself or the other more visible members of your practice team.

Decrease in health, work-life balance, and personal appearance
Stagnant employees are often depressed, either independent of their work or because of it. Signs of depression in individuals can include weight gain; increased consumption of drugs, alcohol and tobacco products; a decrease in sociability; a decreased attention to personal appearance; and more disorder in their workspace (unfinished work, clutter, etc.)

Just getting by
Stagnant employees offer no discretionary effort. A pen falls on the floor, and they leave it there. They see stool on the sidewalk on their way into the practice, and they fail to address it, either themselves or with other team members. Work for these employees feels twice as long as it does for team members who are engaged. They have no interest in CE and it’s been months since they’ve been interested in learning something new.

Pushed out of the picture
Happy workers and organizations tend to marginalize stagnant individuals. They aren’t included in meetings or their absence from meetings is tolerated…probably because they are secretly not wanted. If the organization is planning for the future, stagnant employees aren’t accounted for in the picture. Employees have a diminished sense of their importance in the practice and the employee herself starts to feel increasingly invisible.
What to do
Here are some ideas on what you can do to bring back momentum to your business

Jumpstart a discussion on the value of what you do
Stagnant organizations have lost focus on what really counts: connecting with clients, healing pets, making ill pets feel comfortable, and having a great time while working together. During the lecture, I’ll introduce you to an exercise you can conduct with your team to explore workplace satisfaction. You can use it to begin a discussion on where you are now with your happiness and what you can do to improve upon things. Another helpful conversation starter is the practice’s mission statement. All mission statements should be revisited from time to time. Practice teams should challenge the language within their mission; ask if the language conveys what they are really all about or if it’s merely hyperbole. Many organizations have something they call a BHAG, a Big Hairy Audacious Goal. Being part of an organization that’s out to do something life changing is stimulating. At the time of this writing, Florida high-school students are mobilizing an offensive against the gun lobby. It’s probably the biggest and most effective to date. These students are passionately pursuing their goal because they see it as a reflection of their own power, their merit as individuals, and because they believe their efforts will change all of America.

Engage an outsider to facilitate a strategic planning meeting
Bringing someone into the practice with an outside perspective can be especially stimulating to everyone. Combining the visit with an overall assessment of the practice can lead to all kinds of ideas on how to improve. Typically these sorts of meetings can be sponsored by the drug companies with whom you do business or by distributors. Talk to your representative and see if this is something they can do for you.

Understand that most people naturally fear change
Your organization may be stagnant because the leaders or workers within it fear change. A fear of change is normal, but change is also natural. To ignore change is to live in a bubble outside of what’s happening in the real world. Certainly within our own industry, change is happening so rapidly that any organization or individual that chooses to remain stagnant in the face of it is committing business or career suicide. I have produced an hour-long webinar on change that is available through the Fetch dvm360 website and comes with 1 hour of CE credit. Invite team members who are fearful of change to watch it. It will be helpful.

Fire the deadwood
Organizations can have four kinds of employees
- Learners: Those new to the organization who have yet to realize their full potential
- Stars: Members of the organization that are engaged in their work, exceed expectations, and enjoy giving things their all
- Loyal Citizens: Trustworthy, reliable, solid employees
- Deadwood: Stagnant employees who are just getting by in their work. These team members are disengaged from their work and their co-workers and are often times depressed.

The deadwood part of any organization is a drag on the organization and the people in it. Terminating the deadwood can be a stimulating sign to the rest of the crew that change is sincerely underway. Follow the rules for termination as discussed in my other lecture on the topic included in the Fetch dvm360, 2018 educational line up.

Identify if you are the problem
One stagnant manager, practice owner, or other prominent member of the group can hold back the whole organization. Ask a trusted colleague if you might be the reason the business is stagnant. If the answer is yes, try these solutions:

Join a group
Local associations and local CE events can be a great way for you to meet others who are engaged in the same kind of work as you. Talking with like-minded folk can be stimulating and help you to emerge from your funk.

Discover new opportunities
Today’s veterinary world is a seller’s market. There may be practice sales or partnership opportunities that significantly increase your wealth and the kinds of options that are available to you for daily work. As part of a group of practices or inside a partnership, you will have new colleagues to work with, new challenges, and new goals. It will be a new, invigorating chapter in your life.

See a therapist
Stagnation can be rooted in depression, the accumulation of years of unresolved frustrations and setbacks, or poor thinking patterns. Psychotherapy is excellent at helping people emerge from depression and should be part of every healthy person’s regular medical care. Stress, depression, anxiety, and nonproductive thinking patterns compromise one’s quality of life and can significantly impact one’s physical health. Just a few visits to a psychotherapist can be wonderfully cathartic, stimulating and helpful in jarring one out of a stagnant life pattern.

The era in which we are living will be known as the Internet of Things. The Internet, and technology in general, are rapidly changing the way we do everything in our lives. The veterinary business world is changing too. Remaining stagnant while the rest of the world and industry change so rapidly will drag on your happiness and kill the long-term prospects of your business and your career. Addressing stagnation and embracing change will ironically give you more of a sense of control and a greater appreciation for
your personal strengths and who you are as an individual. Start today or find the help you need to change with the Fetch dvm360 resources.
After more than 20 years of watching veterinary professionals fail, stumble, and succeed, I’ve come to understand that success isn’t predicated on luck, timing, or a winning lottery ticket. It’s predicated on you. Here are the ways we can best influence our success in our jobs and in our lives.

Rethink your outlook on life
Each day the sun rises on 7 billion people and each day it casts the same light and the same shadows on us all. To wake up to a world that you think has it out for you, to a day that is bound to be bad, that’s bound to go wrong, isn’t being awake at all, but a continuation of some nightmare that you were having while you were in bed. Burning the toast is just black bread, not a sign from the universe that you’re a terrible person. Ten minutes late for work isn’t an indicator that you are the worst employee ever, but just another reminder that you should wake up earlier.

Many of us live with brains encased in a buzz of anxiety. Like a fog of noise floating atop our grey matter, this anxiety prevents us from seeing what’s really happening around us, from thinking clearly, from enjoying the world that’s unfolding before us, and from living our lives fully.

I often say that if we treated ourselves like the animals that we care for, we’d be far happier and more successful. With animals, we understand that positives work exponentially better than negatives when trying to get them to perform. So why do we allow our inner voices to be so relentlessly negative?

We simply have to stop the internal flow of negative comments. It’s worthless self-flagellation. Negative thinking makes us underperform and ends up being a self-fulfilling prophecy. Studies show that human minds have a more difficult time emerging from negative thought patterns than they do from positive ones. If we don’t guard against negative thoughts, we feel bad longer.

Failure is part of the normal progression forward. If you’re not failing, you’re not learning. Kittens and puppies that wrestle, fall, stumble, and trip are learning to get very good at running and hunting. Reprogram your mind to think of failure as a mild form of play and as an essential part of growth.

Review goals as a group
I’m convinced that practice managers and owners are generally frustrated because they have only a general notion of what they would like accomplished.

As business leaders, we want great patient care, great client service, a good ‘work ethic’ from our employees, and ‘common sense’, but this kind of terminology is just noise unless it is explored more fully and tailored to fit the needs of your branding and the vision of your leadership.

What is ‘common sense’ exactly? What is a good ‘work ethic’? Mission statements, strategic plans, and job descriptions are designed to help you explore the details of your vision and your needs more fully, but we make the mistake of not investing enough time considering what they actually say.

Organizations aren’t ships that need to be steered; they are organisms. Goals and strategy are only part of the whole; the organization must spend time reflecting and thinking its way forward based on the feedback it’s getting from its environment and itself.

Get healthy
A glass of water is better than a can of Coke. A plate of carrots is better than a bag of chips, and unless you’re suffering from some metabolic disorder, twenty pounds lighter is better than 20 pounds too much.

Exercise feels great, so does stretching, and so does enough sleep. Most of the time, our work schedule is unrealistic. Some humans can probably work 10 hours straight, but most of us benefit from a mental and physical break. It’s not lazy; it’s just optimizing the output you get from yourself and from those that work for you.

I know of one practice that takes over their physical therapy room for 30 minutes to an hour each day. They take advantage of the soft flooring to do jumping jacks, stretch, meditate, and do yoga. Not everyone does it. The practice doesn’t shut down. They do it in waves. They do it as they need to, but the retreat is supported by their culture and employees that need to take a break (not to text, but to reboot their bodies and their minds) are given the leeway to do so.

You don’t have to join a gym, you don’t have to run a mile, but you do have to move your body… in the garden, on a walk with your dog, on a bike, on the dance floor, in the ballet class. Physical fitness is essential to optimal thinking, working and living.
Don’t go it alone
I have been to successful practices where there was one all-star vet surrounded by supportive minions, but there was no dream job in it for me. Managers and practice owners that decide that certain things are too important to be done by anyone else end up burned out, lonely, alienated and resentful. These folks don’t work with others, others work for them, and they create a chasm where work satisfaction would otherwise be.

Those that go it alone naturally select for the worst of employees, because people who have the ability to think, want to think, and want to participate, end up taking jobs where they can do just that.

Avoid being alone in your work by focusing on what and how you want done and then hiring individuals that you believe will be competent enough to learn the job themselves and to one day take it over. Give these individuals a chance to fail at the task…several times. Provided that employees are invested in getting it right, the latitude of failure is essential to learning and understanding how to do a job expertly.

Find joy
People who like what they do perform better than those who don’t like what they do.

Try to hire people that you want to work with, not people that you need to hire because you have no other option.

Regularly ask yourself whether you’re happy doing what you are doing. Working a schedule that provides you no break, no opportunity to finish the job well, and that forces you to apologize for service failures over and over again is not a fun job. You and the rest of the team are in your right minds when you recoil from such work. Stop the insanity and fix such workflow issues. Solving the problem collectively will be most effective and satisfying.

Pull back from your work schedule and take the time you need to clear your head, get healthy again, exercise other talents, and enjoy the company of those you love. If cash flow is an issue, hire an experienced business advisor to come to your practice and find easy opportunities to increase sales or to plug leaky expenses.

The things that are holding you back from succeeding at work are likely the things that have held you back your whole life. Stopping your self-sabotaging behavior isn’t just making more money, it’s improving the life of those that work for you, it’s improving the relationships that you have with your spouse and children, and it’s your pathway to a little bit of heaven without having to first kill yourself to get to it.
In my role as a veterinary business advisor, I visit dozens of veterinary practices every year. From the moment I drive into the parking lot, these hospitals talk to me in pictures. Each image is a snapshot of the business’s health. Here are some of the things that I have learned over the years while pondering these practice picture profiles.

**Wear, tear, and dirt**
Practices with dirty entranceway mats and carpets, with marked walls, and with worn furniture are telegraphing to the client that the practice team is stretched too thin, too busy for details, and frayed at the edges. Threadbare chairs start to look normal in a building where hundreds of animals pass regularly, but such furniture makes well-dressed clients cautious to sit down. Allocate a regular part of your budget for the replacement of worn furniture; hire a professional cleaning crew; and wash/repaint your walls regularly. Stop thinking of these expenditures as money down the drain, but as a kind-of ongoing, passive marketing.

**Clutter**
The average American employee spends one week each year looking for stuff at work. One, 4-drawer file cabinet is estimated to cost $25000 to fill and $2100 dollars per year to maintain if you consider the time it takes to create the documents, search for the documents, and replace the documents that you lose. There are numerous studies that show that workers are less productive in cluttered environments and that their morale is lower than those employees that work in clutter-free spaces. Like dirt and scuff, clutter is communicating to both your employees and your clients that you are stretched too thin and that you aren’t fully invested in the game.

**Wall art**
Think of your workspace as living, not as a memorial to the first person that decorated it. Consider the pictures that you have on your walls. Are they generic art pieces that you picked up at Home Goods or are they saying something specific about who you are and what you are about? Practices that have pictures of employees on the walls, along with the employees’ names, introduce team members before they ever meet. They also telegraph that you are a cohesive group of healthcare providers, not just employees. Think twice about signage. Typically it’s curt and strong-toned (payment due upon receipt of services, no cell phones, no checks, etc.) Such signage is typically printed on office paper, worn, and not effective. Posters can also cheapen your practice’s appearance.

**Magazines, kid’s toys, books**
These items are here to engage your client while he or she waits, but many clients have already brought a distraction with them: their pets! In some practices, these sorts of distractions match their culture, but all practices should revisit their value. Certainly if these things are dirty or covered with hair (and this can happen with only 12 hours of neglect) they are counterproductive to your intentions.

**T.V’s and background music**
Practices that have a T.V. droning on in the background are filling the space with a noxious noise. If the program is a news channel, you are probably making your clients anxious. Personally I find paid-for veterinary education programming so sanitized that it ends up being an alien presence in an otherwise familiar, homely environment. New-age music and meditation channels may seem too soporific for your taste, but they do a good job at calming people down and instilling a sense of safety.

**The great wall of reception**
There is a new trend at hotels: guest-registration attendants stand behind podiums, not desks, because they recognize that hotel staffers should have ready access to the clients they want to help. Tall veterinary reception desks that prevent clients from seeing service team members are undermining the practice’s determination to provide friendly, welcoming care. The tall desk becomes a great wall against interaction, warmth, and connection. Consider rebuilding the desk or inviting your team members to stand during their shift so that they are more open to your clients’ needs.

**Your practice is revealing how open you and your team members feel**
‘Practices pictures’ can also reveal how the practice leaders and workers feel. Consider the following.

**Messy break room**
We have so little time to stop, collect ourselves, and recharge. Imagine doing it in a beat up room where the sink is full of dirty dishes, where the interior of the microwave is caked with splattered food, where the chairs are uncomfortable, where the table is sticky and covered with crumbs, and where the refrigerator is a morgue for spoiled food in Tupperware coffins.

Messy break rooms tell me that team members are strained, apathetic, and tired. Eating and resting in squalor is a sign that the team has resigned itself to defeat. Break rooms have a chance to be areas where employees bond, share ideas, and where practice
teams can meet and grow the business, but just don’t go back to your practice and send an order to the break room to be cleaned. Investigate the reasons behind the apathy. Address that first, and the cleanliness, the willingness of the staff to take pride in their workspace, will follow.

**Piles of stuff on desks and workspaces**

I know of one practice consultant that went into a practice owner’s office and completely removed the mountains of papers, bills, journals and other detritus from the owner’s office, leaving a buff, wooden desk surface in his wake. When the owner saw the change, he flipped.

**“What about all the bills that were there! Those weren’t paid!”**

‘Don’t worry,’ the consultant calmly replied, ‘They’ll send more.’

The piles of stuff in your workspace are an outward reflection of the mounting pile of stuff that’s growing in your head. Both your head and your workspace need a good scrubbing out. Get in the habit of closing windows on your computer, tidying up your desk, and making a fresh to-do list before you leave each day. It’s a great way to clear your mind and tee you up for a productive tomorrow.

**Rumpled clothes, messy hair, and physical unfitness**

Happy people are social, care about what they look like, and care about what others think of them. Team members that have stopped thinking about their appearance, that are abusing their bodies with sugar, soda, alcohol, empty carbohydrates, and a lack of exercise are probably suffering from some level of depression.

Life is hard for these individuals, but it’s in everyone’s best interest that they address their self-destructive behavior. Changing one’s diet and starting some form of exercise can provide an immediate bolus of positivism, but long term, these folks should take advantage of the mental health care services that most insurance companies cover. It’s too bad that psychological counseling is still stigmatized as something that only ‘crazy’ people need. In fact, most of us have aberrant thinking patterns that are every bit as commonplace as they are destructive. How you may be getting in your own way is probably no surprise to the average, experienced psychologist. They can help you recognize your destructive thinking patterns, change them, and give you with the priceless gift of genuine happiness.

**See the big picture**

Every 3 months, the whole team should conduct a practice walk-through. Start in the parking lot, and then as a group make your way past the hospital sign, through the entranceway, and into the lobby where all of you can take a seat. Silently, make a list of the things that you notice and what you believe these things are saying to your clients. Afterwards you can share notes and talk about ways to improve.

In the end, it’s all about doing a great job and feeling terrific about your efforts at work. Take a real or mental picture of what your workspace looks like and what you look like in it. It may be just the perspective you need to see the things that are standing in between you and great, satisfying work.
What the Lion King and Trojan Horses Can Teach Us About Motivating Clients
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We assume that common goals bind clients and veterinary professionals together. You want the pet to be healthy. The pet parent wants their pet to be healthy - there shouldn’t be any conflict there, right?

Not so fast.

Even though we share a common goal of health and wellness of the veterinary patient, the perspective gap between what the client thinks is appropriate health care for their pet and what the veterinary professional think is often disparate. According to Judith White, a psychologist at Dartmouth, this divide between the two groups is due to a concept called horizontal hostility, where two groups with shared goals experience in-fight, power struggles, and an ‘us-versus-them’ mentality. Pet parents have their ideas about animal care, and we have ours. As Sigmund Freud wrote more than a century ago, “It is precisely the minor differences in people who are otherwise alike that form the basis of feelings of strangeness and hostility between them.”

If we want to foster more productive partnership between vet professionals and pet owners, then we must inspire them with our why. If we can learn to communicate the vision and reason behind our ideas and recommendations in a way that is understandable and not condescending, then clients will flock to us. In client communications, shifting the focus away from the ‘how’ of what we are recommending or the ‘what’ we are recommending to the ‘why’ can help clients become less resistant to our recommendations. People don’t care about the how and what until they care about the why. This session will present three tools to will help you develop your why, and therefore facilitate more productive, enjoyable, and profitable client interactions.

Objectives

- Learn through case studies in other industries how framing unfamiliar ideas in a way that was understandable facilitated acceptance
- Learn how to reframe your explanations in a way that makes utter, and complete sense to clients
- Understand that one size does not fit all when it comes to clients, and review three major areas of client concerns
- Learn to present your recommendations/link your agenda with the client’s agenda in a way that is a means to pursue the client’s needs, wants, and desires, i.e. appeal to the audience
- Understand how power and status in a relationship affect client compliance or acceptance of veterinary recommendations
- When humans are aware that someone is trying to persuade them, they natural raise mental shields. Disarm clients by leading with powerless communication.
- Learn how the Lion King and Trojan Horses, and apply your knowledge in the session.
A Face Only a Mother Can Love: Anesthetic Considerations for Brachycephalic Breeds

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Some of the most popular breeds in the country fall into the category of “brachycephalic”—literally translated to mean “short head.” In 2016 Bulldogs ranked #4, Frenchies were #6, and Boxers rounded out at #10. Brachycephalic breeds generally have a skull that is as wide (or wider) than its length. Breeds such as Boston terriers, shar peis, English bulldogs, French bulldogs, pugs, shih tzus, lhasa apsos, boxers, Pekinese, Persian cats, British shorthaired cats, and Scottish fold cats fall into this category.

Brachycephalic dog breeds are generally considered to have “brachycephalic airway syndrome.” They are at risk of having one or more of the following anatomic abnormalities:

- Stenotic nares
- Elongated soft palate
- Everted laryngeal sacculæ
- Hypoplastic trachea
- Laryngeal collapse

Each of these abnormalities decreases the patient’s ability to ventilate appropriately. They increase resistance to airflow in the trachea and can lead to hypoxia, hypoxemia, hyperventilation, hypercapnia, and hyperthermia. Intubation can be extremely challenging due to redundant tissues; hypoplastic trachea may mean the patient is intubated with a very small endotracheal tube. Decreased airflow through the nares and mouth can mean that patients are more susceptible to airway obstruction and decreased ability to ventilate.

When considering premedicants for any brachycephalic breed it is necessary to choose drugs that will not lead to over-sedation. Heavy sedation can leave patients with a decreased ability to ventilate appropriately. Many brachycephalic breed are relatively tractable and adequate sedation can be achieved using more “mild” drugs such as midazolam (0.05-0.2mg/kg)/hydromorphone (0.1-0.2mg/kg) or midazolam/oxymorphone (0.1mg/kg). For non-painful procedures, midazolam/butorphanol (0.2-0.5mg/kg) can be an excellent choice in brachycephalic breeds. There will be situations where patients are too aggressive or nervous for those combinations to work; in those cases, reversible sedatives such as dexmedetomidine may be warranted. Brachycephalic breeds often have increased vagal tone and may require anticholinergics to combat bradycardia. Brachycephalic cases should be closely monitored during the premed and sedation period. This is often the time when patients become distressed. Many brachycephalic patients have shallow ocular orbits and care should be taken throughout the anesthetic period. Overzealous restraint can lead to proptosis. Misplacement of the anesthetic face mask can cause ocular abrasions; inadequate lubrication during anesthesia can lead to corneal ulcers.

Brachycephalic breeds MUST be pre-oxygenated before anesthesia. Pre-oxygenation with a tight fitting mask and oxygen flows at 2-3L/min for as little as 3 minutes can significantly increase arterial oxygen concentrations. As brachycephalic breeds may be challenging to intubate, pre-oxygenation can buy a few minutes before patients become hypoxic. If the patient will not tolerate a tight fitting facemasks, remove the diaphragm on the facemask and increase the flow rate to 4-5L/min. Human designed facemasks work very well in brachycephalic breeds. Leave the facemask on through the induction period, removing it only when ready to intubate. Essentially any anesthetic induction agent can be used in brachycephalic breeds. Propofol is a sedative-hypnotic. It does cause transient apnea and a dose-dependent cardiovascular depression. In cats it is known to cause Heinz body formation following repeated administration. Propofol can cause apnea and hypoxia, but this can be quickly rectified by intubation and assisted ventilation. Alfaxalone is a neuromuscular steroid used to provide general anesthesia. Alfaxalone is metabolized by the liver and is redistributed fairly quickly. Etorphidate is an imidazole derivative that has minimal cardiovascular or respiratory effect. It is expensive and tends to be used for extremely critical patients. Etorphidate can cause suppression of adrenal functioning and acute hemolysis. It often causes nausea, retching, and vomiting at induction. This side effect can be avoided if the patient is appropriately sedated and if an adequate amount of the drug is used. Ketamine and midazolam is readily available and inexpensive. It is safe for use in most patients. Ketamine causes an increase in cardiac contractility, which can lead to an increase in heart rate and blood pressure—often good things to have with brachycephalic anesthetic cases.

Intubation is often the most stressful and challenging part of anesthesia in brachycephalic cases. The key is preparation for any scenario. Have endotracheal tubes in a wide range of sizes. It is not unheard of to place a size 6mm endotracheal tube in a 25kg bulldog. There is no shame in having 8 sizes of endotracheal tube for a brachycephalic case. Choose a laryngoscope with a long blade and a very good light. Check the light before you start the case! The assistant will need to hold the head up and open the mouth as wide as possible. Use the laryngoscope to push redundant tissue out of view. It is safe to use the tip of the endotracheal tube to push through everted sacculæ. NEVER force a tube that feels tight. If there seems to be an unreasonable amount of resistance, chose a

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smaller endotracheal tube. Secure the tube carefully, tying it behind the ears. Check and inflate the cuff to guard against aspiration and escape of waste anesthetic gases.

The gold standard for anesthetic monitoring of brachycephalic cases would include: pulse oximetry, capnometry, invasive blood pressure, blood gas sampling, and electrocardiography. Pulse oximetry measures oxygen saturation of hemoglobin and can give some degree of information about ventilation status and tissue perfusion. The pulse wave can alert an attentive anesthetist to possible arrhythmias. A pulse-ox monitor can be placed in a number of areas—tongue, toes, ears, vulva, prepuce, or rectally.

Capnometry is a vital monitor in these cases and will give information on the inspired and expired amounts of carbon dioxide. Capnometry is a simple, non-invasive tool that can give the anesthetist vital information on ventilatory status. The capnograph wave can highlight hypercapnia, hypocapnia, decreased cardiac output, poor pulmonary perfusion, airway obstruction, and anesthetic system malfunctions. End tidal carbon dioxide levels should be maintained between 35-45 mmHg. Spontaneous or manual ventilation is preferred to mechanical ventilation particularly in hypotensive cases. Mechanical ventilation will impede venous return and will lower blood pressure. This being said, brachycephalic breeds often become apneic under gas anesthesia and may need supportive mechanical ventilation.

Direct arterial pressure monitoring is the best way to obtain constant, accurate, real-time information about blood pressure. Arterial catheters can be placed in the dorsal pedal, lingual, auricular, or coccygeal arteries. Invasive blood pressure monitoring requires a higher level of skill and slightly more equipment than oscillometric monitoring, but provides a wealth of information. In addition to systolic and diastolic numbers, arterial monitors can highlight volume issues, ventilator induced cardiac depression, and cardiac dysrhythmias. Invasive blood pressure monitoring can often be the first to pick up hemorrhage or hypovolemia. Brachycephalic breeds often have an excess of skin and/or dysplastic limbs, so a blood pressure cuff may not provide accurate readings. Regardless of whether invasive or non-invasive pressure monitoring is used, mean arterial pressures should be maintained above 70mmHg to provide adequate tissue and organ perfusion.

ECGs show electrical activity of the heart and can alert the anesthetist to arrhythmias and dysfunctions, but in no way tells the level of cardiac output or tissue perfusion. ECGs are easy to use, non-invasive, and can be easily placed before anesthesia if warranted. ECGs show common dysfunctions that can be linked to electrolyte imbalances, levels of anesthesia, ventilation issues.

Brachycephalic cases must be monitored well into recovery. Capnometry should be monitored until the patient is extubated. Pulse oximetry should be monitored for as long as it will be tolerated. Patients should be recovered in sternal recumbancy with the head elevated. Brachycephalic patients will often remain intubated much longer than a “standard” case. Many brachycephalic dogs are happy to be up and moving and still intubated. It is important to wait until the patient is actively resisting intubation before removing the endotracheal tube. Removing the endotracheal tube before the patient is fully capable of appropriate ventilation and oxygen saturation can lead to airway obstruction and potential respiratory arrest. Be patient when considering “pulling the tube.” Supplemental oxygen should be available and provided as needed in brachycephalic patients. The patient should continued to be monitored until they are saturating at above 92% on room air and are awake, aware, and ambulating normally.
Pain management in veterinary patients has come a long way. There are a myriad of analgesic drugs available and numerous techniques exist to further provide top-notch analgesia and pain management to animals undergoing even the most painful surgeries. Constant rate infusions (CRIs) are increasing in their usage, thanks in large part to their wide versatility. There are pharmacologic agents used as CRIs that can provide analgesia, maintain anesthesia, and provide blood pressure support if needed.

Analgesics

Delivery of analgesic drugs as a CRI is typically far superior to bolus doses for a number of reasons. By administering a constant, steady infusion of a drug, stable levels of tissue concentration are reached. This prevents the peaks and valleys of comfort versus discomfort that occurs when bolus doses of analgesics are given as a patient begins to seem painful. When giving bolus doses, drugs peak, providing analgesia, then fall to below therapeutic levels allowing for breakthrough pain. CRI administration eliminates that rise and fall. CRIs can be easily adjusted to meet the needs of each individual patient, allowing for lower amounts of the drug to be given. Less money is spent on the actual drug as well as on supplies (syringes, needles, etc). Lower doses also decrease the incidence and severity of side effects.

Analgesic choices

Many classes of analgesic drugs can be given as a CRI including opioids, local anesthetics (specifically lidocaine), NMDA receptor antagonists (ketamine), and alpha-2 agonists (dexmedetomidine). Drug dosages are shown in Table 1.

Opioids work by binding with specific receptors in the central nervous system (CNS) and can, depending on the opioid, provide relief for mild to severe pain. Morphine, fentanyl, hydromorphone, and butorphanol are commonly used in opioid CRIs. Morphine, fentanyl, and hydromorphone can be used for moderate to severe pain, while butorphanol is only appropriate for use in mild to moderately painful cases. CRIs tend to reduce the severity of opioid side effects such as vomiting, dysphoria, and respiratory depression, but patients should still be monitored closely for any sign of distress.

Lidocaine works by blocking sodium ion channels and causing membrane stabilization. It can reduce the amount of opioid analgesic and inhalant gases required to maintain anesthesia. Lidocaine is relatively inexpensive and has anti-arrhythmic and anti-inflammatory properties. It is may be useful in cases where gastrointestinal pain is involved (GDV, laparotomies, etc). Cats have an increased sensitivity to local anesthetics and it is currently not recommended to use lidocaine infusion on feline cases.

Ketamine works by antagonizing the NMDA receptors which are responsible for central sensitization, hypersensitization, and “wind-up” pain. Ketamine is not capable of providing adequate analgesia in its own right, however, when administered in combination with opioid analgesics, it can lower anesthetic requirements of the patient.

Dexmedetomidine has mild analgesic properties, as well as anxiolytic and sedative properties. It works by simulating alpha-2 receptors in the CNS. Dexmedetomidine CRIs are most commonly used in the postoperative phase, for patients that are anxious and/or vocal despite an appropriate anesthetic regimen.

Anesthetic maintenance

Total intravenous anesthesia (TIVA) with propofol may be indicated for short, painless procedures. It may also be needed in situations where a patient cannot be intubated (tracheal procedures), where patients need to remain intubated and unconscious for extended periods (cases requiring long term ventilation assistance), or patients with autosomal disorders where inhalants cannot be used (malignant hyperthermia). In these cases propofol is administered with a bolus dose of 2-5 mg/kg followed by 0.05-0.2mg/kg/min. Adequate oxygenation is always a concern, so if possible it is recommended to intubate patients and maintain them on 100% oxygen. Standard anesthetic monitoring is still needed in TIVA patients. It may be harder to control depth when using TIVA, so good monitoring can act as an early alert system to patients that become too light or too deep, much the same as when anesthetizing a patient with volatile anesthetics.

Pressor support

Dopamine and dobutamine are the choices most commonly used to provide blood pressure support in hypotensive patients. Both drugs increase cardiac contractility, and assuming the patient has appropriate vascular volume, these drugs can most boost low blood pressure. The range for both dopamine and dobutamine is 1-10 mcg/kg/min. There can be a wide variation in patient reactions, so it is recommended to begin with low end dosage and adjust as needed.

Equipment

One downfall of CRI administration is that it requires specific equipment for the best and safety route of delivery. It is possible to deliver CRIs using either a fluid pump, syringe pump, or standard drip set. It is ideal to use a syringe pump, which can be programmed with all the variables involved in administering the infusion, but pumps can range from hundreds to thousands of dollars. If CRIs are being used on a regular basis, infusion pumps could be worth the investment—less waste, less man-power needed, fewer errors. Fluid
Pumps are a nice piece of equipment to have if you are simply injecting your drugs into a bag; they are programmable and can be set to deliver specific volumes over time. It is still on the technician to figure out the best concentration of the CRI for the length of time it will be delivered. If neither a fluid pump or syringe pump is available, a standard drip set can be used. However, extreme attention needs to be paid to ensure the patient receives the appropriate fluid rate.

**Table 1: Analgesic CRI drug dosages**

<table>
<thead>
<tr>
<th>Drug</th>
<th>IV Loading Dose</th>
<th>CRI Dose</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butorphanol</td>
<td>0.1-0.2 mg/kg</td>
<td>0.1-0.2 mg/kg/hr</td>
<td>Can be expensive</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>1-2 mcg/kg</td>
<td>0.5-2 mcg/kg/hr</td>
<td>Fewer negative side effects noted</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>2-5 mcg/kg</td>
<td>2-25 mcg/kg/hr</td>
<td>Lower end dose for post-operative management</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.02-0.05 mg/kg</td>
<td>0.01-0.04 mg/kg/hr</td>
<td>Lower end of dose for feline patients</td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.5 mg/kg</td>
<td>2-10 mcg/kg/min</td>
<td>Often used with an opioid</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1-2 mg/kg</td>
<td>25-50 mcg/kg/min</td>
<td>NOT IN CATS</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.1-0.3 mg/kg SLOWLY</td>
<td>0.1-0.3 mg/kg/hr</td>
<td>Lower end dose for cats</td>
</tr>
<tr>
<td>Morphine-Lidocaine-Ketamine</td>
<td>M: 0.1 mg/kg</td>
<td>M: 0.12 mg/kg/hr</td>
<td>Can use fentanyl/ hydro in place of morphine</td>
</tr>
<tr>
<td></td>
<td>L: 0.5 mg/kg</td>
<td>L: 1.5 mg/kg/hr</td>
<td></td>
</tr>
<tr>
<td></td>
<td>K: 0.25 mg/kg</td>
<td>K: 0.12 mg/kg/hr</td>
<td>Omit lidocaine for cats</td>
</tr>
</tbody>
</table>

**References**

Receptor Soup: 
Creating the Right Recipe for Hypotensive Patients
Katrina Lafferty, BFA, CVT, VTS (Anesthesia) 
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Hypotension is a constant plague for patients undergoing general anesthesia. General anesthesia is required to provide muscle relaxation and lack of response to surgical stimuli; however, the drugs required to achieve that state often cause decrease in cardiac output and systemic vascular resistance, leading to a hypotensive state. To further compound the hypotensive conundrum, many patients present as hypotensive even before anesthesia and it is an uphill battle to maintain a normotensive state.

Basic blood pressure physiology
In a nutshell, blood pressure is created by a pump (the heart) pushing fluid (blood) through tubing (vessels). The pressure itself is increased or decreased by the force put out by the pump and the size of the tubing (constricted vs. dilated). The standard formula for blood pressure is:
\[ BP = CO \times SVR \]
- **BP**: Blood pressure
- **CO**: Cardiac output
  - \[ CO = HR \times SV \] (stroke volume)
- **SVR**: Systemic vascular resistance

The cardiac muscle cells, or myocytes, receive both sympathetic and parasympathetic innervation. Sympathetic stimulation of the myocytes will result in positive chronotropic and ionotropic effects. Parasympathetic stimulation of the myocytes will result in blockage of chronotropic effects. Vascular walls are comprised largely of smooth muscle which contracts or relaxes to regulate the pressure within the vessels. Increased contraction or constriction leads to hypertension, relaxation or dilation leads to hypotension. The vascular smooth muscle has sympathetic innervation; increased stimulation or release of norepinephrine will result in an increase in muscle contraction. Decreased stimulation or release of norepinephrine will result in muscle relaxation or dilation.

Blood pressure itself is regulated by baroreceptors and chemoreceptors, which are in turn controlled by the vasomotor center of the medulla. Baroreceptor input is received from the left atrium, aortic arch, and carotid sinus. Sympathetic tone is affected by increases and decreases in blood volume. Chemoreceptors is received in response to situations like hypoxia or hypercapnia.

Blood pressure measurements consist of:
- **Systolic**: maximum pressure of contraction (stroke volume and arterial compliance)
  - Dogs: 90-140  Cats: 80-140
- **Diastolic**: minimum pressure of filling (systemic vascular resistance and heart rate)
  - Dogs: 50-80  55-75
- **Mean**: “average” measurement of pressure throughout the cycle; best indicator of perfusion pressure for organ beds
  - Dogs: 60-100  Cats: 60-100

Causes of hypotension
- **Cardiogenic causes**: cardiomyopathy, valvular disease, arrhythmias, SIRS/sepsis, hypoxia, electrolyte or acid/base imbalance
- Decreased preload: hypovolemia (including hemorrhage), decreased venous return, positive pressure ventilation
- Decreased vascular resistance: SIRS/sepsis, extreme hypoxia, acidemia, anesthetic drugs, histamine release
- Decreased heart rate: cervical disease, hypothermia, anesthetic drugs

Treatment of hypotension
It may be difficult to determine the root cause of hypotension, though there are usually many varying factors. In healthy patients, the causes of hypotension tend to be anesthetically induced. Many anesthetic tools, specifically inhalants, can cause profound vasodilation and hypotension. The front line (again, in healthy patients) for correcting hypotension is to decrease vaporizer settings, adding additional analgesic to facilitate this if necessary. Utilizing a 10ml/kg bolus of crystalloids over 10-15 minutes can also help in correcting a hypotensive state. Overzealous mechanical ventilation can also negatively impact pressure. Adjust the ventilator settings when possible. If these techniques do not correct the issue, it is time to move alternative pressure support.

Receptors
Generally speaking, the pressure support drugs available in veterinary medicine fall into two categories: ionotropes and vasopressors. Ionotropes support blood pressure by increasing cardiac contractility. Vasopressors increase blood pressure by causing
vasoconstriction. There are many pressure support agents that fall into both categories. Vasopressors and ionotropic drugs are sympathomimetics (mimicking the action of the sympathetic nervous system) and work on adrenergic receptors located throughout the cardiovascular system. Adrenergic receptors are classified as alpha-1, beta-1, and beta-2. Alpha-1 receptor agonists can cause vasoconstriction or vasodilation depending on the dose. Beta-1 receptor agonists cause an increase in cardiac contractility and heart rate. Beta-2 receptor agonists cause vasodilation. Deciding which pressure support drug will depend on many factors including pre-existing conditions, primary goal, and to some extent, personal preference. Drugs doses are listed in table 1.

**Dopamine**
Dopamine is an endogenous catecholamine. It works as a CNS neurotransmitter, a systemic hormone, and a precursor to norepinephrine. Dopamine can have varying actions, depending on the dose. In the 5-10mcg/kg/min range, it acts at beta-1 receptors and increases cardiac contractility. In the 10-20mcg/kg/min range, it has some effect on the alpha-1 receptors and causes vasoconstriction. Very high doses or inadvertent boluses of dopamine can cause tachycardia and arrhythmias. Dopamine causes an increase in cardiac afterload and should be avoided in cases with mitral regurgitation, dilated cardiomyopathy, and congestive heart failure. It should also be avoided in cases with chronic renal failure. Vasoconstriction to renal vessels could worsen the condition.

**Dobutamine**
Dobutamine is a synthetic catecholamine that works almost exclusively at the beta-1 receptors. In this way it improves pressure through an increase in cardiac contractility. If patients exhibit tachycardia after beginning an infusion of dobutamine, it may indicate hypovolemia. Dobutamine is typically the inotrope of choice in cases with mitral regurgitation, dilated cardiomyopathy, congestive heart failure, and chronic renal failure. Cats may have an increased sensitivity to dobutamine and should be run at lower rates. In cases of profound hypotension it may be required to add a vasopressor (norepinephrine, vasopressin).

**Ephedrine**
Ephedrine is a non-catecholamine that works at both alpha and beta receptors, though more action is at the beta receptor. Ephedrine causes a release of systemic norepinephrine; the physiologic stores of norepinephrine will be depleted with repeated doses of ephedrine. As a result, ephedrine is typically given as a single bolus dose, without need for a constant rate infusion. Ephedrine is highly concentrated and for easier and more accurate dosing in small patients it is advisable to dilute with saline. Ephedrine should not be used in cases with renal disease as it causes a decrease in renal perfusion.

**Norepinephrine**
Like dopamine, norepinephrine is an endogenous catecholamine. It has strong alpha-1 and beta-1 actions. In normal, healthy patients, norepinephrine is not on the forefront for dealing with hypotension. However, it is often the first line for cases with compensated or vasodilatory shock. In a patient with correct fluid resuscitation it will increase blood pressure. Heart rate increase is variable. Norepinephrine can be piggybacked with dobutamine. As with dopamine, norepinephrine should be avoided in cases with mitral regurgitation, dilated cardiomyopathy, and congestive heart failure. It should also be avoided in cases with chronic renal failure. Vasoconstriction to renal vessels could worsen the condition.

**Isoproterenol**
Isoproterenol is a synthetic catecholamine that works at beta-1 and beta-2 receptors. It is a potent drug, causing increase in cardiac contractility, heart rate, and cardiac output. However, it also causes vasodilation, leading to mixed effectiveness. Isoproterenol is not used for “typical” hypotensive cases. It is reserved for cases of third degree AV block or sick sinus syndrome. When these cases require general anesthesia, isoproterenol can increase heart rate and hopefully improve cardiac output. Isoproterenol has a high likelihood of causing arrhythmias and it is essential to monitor an ECG.

**Phenylephrine**
Phenylephrine is a synthetic non-catecholamine that has primary action at the alpha-1(and 2) receptors. Phenylephrine causes vasoconstriction, but decreases heart rate as a result of the increase in blood pressure. Phenylephrine is typically reserved for cases that are non-responsive to other attempts to increase blood pressure. This is one of the only blood pressure drugs that can be run as a CRI and given as intermittent boluses.

**Vasopressin**
Vasopressin does not work through alpha and beta receptors. Instead, vasopressin exerts its influence on the receptors in the smooth muscles of the vasculature. It causes vasoconstriction. Vasopressin is usually chosen after patients are unresponsive to fluids, and other ionotropes/vasopressors.
Epinephrine

Epinephrine is an endogenous catecholamine like norepinephrine. It has very strong action at both alpha and beta receptors. Epinephrine has a strong likelihood to cause arrhythmias and is not the first choice for hypotensive management. It is used primarily as a bolus injection during cardiac arrest. In severe situations, with patients that are unresponsive to every other attempt to correct profound hypotension, it is worth trying a constant rate infusion of epinephrine. It is vital to monitor an ECG during administration.

Table 1: Drug doses and information

<table>
<thead>
<tr>
<th>Drug</th>
<th>Receptor Site</th>
<th>Agonist Action</th>
<th>Drug Dosage</th>
<th>Cautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>Both alpha and beta</td>
<td>Increase in heart rate, cardiac contractility, may cause vasoconstriction</td>
<td>5-10mcg/kg/min (beta action) 10-20mcg/kg/min (alpha and beta action)</td>
<td>Higher doses may cause tachycardia and arrhythmias</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Beta-1</td>
<td>Increase in cardiac contractility</td>
<td>2-5mcg/kg/min(cats) 5-20mcg/kg/min(dogs)</td>
<td>Possible seizures in cats at higher doses</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>Beta (primary), alpha(secondary)</td>
<td>Increase in heart rate, vasoconstriction</td>
<td>0.05-0.2mg/kg (bolus dose)</td>
<td>Not for cause with renal disease</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Alpha-1(primary), Beta-1(secondary), Beta-2(minimal)</td>
<td>Increase in heart rate, cardiac contractility, vasoconstriction</td>
<td>0.5-5mcg/kg/min</td>
<td>Not for renal disease or some cardiac dysfunctions</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>Beta-1, beta-2</td>
<td>Increase heart rate, vasoconstriction</td>
<td>0.1-2mcg/kg/min</td>
<td>Causes arrhythmias, monitor ECG</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>Alpha-1, alpha-2</td>
<td>Vasoconstriction</td>
<td>5-20mcg/kg/bolus 0.1-0.5mcg/kg/min (CRI)</td>
<td>Bolus can be given every 10-15 minutes</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Smooth muscle receptors in vessels</td>
<td>Vasoconstriction</td>
<td>0.01-0.04mcg/kg/HR</td>
<td>Only for cases unresponsive to other options</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Alpha and beta</td>
<td>Vasoconstriction</td>
<td>0.025-0.3mcg/kg/min (hypotension) 0.02-0.05mcg/kg (CPR)</td>
<td>Generally for CPR; used as a last resort in unresponsive hypotension</td>
</tr>
</tbody>
</table>

References

Breathe In, Breathe Out: 
The Ins and Outs of Capnography
Katrina Lafferty, BFA, CVT, VTS (Anesthesia)
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A capnograph can be the anesthetist’s best friend. Knowing the ins and outs of capnography and understanding what the capnograph is showing can be the difference between life and death. Capnography provides early warning for a number of life-threatening situations: malignant hyperthermia, cardiovascular collapse, endotracheal tube blockage or misplacement, as well as hyperventilation and hyper-/hypocapnia. Capnometry is the measurement of CO2, but is displayed as just a number—ETCO2, respiratory rate, +/- inspired CO2. Capnography is an all inclusive measurement that gives numbers and a real time display of the CO2 waveform, which can provide additional information.

Normal end-tidal carbon dioxide (ETCO2) values should be with 35-45mmHg in the anesthetized patient. ETCO2 is usually closely reflective of arterial CO2, though ETCO2 tends to under estimate by 2-5mmHg in a normal patient with appropriate ventilation and perfusion. Any measurements below 35 mmHg are termed “hyperventilation/hypocapnia” and values about 45mmHg are considered “hyponoventilation/hypercapnia.” A capnograph will give information on the four main phases of a complete exhalation. Phase one—exhalation of anatomic dead space; phase two—end of expiration; phase three—expiratory pause/exhalation of alveolar gases; phase four—beginning of inspiration.

Capnography is a non-invasive monitoring tool that provides a wealth of information regarding the status of an anesthetized patient. It will give insight into the functioning of the patient’s ventilatory and cardiovascular systems as well providing a tool to gauge if the anesthetic machine is working properly. It can be used to confirm endotracheal tube placement and provide an early alert to partial or full tracheal obstruction.

Carbon dioxide monitoring is directly related to ventilation. It provides a respiratory number for any patient that is intubated or utilizing a tight-fighting anesthetic mask. CO2 that is exhaled (or inhaled in the case of carbon dioxide rebreathing) is given a number and provides information about how well the patient is breathing. It provides a respiratory rate which is helpful in cases where there is little movement of the bag or the patient is covered by drapes and actual respiration is difficult to observe. CO2 production and cardiovascular function are inextricably linked. For carbon dioxide to be removed from tissues, delivered to the lungs, and exhaled from the body requires adequate blood flow. If a patient becomes hypotensive and has decreased perfusion, less CO2 is carried to the lungs. A CO2 monitor can be one of the earliest alerts to a failing cardiovascular system.

Capnography can also be an early alert to failure within the anesthetic system. If the inspiratory or expiratory one-way valve is obstructed or incorrectly placed, it will alter the waveform on the capnograph. If the CO2 absorbent has been exhausted, it will be reflected in rising inspired carbon dioxide. One reason for a cessation in the capnograph waveform can be disconnection of the anesthetic hosing.

The type of capnographs generally used in veterinary medicine work by infrared (IR) absorption. CO2 molecules absorb IR light at specific values and those values are reflective of CO2 levels. When the IR light shines through the gas sample, a signal is obtained which can be used to gauge CO2 levels. ETCO2 monitors require specific calibration to ensure the signal obtained is a correct reflection of the CO2 levels.

There are two types of capnographs on the market, mainstream sampling monitors and sidestream sampling monitors. Sidestream, or diverting, monitors involve a small adapter being connected between the endotracheal tube and the anesthetic hoses. A sampling line then draws a sample of the gas and carries it to a unit where it will be analyzed. The main advantages to the sidestream monitors are the light weight of the sampling adapter and the lower cost of the monitor versus the mainstream monitors. Sidestream adapter pieces are easily cleaned and can therefore be used for any wet/messy procedures such as dental procedures, rhinoscopies, or endoscopies. Sidestream monitors can also be connected to nasal cannulas to obtain readings. There are unfortunately many disadvantages to using sidestream sampling monitors over mainstream monitors. Sidestream monitors pull approximately 200mls/minute from the system. In very small patients, this can be a large volume to remove. The sample has to be carried through a line to the analyzing unit and thus the sample can be a few breaths behind real time. The analyzing unit requires several pieces: the unit, water traps/filter, sampling lines, and sampling adapters, which can increase the overall cost.

Mainstream monitors are also called in-line or non-diverting monitors. The sampling box is placed directly between the endotracheal tube and the anesthetic hoses. The IR sample is taken immediately from the patient. There are several advantages to mainstream monitors. They provide very fast response time and can be used on any size patient as there is no sample being removed through an external line. There are fewer pieces with a mainstream monitor—no sampling line and no water traps. The disadvantages to mainstream monitors are primarily cost, they are much more expensive than sidestream monitors. The sampling box can also be heavy and may be too much weight for small patients. As all the sampling pieces are immediately in line with the endotracheal tube, they are more susceptible to damage by water and blood and shouldn’t be used for wet or bloody procedures.
Dead space is defined as areas within the ventilatory system—that are not involved in the exchange of gases. Total dead space is a combination of anatomic dead space (the mouth and trachea—parts of the airway that move gas to the alveoli), alveolar dead space (areas of the lungs that should be involved in gas exchange but are not—due to pulmonary pathology or disease, lack of perfusion, atelectasis), and mechanical dead space (parts of the anesthetic circuit that extend the anatomic dead space—the endotracheal tube, wye-piece, restrictive reservoir bag). Areas of dead space can lead to false readings of ETCO2, giving a CO2 number that is lower than that of the arterial blood levels.

Rebreathing is when a patient is re-inhaling expired gases. It gas that is being rebreathed has not yet been cleared of CO2 by the carbon dioxide absorbent in the anesthesia machine and will cause the baseline reading on the ETCO2 waveform to rise. At a certain point an elevated inspired CO2 level will cause the expired CO2 level to rise as the patient is not able to adequately clear carbon dioxide from the body.

Capnographs are worth their weight in gold and any patient presenting for any type of anesthetic procedure would benefit from their use. However, if a choice needed to be made, patients that would benefit most from capnographic monitoring would be any cases with respiratory disease, neurologic disease, and any cases with metabolic dysfunction. Procedures that would benefit from use of a capnograph are those involving the respiratory system—thoracotomy, lung lobectomy, diaphragmatic hernia, or cases where positioning could cause obstruction of the endotracheal tube—cervical neck procedures, ophthalmic cases, etc.

Causes of rebreathing
- Misplaced/defective expiratory valve
- Low inspired O2 flow
- Exhausted CO2 absorbent

Causes of hypercapnia
- Patient hypoventilation (overly deep anesthetic level)
- Airway obstruction
- Restriction of thorax or abdomen
- Restriction of pleural space
- Pulmonary disorder
- Inappropriate mechanical or hand-ventilation
- Increased muscle activity (shivering, tremors, seizure-like activity)
- Pyrexia
- Defective one-way valve
- Exhausted CO2 absorbent
- Neurologic disease

Causes of hypocapnia
- Patient hyperventilation (pain, pyrexia, light anesthetic level)
- Hypoxemia
- Hypotension
- Sepsis
- Overzealous mechanical or hand-ventilation

Causes of hypoxemia
- Low inspired O2
- Hypoventilation
- Pulmonary disorder
- Diffusion impairment

Absent waveform
- Apnea
- Tube obstruction
- Cardiac arrest
- Machine disconnection
Elevated baseline
  • Rebreathing of CO2

Elevated plateau
  • Hypoventilation/hypercapnia

Decreased plateau
  • Hyperventilation/hypocapnia
  • Hypothermia
  • Leak
  • Partial airway obstruction

Variable plateau
  • Spontaneous breath/breathing
  • Cardiogenic oscillations
  • Curare cleft (from muscle relaxants/paralytics)
  • Movement of patient by surgeons
Many technicians (and veterinarians) do not feel comfortable working with exotics; there is an unfortunate lack of information on how to appropriately handle, medicate, and anesthetize exotic pets. However, the number of exotic pet owners is increasing dramatically and more and more of those pet owners are bringing their family members to veterinary clinics expecting the same kind of care cats and dogs normally receive. Spays and neuters in exotic pets are becoming a common occurrence, as are abdominal surgeries, fracture repairs, and mass removals. The purpose of this lecture is to provide basic information on how to competently provide quality anesthesia for rabbits, ferrets and rodents in the hopes that technicians will feel more confident about providing a better standard of care for the exotic species that come through the clinic door.

The anesthesia of exotic pets can be divided into five parts: restraint, premedication, induction, intra-operative, and recovery/post-operative. There are many similarities in anesthesia between rabbits, ferrets, and rodents, despite the vast differences in the species as a whole.

Improper restraint of exotic patients can have a number of harmful consequences. Rabbits can suffer fractured legs, vertebral dislocation, and damaged pinna; ferrets can suffer broken legs, rodents can suffer broken limbs or tails or torn skin. Handlers are also at risk when employing improper restraint techniques—bites, scratches, and possibly worse if dealing with very fractious animals. When restraining any exotic species, a towel is the restrainer’s best friend. When working with any species, first choose an appropriately sized towel—small for rodents or small bunnies, medium for most rabbits, ferrets or guinea pigs/chinchillas, and large for very large bunnies.

Fractures and vertebral dislocations are unfortunate occurrences in rabbits that are improperly restrained. Rabbits have very powerful hind legs and if they are not held properly a frightened rabbit can kick out and cause irreparable damage. The easiest way to safely pick up a rabbit—and restrain it for an examination, is to wrap it firmly in a towel, creating a “bunny burrito.” An alternative method is to place one hand under the rump, firmly holding the hind feet and placing the other hand under the sternum, grasping the forelimbs. NEVER pick up a rabbit by the ears, or pick it up by the scruff with the hind end unsupported.

Ferrets can be a challenge to restrain based on their long tube-like body structure and their extreme flexibility. Ferrets respond readily to being scruffed and all but the most stubborn ferrets can usually be restrained and examined in this manner. They will yawn repeatedly when in this position and may clamp their jaws together, so it is wise to keep fingers out of the way. It is possible to wrap ferrets up using the “burrito” method, but they are at a higher risk of becoming hyperthermic and still may be able to wriggle out of this particular restraint device. Extremely fractious ferrets are a rarity, but in those cases, leather gloves and cat bags are useful.

For rats and mice there are three main types of restraint: tail restraint, full body restraint, and stockinet restraint. For tail restraint firmly grasp the patient at the base of the tail with one hand and use the other hand to support the body. Never lift by the body unsupported or allow the animal to “dangle.” Place the animal on the examination table (on a towel is usually preferred) and proceed to full body restraint. For full body restraint grasp around the thorax just behind the front legs with one hand and just above the hind legs with the other hand. Apply very gentle pressure to “stretch” the rodent. Be careful not to “over-stretch” the rodent and cause discomfort or restrict respirations. Another method for full body restraint is to hold the tail at the base for security then grasp the loose skin over the neck and back and hold it firmly. For stockinet restraint, a soft cotton stockinet is placed over the rodent. Many rats and mice find this method of restraint almost calming and it can facilitate easier examinations and blood draws. This only is appropriate if the rodent will tolerate it, if they are stressed, remove them immediately and choose a different method of restraint. Guinea pigs and chinchillas are very gentle animals that are usually very easy to handle. They require a secure grip around the thorax and support of the rest of the body.

As with canine and feline patients, exotic species should be pre-mediated with a sedative and analgesic combination before any anesthetic episode. Premedication helps decrease level of stress in patients, reduces anesthetic requirements, and helps to deal with pain associated with the procedure. Almost all opioids and sedatives that are used for canine and feline patients can be used in exotic patients. While it is not typically acceptable to mask induce patients, exotic species are the exception. They are usually not sedate enough from premedication to allow for easy catheter placement. Even if IV induction is an option, many species are require more time to intubate and an induction with an IV agent may not allow enough time for intubation before it reaches a crisis state.

Isoflurane and sevoflurane are both acceptable choices of gas inhalant in exotic anesthesia. When performing a mask induction be extra vigilant in monitoring vital signs. Take care not to over- or under-restrain the patient and use caution not to cause ocular damage with the induction mask.

Rabbits are one of the more technically challenging animals to intubate; however attempts should always be made to intubate, and with practice it becomes much easier. Rabbits are intubated either nasally or using a blind oral technique. Endotracheal tube sizes

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University of Wisconsin
Madison, WI

Crash Course: Anesthesia for Rabbits, Ferrets, and Rodents

Course:

- 378
range from 2.0-5.0mm. Lidocaine dripped into the nose or sprayed in the back of the mouth can help facilitate intubation. Positioning is key to the success of rabbit intubation—patients must be placed in sternal with the neck hyper-extended. This places the trachea in a straight line and makes for easier intubation. The tube is placed in the nose or mouth and advanced on inspiration. Confirmation is achieved through visualization of fog in the tube or placement of a capnometer. The misplaced endotracheal tube can often be felt next to the trachea.

Rats are technically similar to cats in terms of intubation. Tube size ranges from 2-3.5mm, with larger sizes used in males. Lidocaine spray is useful to prevent laryngeal spasms; the mouth can be held open with gauze ties or IV tubing. Positioning is in sternal with the neck extended, very similar to positioning for feline intubation.

Catheters can be readily placed in exotic species. Equipment needed for intravenous catheters includes 22, 24, or 26 gauge catheters, gentle tape—clear tape or paper tape, and lightweight access ports.

Catheter placement in rabbits and ferrets is technically similar to catherization in feline patients. In both rabbits and ferrets, catheters can be readily placed in the cephalic or lateral saphenous veins. The medial saphenous veins are an option as well, but can be fragile and difficult to properly secure. In rabbits, the marginal ear vein is an excellent choice. It is usually easy to see, able to be secured, and can be maintained for long-term use. For rats, the most common catheter location is the tail vein, in guinea pigs and chinchillas, lateral saphenous or cephalic catheters are possibilities. In very small patients, it is often necessary to place catheters intra-osseous.

Similar monitoring techniques are used in canine and feline patients, allowing for a smaller scale. Pulse oximetry can readily be used. Probes can be placed on the feet, ears, tails, and tongues of all species as well as being placed in the rectum or esophagus.

Doppler flow detector crystals can be placed over the dorsal pedal artery in all species and can be used in conjunction with a sphygmomanometer to obtain blood pressure readings. The Doppler probe can also be placed in the femoral area, carotid/thoracic inlet, or on the chest in all species and used for sound and heart rate. Be careful not to restrict respirations if taping the probe on the chest. In rabbits the Doppler can be placed on aural artery. In rodents the doppler can also be placed on the ventral tail.

Capnometry can be used on any intubated patient and even some patients on a tight fitting mask. Dead space adapters exist that can be connected to the endotracheal tube in place of the regular tube adapter. Tube obstruction is a concern in exotic species, especially when using very small endotracheal tubes. A capnograph can be the first alert to an obstruction or inadvertent extubation. It can confirm proper intubation and can alert the anesthetist to issues with ventilation or cardiac output.

Non-invasive blood pressure monitoring is an option on some animals—good pediatric monitors usually work well on larger rabbits and ferrets and some larger rodents. Small blood pressure cuffs can be placed on the fore or hind limbs, or on the tail in ferrets and occasionally large rats.

Electrocardiography can be readily monitored in all species. The ECG gives information about rate and rhythm and can be a helpful tool to use under anesthesia. ECG patches can be placed on both forelimbs and the left hind limb in all exotic species. It is also possible to use 25 gauge needles pierced through skin with an alligator clip attached to the needle. If using alligator clips on skin (without an ECG patch) either flatten the teeth of the clip or use gauze squares to cushion the area and prevent tissue damage.

Temperature monitoring is one of the most vital monitors in exotic patients. Small species are at a high risk for hypothermia. It is important to note that for as quickly as small patients can cool down, they can warm up just as fast. Without careful monitoring of temperature, patients can easily become hyper-thermic. Temperature can be monitored using either rectal or esophageal temperature probes. Be careful when using a rectal temperature probe—the rectal mucosa in many exotic species (particularly rabbits) is very friable and can be torn by thermometer placement.

Monitoring anesthetic depth in exotic species is very similar to monitoring anesthesia in any species. Palpebral reflexes are reliable in ferrets, but not as much in rodents and rabbits. Jaw tone, rectal tone, and pedal reflexes are all accurate indicators of anesthetic depth. As with most species, if an exotic patient is too light, heart rate and respiratory rate will increase, if it is too deep, heart rate and respiratory rate will decrease.

Recovery requires as much monitoring as during the anesthetic period. Small exotic patients can become hypothermic very quickly and temperature should be monitored closely for several hours postoperatively. It can be difficult to maintain catheters in exotics species and a watchful eye must be kept on the catheter. Respiratory depression can quickly lead to cardiac arrest—vigilance can catch an issue before it becomes a critical problem. Always know the reversal drug and dose needed and have an emergency drug dose chart readily available.
<table>
<thead>
<tr>
<th>Rabbit Emergency Drugs</th>
<th>Atropine 0.4 mg/ml</th>
<th>Dexamethasone 4 mg/ml</th>
<th>Diazepam 5 mg/ml</th>
<th>Dopram 20 mg/ml</th>
<th>Epinephrine 1 mg/ml</th>
</tr>
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<tbody>
<tr>
<td><strong>Dosage</strong></td>
<td></td>
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<tr>
<td><strong>1 kg</strong></td>
<td>0.8-1.0 mg/kg IV</td>
<td>2.0 mg/kg IV</td>
<td>1-5 mg/kg IV</td>
<td>2-5 mg/kg IV</td>
<td>0.2 mg/kg IV</td>
</tr>
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<td><strong>2 kg</strong></td>
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<td>0.5 ml</td>
<td>0.2-1.0 ml</td>
<td>0.1-0.3 ml</td>
<td>0.2 ml</td>
</tr>
<tr>
<td><strong>3.0 kg</strong></td>
<td>4.0-5.0 ml</td>
<td>1.0 ml</td>
<td>0.4-2.0 ml</td>
<td>0.2-0.5 ml</td>
<td>0.4 ml</td>
</tr>
<tr>
<td><strong>4.0 kg</strong></td>
<td>6.0-7.5 ml</td>
<td>1.5 ml</td>
<td>0.6-3.0 ml</td>
<td>0.3-0.8 ml</td>
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<tr>
<td><strong>5.0 kg</strong></td>
<td>8.0-10.0 ml</td>
<td>2.0 ml</td>
<td>0.8-4.0 ml</td>
<td>0.4-1.0 ml</td>
<td>0.8 ml</td>
</tr>
<tr>
<td><strong>6.0 kg</strong></td>
<td>10.0-12.5 ml</td>
<td>2.5 ml</td>
<td>1.0-5.0 ml</td>
<td>0.5-1.3 ml</td>
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<tr>
<th>Ferret Emergency Drugs</th>
<th>Atropine 0.4 mg/ml</th>
<th>Dexamethasone 4 mg/ml</th>
<th>Diazepam 5 mg/ml</th>
<th>Dopram 20 mg/ml</th>
<th>Epinephrine 1 mg/ml</th>
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<tr>
<td><strong>Dosage</strong></td>
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<tr>
<td><strong>0.5 kg</strong></td>
<td>0.8-1.0 mg/kg IV</td>
<td>2.0 mg/kg IV</td>
<td>1-5 mg/kg IV</td>
<td>2-5 mg/kg IV</td>
<td>0.2 mg/kg IV</td>
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<td><strong>1 kg</strong></td>
<td>0.3-0.5 ml</td>
<td>0.5-1.0 ml</td>
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<td>0.5-0.1 ml</td>
<td>1.0-2.0 ml</td>
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<td>0.5-1.0 ml</td>
<td>0.6-1.4 ml</td>
<td>0.05 ml</td>
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<tr>
<th>Small Rodent Emergency Drugs</th>
<th>Atropine 0.4 mg/ml</th>
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<th>Dopram 20 mg/ml</th>
<th>Epinephrine 1 mg/ml</th>
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<tr>
<td><strong>Doseage</strong></td>
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<tr>
<td><strong>20 g</strong></td>
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<td>0.01-0.02 ml</td>
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<tr>
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<tr>
<td><strong>100 g</strong></td>
<td>0.03-0.1 ml</td>
<td>0.1-0.13 ml</td>
<td>0.02-0.1 ml</td>
<td>0.03-0.05 ml</td>
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<tr>
<td><strong>150 g</strong></td>
<td>0.04-0.15 ml</td>
<td>0.15-0.19 ml</td>
<td>0.03-0.15 ml</td>
<td>0.04-0.08 ml</td>
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<tr>
<td><strong>200 g</strong></td>
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<td>0.2-0.25 ml</td>
<td>0.04-0.2 ml</td>
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<tr>
<td><strong>250 g</strong></td>
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<tr>
<td><strong>300 g</strong></td>
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<td>0.09-0.45 ml</td>
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<tr>
<td><strong>500 g</strong></td>
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<tr>
<td>Chinchilla Emergency Drugs</td>
<td>Atropine 0.4 mg/ml</td>
<td>Dexamethasone 4 mg/ml</td>
<td>Diazepam 5 mg/ml</td>
<td>Dopram 20 mg/ml</td>
<td>Epinephrine 1 mg/ml</td>
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<td><strong>Doseage</strong></td>
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<tr>
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<tr>
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<tr>
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<tr>
<td>1.25 kg</td>
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<tr>
<td>1.5 kg</td>
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<td>1.5-1.9 ml</td>
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<table>
<thead>
<tr>
<th>Guinea Pig Emergency Drugs</th>
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<th>Epinephrine 1 mg/ml</th>
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<tr>
<td><strong>Doseage</strong></td>
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<tr>
<td>0.5 kg</td>
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<tr>
<td>0.75 kg</td>
<td>0.2-0.38 ml</td>
<td>0.75-0.94 ml</td>
<td>0.15-0.75 ml</td>
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<tr>
<td>1 kg</td>
<td>0.25-0.5 ml</td>
<td>1.0-1.3 ml</td>
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<tr>
<td>1.25 kg</td>
<td>0.31-0.63 ml</td>
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<tr>
<td>1.5 kg</td>
<td>0.38-0.75 ml</td>
<td>1.5-1.9 ml</td>
<td>0.3-1.5 ml</td>
<td>0.15-0.38 ml</td>
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</table>
The 4th vital sign: pain. There are myriad of negative consequences associated with inappropriately managed pain: decreased healing, infection, heightened reactions to pain, changes to the CNS. Utilizing pain scoring systems reduce pain states and improve patient care.

Poorly managed pain, whether before, during, or after anesthesia, can have a number of harmful consequences. Most publications on the subject of pain management in both “traditional” canine/feline patients and “non-traditional” exotic patients show appropriate analgesia and pain management to be essential to wound healing and patient recovery. Improperly managed pain affects the patient and the owner.

Pain is an individual experience and can be somewhat subjective; many factors can affect how pain is interpreted. In humans and animals alike pain is tolerated differently. It can be influenced by genetics (think husky dog versus Staffordshire terrier), learned behaviors, and even culture. In veterinary patients the challenge of adequately recognizing, assessing, and treating pain is further complicated by the non-verbal nature of the patients. Much of the pain assessment techniques used in animals is modeled after what is used in human medicine for infants, toddlers, and other non-verbal patients. In the AAHA pain management guide for cats and dogs it is stated, “It is now well established that animals and humans have similar neural pathways for the development, conduction, and modulation of pain. According to the principle of analogy, because cats and dogs have neural pathways and neurotransmitters that are similar, if not identical, to those of humans, it is highly likely that animals experience pain similarly.” Read this to mean if it would cause discomfort to you, it would cause discomfort to your patient, regardless of species.

To better understand how to manage pain, one should have a fluency in the “language” of pain:

- Pain: an unpleasant sensory and emotional experience associated with actual or potential tissue damage; the perception of nociception
- Nociception: the activity produced in the nervous system by noxious stimuli
- Analgesia: absence of pain in response to normally painful stimuli
- Hyperalgesia: an increased response to a normally painful stimulus
- Allodynia: pain due to a stimulus that does not normally produce pain (i.e. touch)
- Central Sensitization: “Wind up pain;” changes in the central nervous system that occur as a result of repeated or chronic painful stimulus
- Analgesic: an agent or drug that causes or allows for relief from pain; a “pain killer”

The pain pathway in the body can be broken down into four basic groups: transduction, transmission, modulation, and perception. Analgesics and analgesic techniques work to modify to pain felt and transmitted throughout the circuit. Different analgesic drugs work on different parts of the pathway.

- Transduction: change of cellular chemical information into electrical impulses that travel the spinal cord
- Transmission: travel of the pain impulse; transmitting the signal to the brain
- Perception: cognizant recognition of painful stimulus
- Modulation: Brain and spinal cord communicate and work together to change or modify the painful sensation

There are typically describe 6 types of pain:

1. Somatic Pain: at the level of the skin; also involves muscles, tendons, and joints
2. Visceral Pain: Internal organs, soft tissue
3. Acute Pain: Recently occurring, generally less than a few months
4. Chronic Pain: Longer in scope, lasting more than a few months
5. Neuropathic Pain: Atypical pain, i.e: phantom limb pain
6. Referred Pain: Pain perceived in a region disparate from the actual painful site

Pain perception can be broken down into 3 sections: Perception (recognition of discomfort), Threshold (point when painful signal hits the brain), and Tolerance (amount of pain that can be endured). In order to assess pain thoroughly, consider these categories: Incidence, Level, Location, Length. For each patient, each category can have varying levels of intensity. All veterinary patients will be non-verbal, so the difficult task is to use behavioral assessments to answer questions of how painful an animal is. There are species differences in pain behavior, but many pain mannerisms are universal.
Universal non-verbal pain indicators

- Abnormal posture
- Restlessness
- Splinting
- Vocalizing
- Unwillingness to move
- Trembling/shivering
- Anorexia
- Elevated physiologic parameters
- Aggression (towards self or others)
- Lack of grooming
- Self-mutilation

Pain is often characterized and described as it is relevant to the owner. An adequate and thorough “pain history” can be very useful when creating an appropriate analgesic plan. The history should include:

- Previous/ongoing painful states
- Previous methods of controlling pain
- Owner’s expectations for pain control
- How/why the owner feels a patient is painful

The “FLACC” scale is often used in human medicine for non-verbal patients (infants, toddlers, demented patients, critically ill/unconscious patients) and has applications for veterinary patients. FLACC stands for “Face” (relaxed/grimace/tightened face), “Legs” (normal posture/kicking/restless), “Activity” (normal/restless/agitated), “Cry” (quiet/whimpering/frequent complaints), and “Consolability” (content/distractable/unable to comfort). Each area is ranked 0-2, with elevated numbers requiring treatments.

In veterinary medicine, there are 3 primary scales that have been adapted and utilized for pain assessment.

Colorado State University created pain scales for canine and feline patients, based on behavior and physical reactions to stimuli. It is an exceptionally easy to use scale, but does not necessarily have clinical validation to support it. It does have forms for both canine and feline patients.

Glasgow composite measure pain score-short form

This form is based on specific behavioral markers, assigning numeric points to determine level of pain. This scale is used universally, everywhere from small private practices to veterinary teaching institutes. This scale is designed for canine patients. This scale does have some clinical research to support it.

UNESP-botucatu multidimensional composite pain scale

This form is the feline companion to the Glasgow Pain Scale. It utilizing 10 categories to assign numbers for painful behaviors. Using a behavioral checklist is likely the most common way to create a pain assessment chart. Having a chart on hand allows for a standard, equal, reproducible way to evaluate pain in patients.

Behavior based charts should include categories for:

- Attitude or Mentation
- Activity
- Facial expression
- Guarding
- Posture
- Vocalization

Above all, treat each patient as an individual. If pain is suspected, treat. If an animal responds to analgesic intervention, that too is considered a point on the pain assessment scale.

References always available upon request.
We all know that opioids are one of the main building blocks for an effective analgesic plan. Opioids are usually cost effective with and have been shown to have great efficacy in veterinary patients. However, within the past few years, opioids have come under stricter regulations. This has caused a massive shortage of opioid supply available to the veterinary market. While this may seem like a reason to panic, we can also look at this as an opportunity to think outside of the box and provide a more multimodal analgesic experience to our patients.

Multimodal analgesic plans consist of more than one type of analgesic drug exerting effects at more than one pain pathway or receptor type. Because there are many types of pain involved with veterinary medicine (the acute pain of trauma or surgery vs. the chronic pain of osteoarthritis) this creates opportunities to treat each type of pain with different analgesics working on different types of pain. For example: treating somatic vs visceral pain. Visceral pain is pain that results from the activation of nociceptors of the thoracic, pelvic, or abdominal viscera. Visceral pain is diffuse, difficult to localize and often referred to a distant, usually superficial, structure. It may be accompanied by symptoms such as nausea, vomiting, changes in vital signs as well as emotional manifestations (stress). Somatic pain is generally described as musculoskeletal pain. Because many nerves supply the muscles, bones, and other soft tissues, somatic pain is usually easier to locate than visceral pain. It also tends to be more “intense and sharp”. The nociceptors in these tissues pick up sensations related to temperature, vibration, and swelling. This type of pain sensation is usually due to an injury, or from surgery, and results in acute somatic pain.

When thinking of an analgesic plan for your surgical patients, here are the top 5 things you can do to reduce or eliminate the use of pure mu opioids (if your supply has become scarce or disappeared completely).

**Constant rate infusions**

Constant rate infusions (CRI’s) have many benefits. First of all you can dramatically reduce your need for high concentrations of inhalant anesthetic, which will then help to lessen the side effects that go along with inhalant anesthesia. Second, constant rate infusions can provide a “steady state” of analgesic in the system to avoid the “peaks and valleys” of analgesic coverage seen with times dosing. One of the most commonly used adjunctive analgesic CRI’s is ketamine. Ketamine is an NMDA antagonist, which are useful to prevent ”wind up” (central hyper sensitization) of the NMDA receptors found in the dorsal horns of the spinal cord. Chronic pain, surgery or trauma can stimulate these receptors to the point where the response threshold is decreased and neural transmission to the brain causes an increase in pain perception. Ketamine is an excellent addition for management of surgical pain both intra-operative and for post-operative analgesic management.

By decreasing the dosage for CRI administration, undesirable side effects such as dysphoria/hallucinations do not seem to occur.

**Dosage for CRI administration:** Loading dose of 0.5 mg/kg IV followed by a CRI of 10 mcg/kg/minute is an excellent choice intra-operatively for patients with chronic pain experiencing the ”wind up” phenomenon

**NSAIDs**

Non steroidal anti-inflammatory drugs are a cornerstone of analgesic therapy. Generally, the classification NSAID is applied to drugs that inhibit one or more steps in the metabolism of arachidonic acid (AA). Unlike corticosteroids, which inhibit numerous pathways, NSAIDs act primarily to reduce the biosynthesis of prostaglandins by inhibiting cyclooxygenase (COX). NSAIDs can be used post surgically to reduce the need for additional opioids.

**Regional anesthesia**

Regional anesthesia is an important analgesic building block that should be a part of every surgical patient’s protocol. regional anesthesia techniques in veterinary practice have many advantages. These techniques provide effective preemptive and multimodal analgesia, reduce the amount of inhalational agent needed to maintain anesthesia, resulting in improved cardiopulmonary stability during anesthesia, modulate the sympathetically driven stress response to surgical trauma, reduce the development of central sensitization. Multimodal analgesia can even be provided within the regional block with combinations of local anesthetics, opioids, and alpha agonists.

**Long lasting analgesics**

Medications like the 24 hour buprenorphine (Simbadol) used in felines post surgery and the 72 hour bupivacaine (Nocita) used in dogs undergoing knee surgery have benefits of reduced need for additional opioids over time.
Maropitant
Maropitant has been shown to not only reduce the incidence of vomiting after opioid administration, but maropitant also binds to substance P. Substance P is involved in pain pathways, and NK-1 antagonists may have visceral analgesic effects in some species. For example, maropitant has an anesthetic-sparing effect during ovarian manipulation in dogs and cats undergoing ovariohysterectomy, when given at 1 mg/kg IV (followed by a 30 μg/kg/hr CRI in dogs) In a 2017 human study, 1,009 patients having head and neck surgery received general anesthesia without opioids. Instead, patients received various combinations of magnesium, sub-anesthetic ketamine, lidocaine and ketorolac, depending on the patient’s age and health. Surgeons and patients expressed a high degree of satisfaction with the new anesthesia protocol and postoperative pain management. After surgery, only 11 percent of patients experienced nausea, whereas 50 to 80 percent of patients typically suffer from nausea after surgery. Additionally, 64 percent of patients did not require any pain medication in the PACU.

In summary, multimodal plans are a must for proper analgesic therapy in the face of reduced supply of pure μ opioids. Work as a team to create the most multimodal protocol appropriate for your patients.

References
American Society of Anesthesiologists: Eliminating opioids from anesthesia decreases post-surgery nausea, study shows. Accessed 03/01/2018
Waddell, Katy. Constant rate infusions: Indications, calculations and applications in pain management Proceedings for CVC San Diego 2010
The importance of consent
Consent is a tenet of Cooperative Veterinary Care. Unlike historical methods of care, where “brutacaine” was the standard course of action, Cooperative Veterinary Care relies on medical management and training in order to have an animal be a voluntary participant in their own medical care.

Brutacaine is not unique to veterinary medicine. Many pediatric and even adult patients in medical clinics, emergency departments, dentist offices, etc., have experienced physical restraint during painful and frightening medical procedures. The human medical model has been researching how to eliminate brutacaine from their protocols as well. It has been found to be harmful to patients, increase patient stress, increase risks of iatrogenic injuries, and lengthen healing times. 1,2,3

Cooperative Veterinary Care allows patients to communicate with the handler, trainer, or medical professional, about their comfort level with and understanding of the procedure at hand.

We use trained “points of consent” in Cooperative Veterinary Care to give our patients a voice. When they are ready for us to progress, we proceed. If they aren’t ready, we either give medical relief (sedation, analgesia) or postpone elective procedures until more training can commence. Showing respect for our patients’ ability to provide consent is a key part of Cooperative Veterinary Care.

Relevance for companion animals
According to AAFP, 58% of cat owners perceive that their cats hate going to the veterinary hospital due to stress. These cat owners say they are reluctant to take their cats to the vet because of the stress and fear visits produce. According to AAHA, 38% of dog owners are reluctant to take their dogs to the vet because of the stress and fear they believe visits produce. Even trips to the groomer or performing home nail care are skipped by many pet owners due to the perceived risk of stress or discomfort. If we can provide treatment with lower stress for pets, more pets will receive good care. If we can teach pet owners how to train cooperative behaviors for basic care like grooming, nail care and physical examination, pets will receive better care. If we can provide this care for wild animals, potentially dangerous species, and animals with limited human contact using force free methods, why not pets?

Operant conditioning
(*Review from earlier session, included for those who may not have been able to attend the earlier session)

Operant conditioning is casually referred to as “trial and error learning.” It is comprised of two basic principles: reinforcement and punishment. With operant conditioning, the learner operates on/within his environment. Each behavior performed is associated with its immediate consequence. How the learner views the nature of a particular consequence will dictate whether the learner is more or less likely to engage in the particular behavior in the future. If the behavior is MORE likely in the future, the consequence was reinforcement.

If the behavior is LESS likely in the future, the consequence was punishment.

<table>
<thead>
<tr>
<th>R+ Positive (something is added)</th>
<th>R- Negative (something is removed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reinforcement (behavior is strengthened)</td>
<td>Reinforcement (behavior is strengthened)</td>
</tr>
<tr>
<td>Give a dog a treat after sitting</td>
<td>A loud beeping sound comes from the car</td>
</tr>
<tr>
<td>Dog likes treats</td>
<td>I buckle my seatbelt, beeping stops</td>
</tr>
<tr>
<td>Dog sits more frequently in the future</td>
<td>I buckle my seatbelt more quickly in future</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>P+ Positive (something is added)</th>
<th>P- Negative (something is removed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Punishment (behavior is weakened)</td>
<td>Punishment (behavior is weakened)</td>
</tr>
<tr>
<td>I run a red light and am pulled over</td>
<td>A dog jumps on me</td>
</tr>
<tr>
<td>I receive a ticket for running the light</td>
<td>I remove all attention from the dog</td>
</tr>
<tr>
<td>I am less likely to run lights in the future</td>
<td>The dog is less likely to jump in the future</td>
</tr>
</tbody>
</table>
Within the categories of reinforcement and punishment, there are two varieties of each: positive and negative. If the consequence added something to the learner’s environment/experience, it was positive. If it subtracted or removed something from the learner’s environment/experience, it was negative.

These four principles: positive reinforcement, negative reinforcement, positive punishment, and negative punishment make up the four quadrants of operant conditioning. When training animals, we generally try to work within the quadrant of positive reinforcement a vast majority of the time. It is very uncommon for errors in positive reinforcement training to have problematic consequences. However, it is very common for training errors in negative reinforcement and both punishment quadrants to result in problematic issues in the future. If punishment is used, negative punishment can be used with caution by the trainer with good finesse and a clear understanding of the animal. Positive punishment should be avoided in general because the problematic fallout is potentially severe and avoidable by choosing other methods.

Consent options
There are any number of consent behaviors which we can train. These behaviors are limited really only by the physical capability of the animal, the positioning requirements of the procedure, and the imagination of the trainer.

Some common examples of consent points:
- Body target (table, mat, platform)
- Station, medication station
- Hand target
- Nose target
- Offering a body part
- Chin rest
- Remaining stationary without moving away
- Ability to perform an alternate cued behavior
- Remaining in the treatment area
- Eating treats

Some of these consent points involve training, while others are simply observations for changing body language and arousal levels. For simple procedures such as a vaccination, observing body language may be sufficient to establish consent. For more advanced procedures and particularly serial procedures such as pilling, eye medication, ear medication, nail care, home injections, toothbrushing, etc., a specific trained consent point is needed.

Choose a consent point which is appropriate for the patient and for the procedure.

Concept of consent
Teaching patients the concept of consent is easier than it sounds. First, we will establish a consent behavior (several examples will be provided as videos during the lecture). For example, standing on a platform. When the patient is on the platform, the trainer (technician, veterinarian, etc.) will work through the DS/CC, capturing, shaping, or luring process to teach the desired veterinary behavior. If the patient moves off the platform, all training stops.

After only a few repetitions, most patients begin to identify the concept of consent. Stay on the platform = agree to participate in treatment. Move off the platform = communicate you don’t want to participate.

So why would any animal choose to participate? Because all reinforcement happens at the station. If the animal leaves the station, treatment stops – but SO does all reinforcement.

Capturing and shaping
Once a consent point is established, it is time to begin capturing or shaping the desired medical behavior. A number of video examples will be provided during the lecture.

Capturing means that the learner already sometimes does the desired behavior, and we simply want to place it on cue. This is simply a matter of making sure the behavior is marked in some way so the learner detects it is salient and that the behavior is reinforced in some way so it becomes stronger. An example of capturing could be teaching a bird to sit more frequently on a specific perch in the enclosure because that perch is connected to a scale for body weight measurement.

Shaping is the process by which a novel behavior is acquired by breaking the end behavior down into a series of successive approximations. Successive approximations is a fancy way of saying “baby steps” toward the goal behavior. First you have to crawl...
before you learn to run, right? Shaping is the process of installing all of those baby steps toward the goal, with the result being the goal behavior.

A few examples of successive approximations for a goal behavior that is a cooperative medical behavior:

<table>
<thead>
<tr>
<th>Completed Behavior</th>
<th>Cephalic Blood Collection</th>
<th>Nail clipping</th>
<th>Open Mouth</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Successive Approximations</strong></td>
<td>• Desensitize materials if needed</td>
<td>• Desensitize materials if needed</td>
<td>• Teach pinched fingers target</td>
</tr>
<tr>
<td></td>
<td>• Move to collection area</td>
<td>• Move to treatment area</td>
<td>• Close mouth touch fingers</td>
</tr>
<tr>
<td></td>
<td>• Sit in collection area</td>
<td>• Allow handling of proximal limb: 1 sec</td>
<td>• Open mouth touch fingers: brief</td>
</tr>
<tr>
<td></td>
<td>• Present front leg: 1 sec</td>
<td>• Allow handling of proximal limb: duration</td>
<td>• Open mouth touch fingers: duration</td>
</tr>
<tr>
<td></td>
<td>• Present front leg: duration</td>
<td>• Allow handling – sliding hand from proximal to distal: 1 sec</td>
<td>• Open mouth touch fingers, move fingers slightly further apart: brief</td>
</tr>
<tr>
<td></td>
<td>• Present front leg while vein is occluded: 1 sec</td>
<td>• Allow handling – sliding hand from proximal to distal: duration on distal</td>
<td>• Open mouth touch fingers, move fingers slightly further apart: duration</td>
</tr>
<tr>
<td></td>
<td>• Present front leg while vein is occluded: duration</td>
<td>• Allow distal limb handling followed by digit handling: 1 sec</td>
<td>• Repeat, open fingers 1”: brief</td>
</tr>
<tr>
<td></td>
<td>• Present front leg, vein occluded, syringe mime</td>
<td>• Allow digit handling: duration</td>
<td>• Open fingers 1”: duration</td>
</tr>
<tr>
<td></td>
<td>• Present front leg, vein occluded, mime syringe, alcohol placement</td>
<td>• Allow digit handling, mime syringe, mimeclippers: 1 sec</td>
<td>• Continue in 1” increments with brief and then duration until desired opening size is reached</td>
</tr>
<tr>
<td></td>
<td>• Present front leg, vein occluded, syringe mime, alcohol placement, simulate needle stick: 1 sec</td>
<td>• Allow digit handling, mimeclippers: duration</td>
<td>• Open fingers at desired opening size, hold in position: duration</td>
</tr>
<tr>
<td></td>
<td>• Present front leg, vein occluded, mime syringe, alcohol placement, simulate needle stick: duration</td>
<td>• Allow digit handling, mimeclippers, tap nail: brief</td>
<td>• Hold in position, duration, touch mouth with opposite hand: brief</td>
</tr>
<tr>
<td></td>
<td>• Present front leg, vein occluded, real syringe, alcohol placement, simulate needle stick, duration</td>
<td>• Allow digit handling, mimeclippers, tap nail: duration</td>
<td>• Hold in position, duration, touch mouth with opposite hand: duration</td>
</tr>
<tr>
<td></td>
<td>• Present front leg, vein occluded, real syringe, alcohol placement, simulate needle stick, blood collection</td>
<td>• Allow digit handling, clip nail: brief</td>
<td>• Hold in position, duration, touch mouth with opposite hand in multiple locations: brief and then duration</td>
</tr>
</tbody>
</table>

By applying the principles of operant conditioning, shaping, capturing and using force-free, positive reinforcement methods, we can teach animals to cooperate for a variety of procedures. The procedures demonstrated in video today included cephalic venipuncture of the dog and cat, nail clipping of the dog and cat, open mouth of the dog, chin targeting of the dog, lateral recumbency of the dog, dorsal recumbency of the dog, ear medication of the cat, ear medication of the dog, carrier loading of the cat, body weight of the cat and dog, oral medications of the cat and dog, auscultation/brief examination of the cat and dog and toothbrushing of the cat and dog. These video demonstrations are available on request to conference attendees. Simply contact the speaker at using the information provided in the catalog.

**Conclusion**

Cooperative Veterinary Care is the future of veterinary medicine. Zoos figured it out. Pediatricians figured it out. Dentists figured it out. Now is our moment to do this! I think most veterinary professionals joined this profession in order to help animals. Teaching animals how to cooperate without the need of restraint is a huge step forward in providing humane treatment with minimal harm done.
References
No Restraint? No Problem! Cooperative Veterinary Care: Setting the Stage
Monique Feyrecilde, BA, LVT, VTS (Behavior)
Mercer Island Veterinary Clinic
Auburn, WA

What is cooperative care?
Cooperative Veterinary Care is providing care for patients using little or no restraint, distractions, or trained behaviors depending upon the needs of the individual patient. CVC can range from simply offering treats as a distraction during a procedure all the way up to completely training all procedures using operant conditioning.

Before we are ready to transition to CVC, we need to prepare ourselves and our practices to provide these services. This will mean acquiring a combination of equipment and knowledge.

Addressing the behavioral needs of every patient during every visit can be a challenge. In practice, we must leverage our time as much as possible to assure every patient receives optimal care in a reasonable amount of time. In order to include behavior in routine visits and make behavior consultations go more smoothly, I recommend putting together a Behavior Toolbox which will allow you to rapidly provide Cooperative Veterinary Care. Taking the time to prepare the Behavior Toolbox and then keeping the items close at hand will save time, simplify the job of the veterinary staff, and bond the client to the practice. By making behavior a part of every routine visit, we have the opportunity to educate clients and take better care of our patients using Cooperative Veterinary Care. Being well prepared in advance for behavior consultations will impress clients and help patients relax.

Setting the stage: Items & equipment
Routine visit tools
Veterinary professionals may agree that preventing problem behavior, and particularly preventing fear of the veterinary clinic, is key in providing optimal care for patients and clients. Preventive care can start with simple ideas such as the veterinary receptionist advising clients to fast patients before visits and learning common body language and being instructed in unobtrusive greeting techniques. Food lures can be used to assist the receptionist in obtaining a body weight and the client can be invited into the examination room.

While obtaining a history, the technician has a valuable opportunity to build rapport with both the patient and the client. Additionally, this is a perfect opportunity to observe the patient’s behavior and devise a handling plan for the visit.

Each examination room in the practice should contain a behavior toolkit. To help pets feel most comfortable in the exam room, there are a variety of items and products available for practices. Cooperative Veterinary Care can only be successful when the patient is relaxed enough to try and interact. A welcoming environment will facilitate this type of medical care. Many items can be constructed, while others can be purchased.

Non-skid surfaces
- Yoga Mats
  - These can be sanitized in the washing machine (Air dry or NO HEAT dry only)
- Rubber Backed Mats
  - Examples include anti-fatigue kitchen or bath mats, area rugs with anti-slip treatments.
  - These can be sanitized in the washing machine (Air dry or NO HEAT dry only)
- Table Covers
  - Commercially available adhesive table covers can be purchased through http://vetwarming.com/fear-free-table-covers/
  - Table covers can also be used on the floor
  - These can be sanitized between patients using a surface disinfectant, and discarded at the end of each day.

Pheromones
- Pheromone products are commercially available for both cats and dogs
- Diffusers, sprays, and wipes are all available
  - Using wipes in front of the client helps the client see value added Fear Free™ service

Hiding opportunities
- Towels, Blankets (treat with pheromones, warmed when possible)
- Infant Changing Pads
- Carrier bottoms
- Cubbies
Treasures
(items to help with distractions and building positive emotional responses in the exam room)

Edible
Texture variety: Sticky, smooth, crunchy, moist, etc and can be stored at room temperature
- Squeeze cheese, peanut butter
- Baby food (refrigerate after opening)
- Baby food sausages (refrigerate after opening)
- Canned purse type pet food (refrigerate after opening)
- Tiny training type moist treats (easy to toss)
- Freeze dried liver or other highly palatable meat-based soft dry treats
- Crunchy treats
- Dried fish or fish flakes
- Palatable sticky cat treats (for example, Kong Easy Treat Salmon Formula http://www.kongcompany.com/products/cats/treat-dispensers/treats/salmon-easy-treat/)

Food administration tools
- Squeeze tubes (camping supply, I use GoToob brand)
- 3ml and 20ml syringes
- Food retaining toys (For example, Kong Blue by Kong Company https://www.campbellpet.com/products/Kong-Toys-and-Treats/KONG%C2%AE-Blue)
- Kongs (Kong Company), Twist-n-Treat (Premier Pet Products)
- Paper plates
- Plastic spoons
- Tongue depressors
- Pretzel rods or other edible treat delivery system for enthusiastic eaters

Toys
- Feather, fur, and bell toys for cats
- Fishing pole type cat toy
- Tennis balls/Rubberized tennis balls
- Squeaky or plush toys (great for mouthy puppies)
- Tugging toys

The drawer dedicated to CVC in our exam rooms contains:
Training tools
- Clickers
- Treat bags/Treat holders for use during training
- Station opportunities (mat, rug, towel, etc)
- Targets (spoon, target stick, plastic lids or disks)

Medical management
Some patients have moderate to severe anxiety, and would benefit from the use of medical treatment for fear, stress, and anxiety. There are a wide variety of short-acting anxiolytic supplements and medications available. Practitioners should familiarize themselves with these options and stock several choices in-clinic for ease of use and prescribing. Dosages and medication selection are outside the scope of this lecture, but can be found in the Fear Freesm Certification Course as well as numerous texts, the Veterinary Information Network, and much more. The use of acepromazine as a single agent for fearful and especially aggressive patients is contraindicated.

Knowledge: Setting the stage
Patient assessments
To prepare for providing CVC, the team need to be adept at reading animal body language, and then classifying animals into appropriate training categories. This assessment may require only a few seconds, or require several minutes of working with the pet to determine what works well. The tables below offer guidelines for identifying and classifying level one pets based on body language cues:
<table>
<thead>
<tr>
<th>Stage of Visit</th>
<th>Food Acceptance</th>
<th>Body Language</th>
<th>Proximity Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greeting</td>
<td>☐ From any team member</td>
<td>☐ Relaxed body</td>
<td>☐ Greets immediately</td>
</tr>
<tr>
<td>Touching</td>
<td>☐ From any team member</td>
<td>☐ Relaxed during touch</td>
<td>☐ Does not move away</td>
</tr>
<tr>
<td>Exam Treatment</td>
<td>☐ From any team member</td>
<td>☐ Relaxed during handling</td>
<td>☐ Does not move away</td>
</tr>
</tbody>
</table>

**Level two patients.** These patients will require desensitization and counterconditioning at minimum, and may benefit from medical treatment of fear, anxiety, or stress.

<table>
<thead>
<tr>
<th>Stage of Visit</th>
<th>Food Acceptance</th>
<th>Body Language</th>
<th>Proximity Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greeting</td>
<td>☐ High value from any team member</td>
<td>☐ Relaxed body</td>
<td>☐ Greets immediately</td>
</tr>
<tr>
<td>Touching</td>
<td>☐ High value treats only</td>
<td>☐ Mild tension during touch</td>
<td>☐ Moves away</td>
</tr>
<tr>
<td>Exam Treatment</td>
<td>☐ High value treats only</td>
<td>☐ Mild tension during procedures</td>
<td>☐ Does not move away</td>
</tr>
</tbody>
</table>

**Level three patients: These patients generally require operant conditioning and operant counterconditioning as well as medical management for successful treatment**

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**Distractions, desensitization/Classical counterconditioning, and operant conditioning**

In order to provide a full array of CVC services, the health care professional will need to become proficient in distraction methods, DS/CC/CCC, and OC/OCC. These techniques were discussed in-depth in earlier lectures today, and will be demonstrated using case examples in the following lecture this afternoon. Starting with patient assessment is a good way to determine what training style will best bring success. It is important to understand that Level Three patients are in considerable distress, and are unlikely to respond to any form of training.

The goal of patient assessment is to determine what method will work best once the patient is relaxed enough to train. Patients who are assessed at Level Two or Level Three should have a procedural triage performed. Desired treatments for the day should be divided into wants vs. needs. Only needs should be pursued, and any necessary medical management should be provided. Future training should be undertaken to facilitate future care.

While taking a history, I recommend choosing between a variety of strategies depending on the animal involved.

**Getting started**

Outgoing puppies and kittens and their owners will benefit from food toys, treats and play during the history. While questioning the owner, the technician can provide a stuffed Kong, Twist-n-Treat or simply a paper plate with a very thin coating of something sticky and highly palatable like canned pet food, baby food or peanut butter (a “sticky plate”). Given that the pet is relaxed enough to eat, there are 2 immediate benefits to this approach. We are taking action to prevent phobia of the veterinary office as well as classical conditioning for a positive conditioned emotional response at the veterinary office. In addition, we are demonstrating appropriate use of toys and treats for the pet owner.

For outgoing adult dogs and cats, I will often provide a stuffed Kong, Twist-n-Treat or sticky plate during the history. However, for frightened animals I generally take a different tactic. I always sit on a low stool during the history, but my position in the room varies. For frightened dogs, I will often toss treats onto the floor near the dog from the opposite side of the examination table, with the table between us. This allows the dog to take treats if they feel comfortable enough to do so without having to approach me. Most dogs will elect to approach me voluntarily after a few minutes without being approached. Particularly dogs with a prior history of rushed exams with a lot of manual restraint will respond favorably to these few minutes of relaxation prior to handling.

For frightened cats I will provide a small dollop of baby food or canned pet food just inside the door of the pet carrier, and then mist the small towel with Feliway spray and drape the towel over the pet carrier, leaving the door open and facing away from me. This allows the cat to emerge from the carrier if she feels secure enough, and provides a comfortable hiding place if she is not. Adequate hiding provision is a critical part of keeping feline patients comfortable.

Routine visits often involve acquiring a temperature, pulse, palpation and injections. Using sticky plates or other food lures during these procedures is a terrific way to employ classical conditioning as well as educating the owner about alternatives to significant manual restraint. During the temperature and pulse portion of the history, I will enlist the owner to be in charge of keeping the dog or cat interested in the soft treats/sticky plate while I perform the treatment. For needle sticks, I will do light restraint combined with soft treats/sticky plate while the veterinarian or another technician completes the injection or venipuncture.

Not every patient will be comfortable enough to eat in the hospital, particularly if the patient has a learned aversion to the veterinary office based on prior experience. However, for patients who are comfortable enough to eat, these methods work beautifully.

**My patient won’t eat**

For patients who won’t eat for distraction methods, they are either too stressed (Level Two or Level Three), the environment is too stressful, the food being offered is not a preferred food, or the animal is not hungry. Hospitals should develop a protocol for assessing why patients can’t accept food, and either address the problem through environmental management, satiation prevention, changing the food offered, changing the technique used, or medical management. Often a combination of these techniques is required.

**Next steps**

The next steps in Cooperative Veterinary Care involve teaching animals first that they have a voice in their care, and second, HOW to cooperate for given procedures. The concepts of consent and of specific skills training will be discussed in a separate lecture.
Learning happens constantly
Pets and people are learning all the time. Whether we are learning lessons like in school or learning from our daily experiences or emotional events, learning occurs continuously whenever we are conscious. Pets are no different from people in this way—or any other animal for that matter.

Animals sense and perceive differently
Animals have more delicate senses of hearing and smell than people. Their visual acuity is different from ours as well as taste. The distribution of nerves is different from in people and recognizing these differences is one more way to help the environment and interactions within the hospital be easier for pets.

Types of learning: Classical conditioning
Classical conditioning is a type of learning in which two stimuli are linked such that one predicts the other. In people, lightning predicts thunder. A flash of lightning will provoke a thunder-anticipating response in most people. If a white coat predicts an examination, a muzzle predicts rough handling, a set of gloves predicts handling and injections, etc the pet will be classically conditioned to respond the same way to a white coat, a muzzle or a pair of gloves that they respond to the actual exam, handling event or injection.

The responses created through classical conditioning are generally physical, emotional responses. They are not conscious, intentional responses.

It is important to understand how classical conditioning affects pets by accident and know how to use it by intention to help pets.

Types of learning: Operant conditioning
In operant conditioning, the learner's behavior is changed based on the consequence that follows a behavior. For instance, if something pleasurable follows a behavior, that behavior is more likely to be repeated in the future and reinforcement has occurred. An example is giving a dog a treat after he sits. He will be more likely to sit in the future. If something unpleasant follows a behavior, that behavior is less likely to occur in the future and punishment has occurred.

Operant conditioning is a valuable tool to use in the veterinary clinic to shape patient behavior.

Learning about veterinary visits starts before they come in the door
Pets are absorbing information about the hospital from the moment they exit the vehicle. Further, pets who visit more than once may be learning information before the car enters the parking lot. Pets who rarely leave home like indoor cats may only ride in the car to go to “unpleasant” experiences like the veterinarian or groomer. We should not discount this learning and do what we can to counteract it.

Make the exterior of the hospital welcoming. Examine the area for places dogs frequently eliminate. Clean these areas frequently including scrubbing places where dogs may urine-mark. These urine messages can contain stress hormones that pass “danger data” on to incoming visitors before they ever get to the door.

Make the lobby friendly
Dogs need personal space, cats do as well. Make sure the scale is as friendly as possible—no-slip surface and concealed in the floor if possible. Dogs should be escorted into an exam room as soon as possible as long as the dog is amenable to this.

Cats need a waiting area that is quiet and free from strangers and other pets (including other cats). Often, a feline-only exam room is the way to go for this. Making sure the feline exam room is available as soon as feline patients enter is very helpful.

Make the exam room friendly
Feline patients benefit from hiding. Provide cubbies inside the room for hiding opportunities and allow the pet access to his or her carrier for hiding as well. Using pheromones like Feliway® can help cats feel calm. Place a pheromone diffuser in the feline room.

Canine patients can also be helped by pheromones and products like Adaptil®. Both the canine and feline pheromones can be used together, so exam rooms that serve both species can have a plug-in diffuser for each product. Both products are also available as a spray which should be applied to towels on the exam table or covering the pet carrier. Allow several minutes after application for the alcohol carrier to disperse before use.
Make the treatment room friendly
The treatment room is filled with unfamiliar sights, smells and sounds. Muffling sounds, covering kennel doors to limit visual input and using frequent cleaning with good ventilation will all help. Pheromones can be used similarly to exam rooms.

The treatment room should be strategically used so that kenneled pets don’t have to interact with other patients through their kennel doors. This is a very threatening experience and stressful for the kenneled patient. Restrict pets from greeting through barriers.

Many pets are afraid of things like a white coat or stethoscope. They have been classically conditioned to fear these things. Be aware of where your doctors hang things like a white coat or stethoscope. Hanging a white coat from an IV pole outside of a kennel can give the effect of a doctor looming over the pet continuously throughout the day, increasing patient stress.

Provide hiding places for cats that are kenneled whenever possible. Some patients such as seizure watch cases can’t be hidden from supervision, but most other cats can and should be provided a place to hide inside their housing.

Manage noise. Provide soothing music like Through a Dog’s Ear®. Barking is usually a sign of stress or distress and should be managed as needed.

Make interactions pet friendly
Realize how we look to pets during interactions. Take note what a pet sees from inside his or her kennel, from their perspective in the treatment room and during handling events, and from their perspective in the examination room.

Take the initiative
Understand classical conditioning and use it to your advantage. Pair necessary events with something the pet enjoys so that things like auscultation, temperature taking, physical examination, nail trimming, etc predict enjoyable stimuli like food, toys and play.

Understand operant conditioning and how it can be used to your advantage. Patients requiring serial blood draws can be taught to cooperate for voluntary treatment. Patients requiring medication at home such as pills, liquid, eye drops, ear medication or skin spray can be taught to cooperate for treatment as well. Voluntary veterinary behaviors and behavioral husbandry could take up an entire weekend of lectures all on its own!

Remember that when the sight and smell of the ear cleanser bottle predicts a 3-man-otitis-rodeo, that patient with painful ears is much more likely to become upset next time. This is generally a result of a combination of classical conditioning and operant conditioning influencing behavior. While the decision about the appropriateness of chemical restraint always lies with the veterinarian, fostering a clinic culture where the behavioral needs of the patient are considered is crucial to long-term success with patients.
Is heparinized saline necessary to keep catheters patent?
Proper maintenance of peripheral intravenous (IV) catheter is critical in the treatment of patients in critical care, used for fluid delivery, drug administration, blood product transfusion, and parenteral nutrition. The best method in maintaining the catheter is of interest, including catheter patency, maintenance protocol, and dressing methods. Occlusion of IV catheters is a common complication that necessitates replacement of the catheter and leading to additional patient discomfort and medical care cost. While catheter material and patient-related factors can be contributors to clot formation, one of the key elements to maintaining patency has been flushing of the catheter with heparinized saline. The use of heparinized saline has associated concerns including coagulopathy, drug incompatibilities, allergic reactions, thrombocytopenia, and thrombosis syndrome.

The first veterinary study conducted to determine whether there is any difference in effectiveness between heparinized saline and normal saline compared the use of 10 IU/mL heparinized saline with 0.9% sodium chloride. An 18-ga catheter of 1.25 inch in length was placed in each test subject that were separated into three groups. The first group had their catheters flushed with heparinized saline every 6 hours throughout a 42 hour period. The second group had their catheter flushed with normal saline every 6 hours throughout a 42 hour period. The third group served as a control group used to determine the amount of time it took for a catheter to clot if it were not flushed. Blood was attempted to be aspirated from the catheter prior to each flushing, and also evaluated for any signs of phlebitis.

The study observed that all catheters of both treatment groups were able to be flushed without any resistance or occlusion. The number of catheters that allowed for blood to be aspirated back were higher in the heparinized saline group, but the difference was not statistically significant (9 of 12 vs 5 of 12 at 42 hours, p = 0.065). No signs of phlebitis was seen in any group. The authors concluded that the use of heparinized saline flushes did not yield benefits when compared to 0.9% sodium chloride in maintaining peripheral 18-ga catheters in a 42 hour period.

This veterinary study follows some of the human studies observing that intermittent flushing of IV catheters with normal saline is as effective as flushing with heparinized saline. It also suggests that 18-ga IV catheters might not require any flushing at all for the first 24 hours. There are some studies, however, that observed heparinized saline as being superior, muddying the waters on the issue. Some other limitations should be considered, such as the duration of the study being 42 hours. Many catheters in critical care settings are used longer than 42 hours, making information beyond 42 hours desirable before heparinized saline is replaced with normal saline completely for flushing. The patency of the catheter was determined through a qualitative evaluation of resistance by the investigators, and so objective measurements of clot formation were available. The study also evaluated a single size of catheter, and the data’s applicability to other sizes and lengths are uncertain. For example, studies surrounding heparinized saline use in central venous catheters currently provide even less definitive conclusions due to the variability in maintenance protocols in regards to heparin concentration and flushing frequency. Other factors within the study that can be different from clinical situations include the catheter diameter as well as the disease state of the patient (hypercoagulable patients could be more prone to catheter occlusion). A future study of longer duration measuring the effects objectively is desirable to shed more light on the topic.

Should IV catheters be replaced routinely?
Hospital protocols often recommend replacement of IV catheters in a patient every 72-96 hours as it is thought to reduce the risk of phlebitis and bloodstream infections. The US Centers of Disease Control guideline recommends replacement no more frequently than 72-96 hours. Routine replacement of IV catheters exposes the patient to additional stress and discomfort, venipuncture, and restraint. It also adds financial burden to the owners or at the very least increased staff time and supply demand to the hospital. More recently, many practices have instituted protocols calling for catheter replacement only when clinically indicated, attempting to alleviate the morbidity and costs associated with routine replacement. An assessment of the effects of the two approaches would be beneficial in setting hospital protocols.

Numerous studies related to the topic has been conducted on human subjects, being summarized as a systematic review through the Cochrane Collaboration. The systematic review summarized that there were no significant difference in occurrence of catheter-related bloodstream infection (CRBSI) for clinically-indicated or routine replacement with 1 of 2365 and 2 of 2441 patients, respectively (p=0.64). There was no difference in phlebitis seen with 186 of 2365 cases seen in clinically-indicated replacement and 166 of 2441 cases seen in routine replacement methods (p=0.75). Significant reduction of catheter placement costs were seen in the clinically-indicated group, of approximately AUD 7.00. The reviewers concluded that there is no clinically significant difference between clinically-indicated replacement and routine replacement of peripheral IV catheters.

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Because there is no difference seen between the two methods, a recommendation can be made to adopt a protocol to replace peripheral IV catheter only when clinically-indicated. Patients will avoid being subjected to unnecessary pain and the clients and practice will not incur unnecessary drain on resources. There are currently no veterinary evidence available to provide insight in our practice. There could be differences between species or practice setting such as the higher tendency for veterinary patients to soil or tamper with the catheter insertion site, and any unexpected differences in physiology. With that said, many practices have instituted a clinically-indicated replacement approach without subjective increases in complications. If the clinically-indicated replacement approach is taken, structured protocols on routine inspection of the catheter site of at least every 24 hours for signs of inflammation, infiltration, occlusion, or infection should be followed.

Do antimicrobial impregnated central venous catheter supplies prevent CRBSI?

Central venous catheters (CVC) are often used in critical care for a variety of reasons including blood sampling, central venous pressure measurement, infusion of high osmolarity fluid, simultaneous infusion of incompatible drugs through multiple lumens, and parenteral nutrition. A major concern with placement and maintenance of CVCs in a patient is the possibility of CRBSI adding to patient morbidity and mortality. A variety of strategies have been adopted to prevent this common complication, including catheter maintenance bundles, antimicrobial treatment of the catheter, and antimicrobial treatment of catheter insertion site or dressings.

A Cochrane review of antimicrobial-treated (AMT) (antiseptic or antimicrobial impregnation, coating, or bonding) CVCs compared the difference between AMT and non-AMT CVCs, the difference between the effect of antimicrobial impregnation and antimicrobial modification (antiseptic dressing, hubs, tunneling, needleless connectors, etc), and any differences between identifiable subgroups such as length of catheter use and practice setting. The review consisted of 16,784 catheters and 11 impregnation types. The review summarized that catheter impregnation significantly reduced CRBSI. However, it did not reduce the incidence of sepsis, mortality, and catheter-related local infections. There were significant benefits seen in ICU settings when compared to hematological and oncological units or CVC use for parenteral nutrition. AMT did not affect the incidence of other adverse events such as thrombosis, thrombophlebitis, bleeding, erythema, or tenderness at the insertion site.

A review of antimicrobial dressing in CVC placement in human infants saw that chlorhexidine dressing/alcohol skin cleansing reduced catheter colonization in a similar manner to polyurethane dressing/povidone-iodine cleansing, and was no different in its effect on sepsis and CRBSI. Chlorhexidine dressing seemed to cause higher incidence of contact dermatitis, however. Silver-alginate patches did not cause adverse effects, but their efficacy is unclear. A separate review of dressing and securement devices for CVCs evaluated various devices and their effect on CRBSI, catheter colonization, site infection, skin colonization, skin irritation, failed securement, dressing condition, and mortality. The review summarized that chlorhexidine gluconate-impregnated dressing reduced incidence of CRBSI and catheter tip colonization when compared to standard polyurethane dressing. Medication-impregnated dressing reduced CRBSI rate when compared with non-impregnated dressing. Of all, the use of sutureless securement device was the most effective and chlorhexidine gluconate impregnated dressing the second most effective in reducing CRBSI.

While the evidence evaluated by these reviews are of human subjects, some messages can be extracted for potential benefits in the veterinary field. The use of AMT CVCs might not be as effective as theorized as incidence of sepsis and mortality was not significantly different. The use of antimicrobial-impregnated dressing should be encouraged, and implementation of sutureless securement devices explored. Implementation of antimicrobial-impregnated dressing is a relatively inexpensive intervention available, and should be considered if current protocols include the use of standard polyurethane dressing or gauze.

Should patients with gastroenteritis be fasted?

Patients exhibiting gastroenteritis with signs of vomiting and diarrhea are often placed on a nil per os (NPO) nutritional plan as it is considered to be beneficial for the patient. The reasoning behind this thought are various. One of which is the resting of the bowels by minimizing stimulation for contractions, reducing fecal volume, and frequency of defecation. Another is to reduce the chance of vomiting due to stimulation through distension of the stomach. By fasting, the vomitus is thought to contain less nutrients that can increase the chances of bacterial proliferation and pneumonia if aspirated. The presence of undigested food in the gastrointestinal system is also thought to have detrimental effects such as promoting bacterial proliferation and secondary infections, or inducing osmotic effusion into the gastrointestinal lumen leading to exacerbation of diarrhea. Offering of food while a patient is nauseated can also lead to food aversion, contributing to the delay in regaining of appetite when the patient is feeling less ill. Because of these reasons, a traditional approach to gastroenteritis is to withhold food for 24-72 hours before offering food.

However, there are numerous veterinary studies that support the institution of enteral nutrition early in the stages of hospitalization. A study involving patients with hemorrhagic gastroenteritis having a hydrolyzed protein diet introduced early in hospitalization observed that it did indeed increase the frequency of vomiting, but only initially. These patients saw a reduction in the frequency of vomiting and regained tolerance to feeding within 2 to 3 days. It is thought that the introduced food serves as a prokinetic and thus reduces the amount of vomiting when compared to a fasted state. Another study involved patients with parvoviral enteritis being split into a group that was fasted, and a group that was given enteral feeding. The investigators observed that patients that were fed stopped
vomiting significantly sooner than patients that were fasted, leading to the conclusion that early enteral nutrition is beneficial for cessation of vomiting. However, food-high in fat, soluble fiber, or poorly digestible starch can promote emesis instead. Gastric distention does contribute to stimulation of vomiting as well. With these points in mind, feeding small, low fat meals frequently is recommended.

Providing nutrition early will also prevent patients from experiencing vigorous peristaltic action that is described by people as “hunger pains”, as the presence of food promote normal peristaltic action. The presence of volatile fatty acids such as propionic acid and butyric acid provides an acidic environment in the gastrointestinal lumen suppressing the proliferation of pH sensitive pathogens such as Campylobacter and Clostridium spp. likely having some beneficial effects in preventing secondary bacterial infections. In terms of structure of the gastrointestinal mucosa, fasted animals experience a reduction in villous height and crypt depth, decreased antioxidant content in mucosal tissues, and increased induction of enterocyte apoptosis. The gastrointestinal mucosa provided food will instead experience healthier mucosal turnover and strengthening of the mucosal barrier. The gastrointestinal mucosa seems to rely on luminal nutrients to passively obtain glutamine, amino acids, essential fatty acids, folate, zinc, vitamin A, and vitamin B₁₂, which are all necessary for healthy mucosal turnover. Each of these factors serve to reduce chances of bacterial translocation in patients provided nutrition. Presence of luminal nutrients also reduce the expression of adhesion molecules and subsequent neutrophil sequestration and activation, and keeps the function of T and B lymphocytes to produce IgA and cytokines intact, providing benefits to immunologic functions.

These reasons support providing enteral nutrition as soon as fluid deficits are replenished. Many negative effects of feeding can be alleviated through providing smaller amounts of a highly digestible diet that is low in fat. Other evidence supports the importance of earlier nutritional intervention in many critical illnesses.

**Nasogastric tube or nasoesophageal tube?**

Nasoenteral tubes are used in hospitalized patients to provide enteral nutrition with liquid diets on a short term basis, especially when anesthesia is undesirable. Nasoenteral tubes can be inserted to be terminated either in the esophagus or the stomach, called nasoesophageal (NE) and nasogastric (NG) tubes, respectively. Each of these tubes are associated with shared complications such as epistaxis, dacrocystitis, rhinitis, aspiration pneumonia, occlusion of tubes, diarrhea, vomiting or regurgitation, and unintended removal.

The selection of NE versus NG tube placement is a choice presented to the veterinary team. NG tubes had been avoided by some because of the potential for an increase in the risk of regurgitation, gastroesophageal reflux, and resultant esophagitis or stricture as the tube being placed across the lower esophageal sphincter prevents full closure. NE tubes will allow these risks to be circumvented, though the potential for unintended displacement of the tube might be increased, and NE tubes will also deny the ability to decompress the stomach or measure gastric content that NG tubes provide. The optimal type of nasoenteral tube chosen has been largely up to the clinician’s preference.

A retrospective veterinary study evaluated the incidence of complications between the two methods to determine any advantage of one over the other. The study evaluated the occurrence of complications including epistaxis, vomiting, regurgitation, diarrhea, clogged tube, tube malpositioning, aspiration pneumonia, hyperglycemia, and refeeding syndrome. The study also evaluated differences including feeding method (bolus vs CRI), amount fed (% RER), and administration of medications by tube. The study observed no significant difference of complication rate between NE and NG tubes, nor other factors (feeding methods, amount fed, and medications).

The lack of a difference seen in the study makes us think that there is likely no difference between the placements of NE or NG tubes. While there is a possibility that subclinical esophagitis existed, there were no patients that showed clinical signs of esophagitis. There was a significantly higher amount of deaths seen in patients receiving NG tubes, though this is likely to be attributed to NG tubes being utilized in more critically ill patients and an artifact due to the retrospective nature of the study. Because NG tubes provides the benefit of allowing gastric decompression and there seemingly being no clinical signs of resultant esophagitis, clinicians should be feeling less hesitation on using NG tubes over NE tubes.

**Can RBCs be given through an infusion pump?**

Whether there is an optimal method of red blood cell transfusion administration has been a point of discussion. Studies evaluating the effect of various administration methods on the integrity of blood cells exist, focused on the in vitro effect of infusion pumps, measuring the degree of free RBC content (free hemoglobin, potassium, lactate dehydrogenase, bilirubin) and osmotic fragility. The results vary from observing significant increases to insignificant increase in values, while transfusions with red cells with longer storage time resulting in a larger increase of hemolysis markers than those with shorter storage times. The variability in results, in addition to the anecdotal evidence of patients benefiting from RBC transfusions administered with infusion pumps are a cause for varying opinions.
A study assessing in vivo survival time of RBCs infused with various infusion methods, compared the use of gravity flow, volumetric peristaltic pump, and syringe pump in autologous transfusions in dogs. Blood was collected from 9 healthy dogs, washed, and separated into 3 portions labeled with different densities of biotin. These labeled red cells were transfused through either gravity flow with a 170-260 µm filter, volumetric peristaltic infusion pump with a 170-260 µm filter, or a syringe infusion pump with an 18 µm aggregate filter at 2mL/kg/hr. Blood was sampled from test subjects at day 1, and every 7 days until day 49, measuring the proportion of red cells with biotin labels through flow cytometry. Additional in vitro testing was conducted, measuring plasma hemoglobin and osmotic fragility testing.

Labeled RBCs infused through gravity flow, volumetric pump, and syringe pump were detectable in 100% (8/8), 50% (4/8), and 14.3% (1/7) samples, respectively post-transfusion. The quantity and half-life between RBCs infused by gravity flow and volumetric pump that were detectable (4/8) were not different. The RBCs infused via syringe pump detected at 24 hours post transfusion was no longer detectable at 7 days, indicating complete removal of those cells from circulation sometime between 24 hours and 7 days post transfusion. No differences were seen in in vitro values examined.

The study concluded that delivery of RBCs with a syringe pump and microaggregate filter is associated with significant decrease in in vivo survival time. Volumetric pump delivery was associated with a 50% probability of loss of transfused RBCs within the first 24 hours, and gravity flow allowed for highest chance of RBC survival. The reason behind this difference is speculated to be the mechanical shear damage to the RBC membranes when transfused through the microaggregate filter, causing preferential removal of damaged cells upon entry into the circulation and exposure to the mononuclear phagocytic system. Though unconfirmed, there is a potential for microclots to have formed in the blood during resuspension in sub-room temperature plasma, which placed a higher degree of shearing stress on the RBCs going through the filter, causing this effect. Early denaturation and oxidation of hemoglobin due to the mechanical stress induced by syringe pump and volumetric pump methods, leading to IgG binding to the red cell surface and removal from circulation, is another possible cause for early removal.

Small sample sizes limiting the power of the results is a common limitation in the veterinary field, and this study is no exception. The results are most relevant to exact methods used in the study, and we can only make speculations on alternate setups to remove the use of microaggregate filters with the syringe pump (use of an in-line pediatric 170-260 µm filter or extraction of blood through a 170-260 µm filter administration set into a syringe, for example).

The authors of the study recommended against using a syringe pump with 18 µm aggregate filters in the light of the results of their study, though considering the limitations, drastic changes to clinical protocols was not stated to be necessary. The current best practice considering this evidence would be to administer blood products via gravity flow for larger volume, higher flow rate transfusions as long as consistency in flow rate is monitored closely (as it can be influenced by catheter patency, positioning and motion by the patient, and amount of blood left in the bag). The syringe pump method is particularly useful when performing small volume transfusions such as in felines. A similar study performed with feline blood stated their observation of RBC survival time being unaffected by the syringe pump method.

There are a couple of infusion pumps approved for blood product, one of which is an internal approval, and the other of FDA approval for human blood products. These pumps could be the next best solution and validation with veterinary blood products is warranted.

Premedicating reduce chances of reactions?
Premedication, or administration of antihistamines, glucocorticoids, or antipyretics in anticipation of immunologic complications to counter histamine and inflammatory mediators and suppress the effects, have been a traditional practice in transfusion medicine. There are a number of human studies observing no difference in incidence of type I hypersensitivity reactions (allergic reaction) or febrile non-hemolytic transfusion reactions (FNHTR). Some clinicians reason that administration of premedication potentially masks early symptoms of immunologic complications delaying required interventions for treatment, advocating against it. Evaluation of the difference in severity between recipients with premedication or without premedication has not been performed, and remains a question whether this reasoning is valid. Human evidence is unfortunately not always directly translatable into veterinary practice, though expectations of similar physiological mechanisms exist. A recent veterinary retrospective study evaluating the effect of premedication on acute transfusion-related reactions saw no beneficial effect. There might be a beneficial effect to administration of diphenhydramine in decreasing chances of acute allergic reactions, though further studies were recommended by the authors since the incidence of allergic reaction in the non-premedicated group was already low (2.6%). Studies evaluating effects of premedication and efficacy in prevention of hemolytic transfusion reactions are not apparently available, and the theoretical benefit is no justification for foregoing proper compatibility testing.

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**Keywords:** Transfusions, Evidence-based Medicine, Premedication, Transfusion reactions, Blood pump
Why Your Parvo Patient Should Be Fed Right Away
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Canine parvovirus (CPV) infections cause severe gastroenteritis, lead to dehydration, shock, disseminated intravascular coagulation, bacterial translocation and sepsis when left untreated. With aggressive treatment, the mortality rate can be reduced to between 0% and 30% from 90%. Therapeutic options in addition to fluid and pharmacologic therapy speeding gastrointestinal recovery is desirable to reduce patient mortality and morbidity, as well as financial strain to the clients for prolonged aggressive treatment. Use of elaborate treatment options such as oseltamivir (Tamiflu), interferon omega, recombinant bactericidal proteins, equine lipopolysaccharide antitoxin, human recombinant factors, and antibody rich plasma have not shown promising results. One simple to implement, therapeutic option that makes a significant difference in survival chance is enteral feeding started within hours of admission, even when vomiting.

Feeding when vomiting
Feeding a patient when they are vomiting goes against most traditional approaches to nutrition in a patient suffering from gastrointestinal ailments. A typical approach in such a case is to withhold food, designating an NPO (nil per os, or no food or water from the oral route) to prevent nausea from introduction of food and distension of the stomach. Small amount of easily digestible food is introduce, being increased to a normal amount gradually.

There are several reasons why withholding of food is thought to be beneficial. These include the idea of resting the bowel, as it is irritated and has a reduced ability to absorb the nutrients. Vomiting is also thought to be reduced in frequency when the stomach is empty, giving decreased chances of vomiting and potential aspiration in a critically ill animal. In addition, food contains fat and fibers that are difficult to digest potentially increasing the chances of vomiting even further. In a patient with dysfunctional gastrointestinal system, food will pass into the intestines undigested, leading to bacterial proliferation and osmotic diarrhea by pulling water into the intestinal lumen. All of these reasons combined, it may seem reasonable to withhold food in a patient with gastrointestinal issues. However, many of these beliefs actually might not be true.

One argument for feeding early, is that the guts are actually not able to rest when being fasted. Fasting causes a phenomenon called “hunger pains”, which arises from intense peristaltic contractions migrating down from the pylorus to the ileum. The peristaltic activity is decreased when nutrients are present in the intestinal lumen, allowing for better rest of the guts, and nutritional absorption. If fasting leads to less rest and pain to the patient, feeding is the better option.

Does feeding actually lead to more vomiting? A study evaluating this with patients with hemorrhagic gastroenteritis indeed did observe an increased frequency of vomiting upon feeding when compared to fasting. However, they also saw that the frequency of vomiting decreased below the fasted dogs by day 2. Feeding creates a prokinetic effect, and reduces emesis, leading to an overall shortening of the time before the patient stops vomiting. The presence of food in the gastrointestinal lumen also decreases insult to the mucosa from toxins, reducing vomiting. The concept behind food minimizing chances of vomiting when certain medication is given along with it applies here as well. Feeding causes more vomiting initially, but leads to a quicker subsiding of vomiting.

Food high in fat and soluble fibers and large volume feedings can indeed increase chances of vomiting. Malabsorption and gastrointestinal distention stimulates vomiting. Small, frequent feedings are recommended to reduce gastric acid release, leading to reduced vomiting. In many cases, employment of anti-emetic drugs help in preventing vomiting, allowing earlier enteral nutrition. Adequate antiemesis is especially important for inappetant patients requiring placement of NE or NG tubes.

Undigested food being present in the intestinal lumen does increase nutritional resources for microorganisms leading to bacterial proliferation. However, feeding increases levels of volatile fatty acids such as butyrate and propionate, reducing the population of bacteria sensitive to acidic environments (Campylobacter and Clostridium spp.). The presence of food helps maintain enteric barriers, preventing bacterial translocation (movement of bacteria from the guts into the blood stream) and subsequent sepsis. Diarrhea in dogs is attributed to unabsorbed nutrients and endogenously derived osmotic elements instead of osmotic pressure created by undigested food. Insult to the intestinal mucosa preventing absorption of water and increased effusion through leaky blood vessels (increased vascular permeability) is alleviated with enteral nutrition, which helps reduce diarrhea when compared to a fasted state.

Fasting causes a plethora of negative effects that outweigh the small benefits it may have. Fasting causes reduced expression of digestive enzymes, impairing digestive function when food is reintroduced. Presence of nutrients reduces inflammation by inhibiting expression of adhesion molecules, preventing activation of neutrophils which contribute to mucosal damage and impair immune response. Malnutrition leads to protein, essential fatty acid, mineral and vitamin deficiencies preventing healthy turnover of gastrointestinal mucosa. Feeding leads to faster intestinal recovery, even when compared to parenteral nutrition, indicating benefits of passive luminal nutrition. Feed those guts!
Specifically for parvoviral enteritis, early enteral nutrition reduced the time for patient to have normal attitude, appetite, ceasing of vomiting and diarrhea, increased body weight, and improved musical permeability when compared with fasting.

**Nutritional tubes**

Nutritional interventions are a vital part of successful treatment of critical care patients, but often overlooked. An animal who is anorexic for as short as 3 days can develop nutritional deficiency related detrimental effects (metabolic derangements, depressed immune system, catabolic wasting, and deteriorating GI system, to name a few), and should receive nutritional support at latest by 5 days into anorexia. While enticing voluntary eating is most comfortable and beneficial for patients, this is not always sufficient to meet nutritional and caloric needs. When a patient is anorexic, there are several methods at which nutritional supplementation can be performed, divided into enteral and parenteral routes.

The least invasive of tubes utilized in nutrition are nasoenteral tubes. Nasoenteral tubes are inserted through the nares and down into the GI tract. Tubes terminating in the esophagus are called nasoesophageal (NE) tubes, while nasogastric (NG) tubes terminate in the stomach. Nasoenteral tubes are used for short term feeding and are able to be used immediately after placement and typically no longer than 10 days. The tubes are typically too narrow to feed blended canned foods, and require a liquid diet to be infused. When a nasogastric tube is in use the gastric content may be evacuated and measured to determine the degree of functional gastric motility. The placement is typically well tolerated with none to minimal sedation. Contraindications include patients with intractable vomiting, poor mentation, respiratory distress, facial trauma, or nasal diseases. One of the most important aspects of nasoenteral tube placement lies in prevention of tracheal placement. Food infused into the trachea can very easily turn into life-threatening respiratory compromise. Radiographic confirmation of appropriate placement should always be performed to prevent this. Other complications include epistaxis, rhinitis, and vomiting. If vomiting occurs, the placement of the tube should be rechecked make certain the tube did not come up the esophagus and inhaled into the trachea. The tube can also clog due to its narrowness, and requires constant infusions or flushing after bolus feedings.

Esophagostomy tubes may be opted to be placed through the side of the neck in a surgical procedure for patients requiring longer term nutritional intervention. Esophagostomy tubes can be used immediately upon placement and for up to 20 weeks when cared for properly. The larger diameter when compared to nasoenteral tubes allow for feeding of blended canned foods, providing the ability to meet nutritional needs more easily. It is also useful in patients with facial trauma or nasal diseases as it bypasses the mucus into the GI tract. The procedure does require anesthesia to perform. Another significant advantage is the ability for an owner to take a patient home with an esophagostomy tube for long term care and relieving the need for hospitalization for nutritional management. The tube should be flushed with 5-10mL of water after feeding to prevent the tube from being clogged. Main complications include blocked tubes, displacement of the tube by vomiting or intentional removal by the patient, and stoma site infections. The stoma site (insertion site) of the tube requires regular monitoring for redness, swelling, and signs of infections.

Gastrostomy tubes (G tubes) are another surgically placed enteral feeding tube. G tubes extend from the skin on the side of the abdomen and into the stomach. G tubes are used for long term enteral feeding, capable of providing nutrition for years after placement. Polyurethane or silicone based G tubes are required for long term use as they are resistant to loss of integrity from digestive enzymes. Fibrin sealing of the stoma site will occur within 12-24 hours after placement, and feeding should not be started until this seal is formed to avoid contamination and infection of the site. This option may be taken when the patient has esophageal disorders, facial or oral trauma. There is a higher cost associated with the procedure, which may make it cost prohibitive. A this is a more invasive surgical procedure, there is a higher anesthetic risk involved, and should be avoided with patients with healing impairments. Patients vomiting consistently should be held off for the procedure until vomiting is under control. G tubes require similar attention to the stoma site as esophagostomy tubes. Complications include vomiting, aspiration pneumonia, peritonitis, accidental tube removal, pressure necrosis, and stoma site infection.

Jejunostomy tubes are more rarely placed, by passing the stomach and into the jejunum. The tube may be used from week to months, and is used when resting of the upper GI tract is necessary. These include patients with pancreatitis, uncontrolled vomiting, gastroparesis, and recent gastric surgery. Feeding can commence 12-24 hours after placement. Liquid diets are necessary since the tube diameter is narrow. Constant infusions alleviate the risks of cramping and diarrhea. Patients with jejunostomy tubes require close monitoring and will need to be hospitalized. Complications include osmotic diarrhea and vomiting. Obstruction of the tube is a common complication, and can be best avoided through periodic flushing (every 4 hours). If there is leakage of GI tract content, a peritonitis can develop and is a serious complication. When the GI tract is dysfunctional all together, parenteral nutrition should be used, and can be provided through a dedicated sterile port through a central line.

Enteral nutrition is very important in mainlining a healthy GI tract and mucosa. Many of the traditional thoughts of benefits to withholding food does not hold up to be as detrimental when compared to the benefits of early enteral feeding. Because of this, knowledge of use and maintenance of enteral feeding tubes will allow one to help influence a positive patient outcome. In the case of parovirus gastroenteritis, anything more invasive than nasoenteral tubes are rarely used.
Nutritional plan
When the decision is made to feed the patient, there are a few key points to consider. What will we feed? How much of it will be fed and how fast?

Current recommendations include oral rehydration over 3-4 hours, and then introducing food. It is unreasonable to attempt feeding their full maintenance energy requirement for patients suffering from acute diarrhea or frequent vomiting. The amount that can be reasonably be fed initially should be targeted for 1/4 resting energy requirement (RER), as a highly digestible, low-fat diet in order to ensure healthy gut recovery and minimal stimulation of vomiting and diarrhea.

An animal’s RER can be calculated by the formula: RER = 70 x (BW in kg)^0.75 kcal per day. As an example, a patient that is 15kg would have a RER of 70 x 15^0.75, or 533.5 kcal per day. ¼ of this will be 133.4 kcal, leading to a feeding rate of 5.6 kcal per hour. Depending on the energy density of the diet being fed, the volume will differ.

The type of food fed in these cases ideally should be highly digestible. There are many commercial diets available which are highly digestible (gastrointestinal diets). The diet should also be low in fat content, with less than 20% of metabolizable energy coming from fat. Excessive fiber content should be avoided since it can cause delayed gastric emptying, diarrhea, flatulence, and abdominal pain. The recommended level is no more than 8% dietary fiber.

In critical care, especially for feeding through a nasoenteral tube, liquid diets are employed. Products such as Clinicare has been a commonly used diet due to the simple calculation of the volume required to fulfill RER through its caloric density of 1 kcal/mL. The product also comes readily made as a liquid in a can, making preparation simpler. There are newer products on the market such as Emeraid Critical Care HDN specifically formulated for dogs and cats and being highly digestible. This product is formulated for critical care patients at a higher default caloric density of 1.5-2.4 kcal/mL (concentration is adjustable as it is mixed with water on preparation), allowing for lower volume feeding. The choice of liquid diet may vary depending on clinician preference.

Regardless of what product is used, early implementation of nutrition is the key element to encouraging swift recovery of patients with parvoviral enteritis, providing passive nutrition for the enterocytes which prevents mucosal breakdown and bacterial translocation. Technicians play a large role in advocating for patients and their proper nutrition to influence patient outcomes.
The primary goal of any dentistry is to leave the patient with a healthy and comfortable oral cavity. Most practitioner’s also want to salvage as many teeth as possible. Sometimes this is attainable with advanced dental therapies to treat periodontic and endodontic disease. As technicians we are responsible for alerting the veterinarian to observed pathology and assisting in needed treatments. We must be aware and anticipate how these therapies are performed to facilitate these treatments. We must also educate the client of the commitment to follow up care prior to undertaking these types of cases.

**Periodontal disease**
The treatment of periodontal disease is designed to manage the patients’ immune response to inflammation caused by plaque and bacteria by removal of these disease causing agents. Professional periodontal therapy of supra and subgingival scaling, root planning, polishing, sulcular lavage, and advanced periodontal treatments should always be performed under general anesthesia. The following are examples of advanced periodontal disease that require further treatments.

**Gingival hyperplasia**
Benign gingival enlargement/overgrowth that create “pseudopockets” with increased plaque and calculus accumulation leading to periodontal disease.

**Gingivectomy/gingivoplasty**
Treatment of choice for gingival hyperplasia. To re-contour the gingival margin to a normal sulcus depth the pocket is measured and then the tissue is marked with a needle or bleeding point forceps, 3 mm coronal to the base of the pocket at several locations around the tooth. A 45 degree beveled incision is used to excise the tissue and create a natural contour to the gingival margin. This can be performed with #11, #15 scalpel blades, coarse diamond burs, orban knives, laser and radiosurgery. Gingival hemorrhage can be significant but generally stops on its own within a few minutes with digital pressure applied to the area.

**Periodontal pockets**
Pockets less than 5mm can generally be cleaned with closed root planning. This is performed with an ultrasonic scaler as well as a dental curette in a cross-hatch pattern to leave the root surface smooth. Additionally, a placement of a periodontic to further treat the infected area due to their antibiotic and anticollegenase activity.

Pockets greater than 5mm require visualization of the root for adequate cleaning.

**Open root planing**
A full flap or an envelope flap incision is made with a #11, #15, or #15c scalpel blade, the gingival is carefully reflected with a sharp periosteal elevator and handled as gently as possible. The root surface and pocket are cleaned with a curette in a cross-hatch pattern with an effort to remove diseased plaque and calculus but not healthy cementum. Cementum is what attaches the tooth to the periodontal ligament. An ultrasonic scaler with a periodontal specific tip can be used to clean and debride the pocket and root. Ultronics provide water lavage, cavitation that disrupts bacterial cell walls, minimal gingival trauma, and are more efficient at cleaning the root. A coarse diamond bur is used to smooth any bony edges of the alveolar bone (alveoplasty). Root conditioning is then performed using citric acid to remove the smear layer and expose the dentinal tubules. Care must be taken to avoid contact of citric acid with the gingival tissues as this may can have a necrotizing effect. The gingival is then repositioned and sutured with a 4-0 to 6-0 quick dissolving suture material such as polyglicapron.

**Regenerative osseous surgery**
Alveolar bone loss is non-regenerative and will not rebuild without help. The goals of regenerative surgery are to reduce the progression of the periodontal lesion and encourage healthy periodontal attachment. Alveolar bone loss can be vertical or horizontal and further defined by the number of bony walls of the defect. Radiography and periodontal probing are the diagnostic tools for evaluation of bone loss. Three and four walled pockets are excellent candidates for guided tissue regeneration whereas 2 walled pockets have a good to guarded outcome. Those with 50% or greater bone loss should be extracted. The bone graft particulate is placed into the bony defect after thorough open root planning to remove all disease elements. There is biosynthetic glass particulate material or biologic allografts and autographs that can be used in the bony defects. Placement of a resorbable barrier membrane is recommended to allow the alveolar bone and periodontal ligament to regenerate first. Without a barrier membrane the quick growing gingival tissues will infiltrate the area first and not allow the periodontal ligament to regenerate. The defect is then closed without tension. Bone graft material can also be used in the alveolus from large extraction sites to provide a matrix for bone healing. The extraction site must be cleaned of all infectious debris and tissue for the graft to adhere. Meticulous homecare of brushing and oral antibacterial rinse should be started within 5 days. Follow up radiographs and periodontal evaluation should be performed 4-6 months later.
**Endodontic disease**

Endodontic therapy involves treating the pulp of the tooth. The pulp can be described as the living part of the tooth with blood, nerve and lymphatic components. When the tooth suffers a traumatic event the pulp can be exposed to pain, inflammation, and infection.

**Restorative therapy**

For teeth that have an uncomplicated crown or enamel only fractures (FX/UCCF) (FX/EF) or teeth with enamel hypoplasia placement of a composite bonded sealant restoration can be utilized. The fracture does not extend to the pulp of the tooth but the dentin layer is exposed. The tooth should be evaluated radiographically to ensure there is no endodontic disease. The tooth is carefully probed and explored to “map” the defect. The defect is then contoured and beveled with a white stone finishing bur in the high speed handpiece. Then sanding discs varying from coarse to fine are used to smooth the defect with a water rinse in between each disc. The defect is then dried slightly and a 40% phosphoric acid etch is applied and rinsed after the recommended time. Care is taken to not allow the etch to come into contact with the gingival tissues. The tooth is then dried slightly until it appears chalky white. The bonding primer is then applied with a disposable brush and light cured. Lastly the bonding sealant is applied with a disposable brush and light cured. The sanding discs can then be used to smooth the restoration site.

**Root canal therapy**

Involves removal of the pulp of the tooth, disinfection of the canal with sodium hypochlorite (household bleach), shaping with various endodontic files, filling of the canal with an inert material called gutta percha, and a composite restorative placement. For teeth that are non-vital (NV) or have complicated crown fractures (FX/CCF). This treatment will make the tooth non-vital and will weaken the integrity of the tooth structure, therefore a crown placement should be considered. It is necessary to repeat radiographs throughout the procedure to determine successful treatment.

**Vital pulp therapy**

This procedure can be performed on a very freshly fractured tooth (within 48 hours) on a relatively young animal with a wide pulp chamber to allow for the pulp to remain vital and apexogenesis to occur. Failure of vital pulp therapy is possible and future root canal therapy may need to be performed. The pulp is exposed and a thin layer of lining cement (MTA) is applied to irritate the pulp. A layer of glass ionomer is placed on top of the MTA and a final layer composite restoration is applied. The tooth should be radiographed in 6 months to check for maturation of the apex and continual dentin formation.

Being prepared to offer clients’ additional information on advanced treatments helps them make appropriate decisions for their pets’ care. Advanced dentistry treatments should only be recommend to those owners who are willing to invest the commitment to maintaining their pets’ oral conditions. Although many general practitioners perform general dentistry it is necessary to obtain additional training to acquire the skills needed for the above treatments. In many instances it is recommended to refer these advanced cases to the nearest veterinary dentist as the success rate is much higher in their skilled hands.

References available upon request.
The oral examination involves assessing the patient while in a conscious state as well as under general anesthesia. The oral examination should be a systematic approach and a record of findings is then added to the patients’ medical record. The technician can aid the veterinarian in identification of abnormalities and bring them to his/her attention and record the findings on the patients’ dentistry chart. It is common practice to develop a dentistry chart that encompasses more information than the “old” dental stickers. When charting the patients’ mouth, several abbreviations and marks can be utilized to document pathology. A current list of abbreviations can be found at the American College of Veterinary Dentistry website (www.AVDC.org). Your clinic may find it necessary to develop their own key of commonly used abbreviations and refer to them as necessary.

Triadan system
Identifies each tooth with a three digit number. The first number identifies the quadrant, the second and third numbers identify the tooth. The quadrants are identified as 1xx for right maxillary, 2xx for left maxillary, 3xx for left mandibular and 4xx for right mandibular. The teeth are numbered from the midline and move distally. The central incisor is 01, intermediate incisor is 02, and corner incisor is 03. An easy reference is to remember that canine teeth are always 04 and the first molars are always 09 (the rule of 4’s and 9’s). Deciduous teeth are identified as 5xx for right maxillary, 6xx for left maxillary, 7xx for left mandibular, and 8xx for right mandibular.

Directional terminology
Terms used to describe areas related to location, direction and position within the mouth
- Medial=towards the midline of the face
- Mesial=toward the central incisor
- Rostral=towards the front
- Caudal=towards the back
- Distal=away from the midline of the face
- Vestibular= towards the vestibule or lips (labial or buccal)
- Labial=toward the lips (used for incisors/canines)
- Buccal=toward the cheeks (used for premolars/molars)
- Facial=surfaces of rostral teeth visible from the front
- Lingual=toward the tongue (used in the mandible)
- Palatal=toward the palate (used in the maxilla)
- Coronal=toward the crown of the tooth
- Apical=towards the root apex
- Contact/Occlusal/Proximal=toward adjoining teeth in same jaw
- Interproximal=between two teeth
- Cusp=point of a tooth
- Cervical/Neck= area of the tooth where the crown and root meet

Conscious oral examination
Evaluate the face, zygomatic arch, TMJ, salivary glands and lymph nodes. Identify any asymmetry or abnormalities
Oral exam- check occlusion, mucous membranes, teeth number and periodontium. Identify malodor, tooth abnormalities, and possible periodontal inflammation

Occlusion
Normal occlusion in dogs and cats is a scissors bite where the teeth are evenly spaced and in alignment. The mouth is closed and evaluated for the following criteria. The cusp of the mandibular incisors rest on the cingulum on the palatal side of the maxillary incisors. The mandibular canines fit midway in the diastema between the maxillary third incisors and the maxillary canines. The premolars create a “pinking shear” interdigitation and the mandibular first molars sit palatally to the maxillary fourth premolar. Any deviation from this is a malocclusion and is categorized as follows:

Class I malocclusion
Neutroclusion; normal relationship between the maxilla and mandible but 1 or more individual teeth are out of alignment
- Rostral Crossbite- maxillary incisors are displaced palatally and/or the mandibular incisors are displaced labially
• Base Narrow Canine-lingually displaced mandibular canine- canine may come into contact with the palate
• Base Wide Canine- mandibular canine may “flare” may be attributed to lance teeth
• Lance Teeth- mesioversion or retroversion of the maxillary canine tooth, closes the diastema between the maxillary third incisors and the maxillary canine tooth

Class 2 malocclusion
Mandible is shorter than the maxilla “overbite”, mandibular brachy gnathism

Class 3 malocclusion
Maxilla is shorter than the mandible, or mandible that is longer than normal, brachycephalic breeds commonly have class 3 malocclusion

Oral Examination under general anesthesia
Oropharynx-should be examined after anesthetic induction and before intubation; soft palate, tonsils, fauces, palatoglossal arch

- Soft Tissue Exam- after completion of intubation; oral mucosa, gingiva, papillae, hard palate, floor of mouth and tongue
- Teeth- primary/permanent dentition, tooth count-to identify missing or supernumerary teeth, wear patterns, abnormal size/shape/position, pathology (fractures, resorption)

Periodontal exam
A thorough, systematic examination of the periodontium and tooth structure to evaluate for periodontal disease. The following criteria should be evaluated for each tooth:

- plaque index (PI 0-3) - measurement of plaque on the surface of each tooth, PI0= no plaque, PI1 =plaque that covers less than 1/3 of the surface, PI2= plaque that covers 1/3 to 2/3 of the surface, PI3= plaque that covers more than 2/3 of the surface
- calculus index (CI 0-3) – measurement of calculus on the tooth surface, CI0= no calculus, CI1= calculus that covers less than 1/3 of the surface, CI2= calculus that covers 1/3 to 2/3 of the surface, CI3= calculus that covers more than 2/3 of the surface
- gingivitis index (GI 0-3)- visual and periodontal probing evaluation to identify inflammation and bleeding. GI0=no inflammation, GI1=inflammation but no bleeding, GI2= moderated inflammation and bleeding upon probing, GI3= severe inflammation and spontaneous bleeding
- periodontal probing (PP)- evaluation of periodontal pocket formation with a periodontal probe “walked” around the tooth in atleast 6 places along the junctional epithelium. Normal gingival sulcus depth in dogs=0-3 mm, cats-0.5-1mm, consider the size of the patient you are evaluating, a great dane may have a normal gingival sulcus probing depth of 5 mm whereas a yorkie with a 5 mm probing depth would have periodontal disease.
- gingival recession (GR)-measured (in mm) from the cement-enamel junction to the free gingival margin.
- tooth mobility (M1-3)–assessment of how much a tooth moves from its axis using a suitable instrument such as a periodontal probe. M0=no mobility, M1=horizontal movement of 1 mm or less, M2=horizontal movement of more than 1mm, M3= vertical as well as horizontal movement of more than 1 mm
- furcation exposure (F1-3) -furcation exposure occurs when the bone between the roots of multi-rooted teeth is destroyed. Using a periodontal probe the furcation is examined. In a healthy mouth the furcation is not visible. FE1=furcation can be felt but the probe extends less than halfway under the crown, F2=probe can be passed more than halfway under the crown but not through and through, F3=probe passes completely under the crown from buccal to palatal/lingual
- Wear- 2 different types that occur from repeated friction on the teeth damaging the enamel and dentin.
  - Attrition (AT): occurs as a result of tooth to tooth contact often due to a malocclusion
  - Abrasion (AB): occurs as a result of external objects; tennis balls, cage biters etc
- Missing, malposition, malformed teeth–missing teeth are noted on the dental chart by circling the affected tooth/crown. A retained tooth root (RTR) may be present radiographically in the absence of a crown. Supernumerary (SN) “extra” teeth or roots that are common and can be illustrated on the chart where they may be located. Malpositioned teeth can be crowded (CWD) to close to one another or rotated (ROT) and can be drawn in on the dental chart.
- Tooth Trauma- injury to the tooth resulting in fracture, luxation, avulsion, pulpal hemorrhage
  - Tooth fracture (T/FX/fracture type)- classified by the degree of fracture
  - Enamel fracture (EF)-enamel is the only involved component
  - Uncomplicated Crown Fracture (UCCF)- crown fracture that does not involve the pulp
  - Complicated Crown Fracture (CCCF)- crown fracture that involves the pulp
  - Uncomplicated Crown Root Fracture (UCRF)- crown and root fracture that does not involve the pulp
  - Complicated Crown Root Fracture (CCRF)- crown and root fracture that involves the pulp
  - Root Fracture (RF)- fracture involving the root
Pulpal hemorrhage (NV)-when bleeding has occurred in the pulp canal causing increased pressure on the nerves and blood supply usually resulting in tooth death. Causes discoloration of the tooth from purple or tan/brown.

- Luxation (T/LUX)- partial displacement of the tooth from the alveolus, tooth may still remain vital if immediately replaced and splinted.
- Avulsion (T/A)- complete displacement of the tooth from the alveolus

- Retained deciduous teeth (RD)- retained deciduous teeth are drawn in on the dental chart. They can cause occlusion problems as well as increased incidence of periodontal disease.
- Oral Masses (OM)- can be drawn in on the dental chart
- Gingival Hyperplasia (GH)- a proliferation of gingival cells resulting in hyperplastic tissue. Can result in “pseudo-pocket” and periodontal disease. Common in some breeds, boxers, cocker spaniels
- Enamel hypoplasia (EH)- loss of enamel on the tooth surface, can be localized to one tooth or generalized
- Oro-Nasal Fistula (ONF)- open tract between the oral and nasal cavity
- Tooth resorption (TR)- loss of tooth mineral structure, explorer tip will “snag” on defect in the tooth. Classified into 5 stages:
  - TR1-mild dental hard tissue loss
  - TR2-moderate dental hard tissue loss
  - TR3-deep dental hard tissue loss-extends to the pulp chamber, most of the tooth remains intact
  - TR4-Extensive hard tissue loss; most of the tooth has lost its integrity
    - TR4a-crown and root equally affected
    - TR4b-crown more affected than root
    - TR4c-root more affected than crown
  - TR5-crown no longer visible; remodeling of hard tissue on radiograph

There are also abbreviations to use once the tooth has been treated.

- Extractions: (X)- simple closed extraction
  - (XS)- extraction with tooth sectioning
  - (XSS)- surgical extraction
- Crown Amputation (CRA)- amputation of the crown where type 2 root resorption is present
- Root Canal Therapy (RCT)
- Vital Pulp Therapy (VP)
- Root Planing- (RPO)- Root planning open
  - (RPC)- Root planning closed
- Perioceutic- (PCT)
- Biopsy- (B/I)- biopsy incisional
  - (B/E)- biopsy excisional

As this is but a few of the commonly seen oral pathological occurrences and treatments, I encourage each individual to continue to advance their learning by reading additional dentistry texts and visiting the AVDC website for further information.
Radiography in veterinary dentistry is one of the most valuable tools in our arsenal. It allows us to see what may be hiding in the oral cavity. It is important to understand that nearly half of the tooth structure lies in the periodontal bone below the gingival margin. Teeth that may look normal on the visible crown portion can have extensive pathology that can only be detected radiographically. In order to be performing higher standards of veterinary dentistry, one must be utilizing dental radiographs. Without dental radiographs, our patients could be left with painful unresolved issues. With the addition of dental radiography, many additional treatments can be provided for our patients. This means the addition of tens of thousands of dollars in potential revenue for the practice. The end results will be beneficial for the patient, client and the practice.

As skillful veterinary technicians we are the perfect people to perform this task for our veterinarians. Dental radiography techniques and positioning can be daunting at first, but with some practice and patience you will come to enjoy the treasures you will find in your images.

**Indications for radiographs**

Anything out of normal anatomy requires a radiograph to make sure you are not missing potential problems.

- Missing teeth
- Fracture teeth
- Oral Masses
- Evaluation of prior treatments
- Resorptive Lesions
- Periodontal Disease
- Draining tracts
- Evaluating Vitality of teeth
- Periodontal Pockets
- Nasal Discharge
- Pre & Post Extraction

**Equipment**

**Dental x-ray unit**

- either mobile or wall mount
- 3 parts
  - flexible arm - allows for compact storage and ease of use
  - tube head and cone - generates and directs the x-ray
  - control panel - sets the exposure

Exposure of the dental radiograph is controlled by 3 components, KVP, MA and exposure time. Due to the fact that there is not much variation in the tissues exposed during oral radiographs, the KVP and MA are constantly set on dental radiology units. That leaves the exposure time as the variable factor. This can be adjusted based on the teeth and area being exposed. Some dental x-ray units’ control panels have adjustments for specific teeth or area of the mouth being evaluated.

**Dental film**

- packaged in waterproof packet
- various sizes commonly used in veterinary dentistry: #0, #2, #4
- various speeds available: “D” speed most forgiving
- need for chair side developer or other developing system
- chair side illuminator for image viewing
- drawbacks such as longer time to develop (2-3 minutes), errors in positioning technique, storage of films and additional anesthesia time
- should be available as a back-up to digital sensor

**Digital dental imaging**

- Digital sensors (DR)
  - limited #2 size
  - image can be viewed within a few seconds on computer screen
- Phosphor Plates (CR)
  - multiple sizes: 0,2,4,6
- plates are fed through a scanner and image is available for viewing in 1-2 minutes
- initial higher investment cost, but less retakes=less anesthesia time
- requires computer: image storage and viewing is more convenient, software allows for manipulation of the digital image
Dental radiography positioning & techniques

All dental radiographs should be taken while the patient is under general anesthesia. If an attempt is made to take radiographs while under sedation, damage to digital sensors or positioning fingers will occur! The cost of a new sensor or a bitten finger will hopefully discourage anyone from trying this no matter how tempting it is!

Positioning is essential when learning to take diagnostic dental radiographs. Correct positioning requires tube angulation, tube position, sensor position, and patient position. There are many different techniques to positioning. I find the technique that makes the most sense to you and allows you to obtain high quality images, is the best one.

Patient positioning

I find it is easiest when the patient is in sternal recumbency with the palate as parallel to the table surface as possible for maxillary views. The head usually needs to be propped with a sturdy object under the chin to obtain the parallel position. For mandibular teeth, the patient is placed in dorsal recumbency with the mandibles as close to parallel to the table as possible. A towel roll under the neck can be used as a positional aid to accomplish this. This allows for one variable of positioning to be the same every time.

Tube angulation

The angles used to obtain diagnostic images are the parallel and the Bi-Secting Angle techniques.

- **Parallel Technique**: easiest to master, but limited to views of the mandibular premolars and molars. In this technique the plane of the tooth and the sensor are parallel, and the beam directed perpendicular to them both. This is the same technique used when taking a lateral radiograph of an extremity of a dog or cat. The result is an image that is minimally distorted.

- **Bi-Secting Angle Technique**: used to obtain images of all remaining teeth. This technique is required due to the fact that the oral anatomy does not allow the film to be placed on the same plane as the tooth. To compensate for the anatomical limitations, the x-ray beam is angulated perpendicular to an imaginary line halfway between the horizontal sensor and the vertical long axis of the tooth. When we look at the teeth we want to radiograph with the bi-secting angle, we need to imagine where the vertical roots of these teeth lie hidden within the periodontal bone. This is the vertical line angle. Next, we find the horizontal angle of the sensor as it lies in the patient’s mouth, this is the second line angle. We utilize these two line angles and imagine a line that bi-sects them in half. This is your bi-secting angle. We then take the edge of our cone, parallel to this imaginary bi-secting angle and take the radiograph! Most bi-secting angles are approximately 45° to 60° on the tube head scale, depending on the size of the dog and skull type.

Tube position

It is important that the tube head be centered on the whole tooth near the gingival margin not just the crown. If the tube is centered on the crown only the roots will be cut off the image, (“cone cut”). Some teeth are large enough that two images may need to be taken. One image of the root structure and one image of the crown. Cranial and caudal angulation can be utilized to separate any overlapping structures, such as the roots of the upper fourth premolar. This angulation can also be used to determine if a radiographic abnormality is truly associated with the tooth or an artifact.

Film position

The film must be placed where the image will be “projected”. With the angles utilized in the parallel technique it is easy to imagine where the image will be projected. The bisecting angle is the same principle of the sun casting a shadow of a person. The film must be positioned to catch the “shadow”. If you cut off the image, move your film over in the direction of the image that was cut. Usually the film needs to be placed as far as possible in to the mouth.

General techniques cheat sheet

- **Lower premolars and molars**: “Parallel technique” place sensor in the vestibule between the tongue and teeth. The beam is angled perpendicular to the sensor. Sometimes the bi-secting angle is need to obtain an image of the first and second mandibular premolars

- **Lower incisors and canines**: sensor placed on tips of incisors or canine teeth and the first premolars. Start by aiming the beam perpendicular to the sensor along the ventral midline, then adjust the beam to a 70 degree angle. To obtain the roots of the canine teeth, move the sensor caudally. You can also “oblique” the beam 30 degrees to either side to get additional views of the canines.

- **Upper incisors**: sensor is placed on the cusps of the incisors, parallel to the palate. The beam is aimed perpendicular to the dorsal midline then the tube head is tipped 20 degrees so the beam is at 70 degrees.
Upper canines- two view are often needed to obtain an image of the crown and root. The sensor is placed on the cusp of the canine, gauze may be required to keep the position of the sensor parallel to the palate. The tube head is then aimed perpendicular to the dorsal midline, then tipped to 60 degrees and the tube head moved 90 degrees laterally (panoramically). The sensor can then be re-positioned to obtain the root by moving it off the cusp and further caudal and medial to the crown. The same tube angulation is utilized for this view.

Upper/Lower premolars and molars- place the sensor mostly over the palate. Gauze may be required to keep the sensor parallel to the palate. The tube head is initially directed along the dorsal midline then angled at 45-50 degrees on the lateral/panoramic aspect of the maxilla/mandible.

Feline upper premolars and molars- Due to superimposition of the zygomatic arch over the maxillary premolars and molar in cats, an adjustment in technique is sometimes necessary. The above technique can be utilized with a tube head angle of approximately 35 degrees. This will cause some elongation of the roots. Another technique is the near parallel technique in which the sensor is place diagonally across the mouth from the inside of the opposite maxillary teeth to the ipsilateral mandibular teeth to be imaged. The patient is placed into lateral recumbency with teeth to be imaged on the top side and parallel to the table. Some paper towels can be positioned under the mandible to keep the head from tipping. The tube head is first angled lateral to the maxillary premolars then tipped 30 degrees over the top of the nose. If the beam is tipped to far the zygomatic arch will be in the way, tipping to little will cut off the targeted teeth.

All positional errors involve tube angulation, tube position or film position. Here are a few guidelines to correct these errors.

If the image is foreshortened or elongated the tube angle needs to be adjusted. You either need more angle or less angle.

• If the image is foreshortened or elongated, the tube angle needs to be adjusted. You either need more angle or less angle.

• All positional errors involve tube angulation, tube position or film position. Here are a few guidelines to correct these errors.

• If you end up with cone cut on the edge of your target, move the beam over toward that area.

• If you end up cutting off the target on the edge of your sensor, move your sensor toward that area.

Image orientation and interpretation

Although it is not the technicians’ responsibility to make a diagnosis based on the radiographs obtained, it is still important that the image is oriented properly for viewing by the veterinarian. The technician should also be familiar with the anatomical structures to accurately identify the area being radiographed. With many software systems the image is oriented with a specific template provided. The viewer should be able to orient the image without a provided template by following some basic rules.

• As with other general x-rays, dental x-rays are a mirror image. The right side of the patient is on your left and the left side is on your right.
  o Images should be oriented as if you were looking at that side of the patient
  o Maxillary views should have the roots pointing upward
  o Mandibular views should have the roots pointing down

• Maxillary teeth can be identified by a thin white radiodense line the “palantine process” above the roots, as well as the sinuses.

• Mandibular teeth will have the dark mandibular canal visible below the roots as well as the mental foramina

• When looking at an image of the right arcade, the molars should be on the left side of the film with the premolars on the right and vice versa for the left arcade

• Imagine the patient standing in front of you, the direction the nose is pointing is the side you are viewing

• Rotating the image is okay, flipping is not

Once the image is correctly oriented, assess the quality of the image. Is the tooth in question visible in the image? The image should be clear without blurring or fogging. All roots should be visible with a minimum of 2 mm visible beyond the apex of the root. Make sure you read the entire film, not just the most obvious defects.

Radiographic anatomy

Learning to identify the normal anatomy of a dental radiograph will help you decide what is abnormal. Normal anatomic features to identify are enamel, dentin, pulp chamber, cement-enamel junction, lamina dura (white line), cortical bone, periodontal ligament, root apex, root canal, intradental trabecular bone, and alveolar crestal bone.

• A normal tooth has a complete covering of enamel on the crown.

• The root is covered with cementum but is not visible radiographically.

• The middle “black line” of the tooth is the pulp chamber and root canal.

• Sandwiched in between the enamel and the pulp chamber is what the majority of the tooth is made of, dentin.

• At the end of the root is the apex and where the roots diverge in multi-rooted teeth is the furcation.
• The periodontal ligament (PDL) is a soft tissue structure that surrounds the root and is identified by the radiolucent area (black line) between the root and surrounding bone. The PDL should be uniform width and visible around the entire root.
• The alveolar bone is the specialized bone that forms the alveolus or socket of the tooth and can be identified by the lamina dura or white line.
• Alveolar crestal bone should be present between the teeth and extend to the cement-enamel junction
• Intradental trabecular bone should completely fill the space in between teeth

Common pathology that a technician can recognize and bring to the veterinarians’ attention include:

**Periodontal Disease**: the presence of horizontal or vertical bone recession (bone loss) is an indication of periodontal disease. Normal bone should reach 1-2 mm apical to the cemento-enamel junction and fill the furcation of multirooted teeth. Horizontal bone loss is recession of bone apically in a horizontal line. Vertical bone loss is bone recession that follows one or more roots apically. Widening of the periodontal ligament space is also an indication of periodontal disease.

**Endodontic Disease**: Endodontic disease may not be detected until a radiograph is taken. This can be identified as a periapical lucency or “halo” at the root apex. This “halo” can be mimicked by the foramen so know your anatomy. It may also present as a pulp canal that is wider than the adjacent or contralateral teeth as seen in non-vital teeth. A subtle change in the lamina dura or periodontal ligament space beginning apically and moving coronally can also be associated with endodontic disease.

**Root Abnormalities**: varying abnormalities such as extra roots, roots that curve in an unusual direction, fractured roots and retained root tips can be identified radiographically. This information is valuable when planning for extractions or additional therapy.

**Tooth Resorption**: Tooth resorption can take place in dogs and cats. There are different stages of tooth resorption that can be identified radiographically. Most commonly teeth with resorption will have a crown with a large piece missing or a moth eaten appearance. The roots can have a normal appearance or may be in the process of becoming ankylosed with a large amount of root destruction. Radiographs are important in tooth resorption cases to determine whether complete surgical extraction or crown amputation can be performed. Cats with one or more teeth affected with tooth resorption should have full mouth radiographs to identify any additional teeth that may be affected.

There many other areas of radiographic pathology that can be recognized. Once you have become familiar with what is normal anatomy you will quickly be able to discover what is abnormal. This will expedite the process of diagnosis and treatment of several diseases in the oral cavity.

**Quick guide for tube head angulations**

- Maxillary/Mandibular Incisors - 70⁰ degrees
- Maxillary/Mandibular Canines - 70⁰ degrees (apex) “Cheater” Crown View - 45⁰ with sensor tilted behind crown
- Maxillary Premolars & Molars (canine) - 45-55⁰ degrees
- Maxillary Premolar & Molars (feline) - 35⁰ degrees
- Mandibular 1st & 2nd Premolar (canine) - 50⁰ degrees
- Mandibular 3rd Premolar (feline) - 50⁰ degrees (tongue on top of sensor)
- Mandibular 3rd, 4th Premolars & Molars - Parallel technique
- All maxillary images are taken with the patient in sternal recumbency and mandibular images with the patient in dorsal recumbency.

*All tube head angulations are using the tube angulation scale on the x-ray generator head. Remember to set the degrees on the scale side nearest the patient.
As veterinary technicians, we advocate for the highest quality of medicine for all of our patients. This ideal standard has evolved over the years to include the prevention of all painful stimuli while they are in our care. Pain management is an imperative aspect of veterinary dentistry, and nerve blocks for surgical procedures are a necessary resource that technicians can utilize as part of a multi-modal approach.

Defining pain is difficult but generally one can safely state that pain is an unpleasant sensation occurring in varying degrees of severity as a consequence of noxious stimuli. A general understanding of how pain is received and transmitted is vital in order to control it. Nociception is the neural processes of encoding and processing noxious stimuli. Noxious stimuli are detected by peripheral nerve endings called nociceptors, this is also known as transduction. Once stimulated, a nociceptor transmits a signal along the spinal cord (transmission) to the brain (modulation), which results in a perception of pain by the patient.

A multi-modal approach to pain control is widely accepted and utilizes a combination of multiple analgesics, which aim to prevent the transmission of pain signaling at various points along the pain pathway. It is far easier to prevent the perception of pain than to treat pain after the fact. The goal of a multi-modal approach is to prevent the sensation of pain by utilizing several different analgesics to prevent the patient from perceiving pain signals. Examples of appropriate analgesics are as follows:

- Local anesthetics, alpha-2 agonists, and opioids are used at the point of transmission
- NSAIDS (non-steroidal anti-inflammatories), opioids, corticosteroids, and local and regional anesthetics work at the point of transduction.
- Inhalant anesthetics work at the point of perception/modulation.
- A post-operative protocol for continued pain control should include a synergistic approach, which include opioids and NSAIDS.

Dental nerve blocks

Regional and local blocks can be utilized several minutes before an anticipated painful procedure. These should be considered before extractions, gingivectomy, mass removals, endodontics, mandibulectomy, maxillectomy, or any advanced periodontics. A discussion with the veterinarian can aid in determining what the anticipated pain level might be and adjust the pain management plan as needed.

- **Lidocaine**: short onset of action, but only lasts 1-2 hours. Dosage not to exceed 5 mg/kg Lidocaine w/epinephrine: constricts blood vessel but should be avoided in patients with heart disease.
- **Bupivacaine**: delayed onset of action, but lasts 6-10 hours. Dosage not to exceed 2 mg/kg

Lidocaine and Bupivacaine can be combined together in the same syringe to achieve the quick onset with lidocaine and longer duration with bupivacaine or bupivacaine can be used alone. It has been shown that it is no longer necessary to combine the two medications and may be detrimental due to the fact that the lidocaine wears off quickly it can then open a site on the nerve receptor. If necessary saline can be added to the syringe at a 1:1 ratio to increase volume without lessening the effectiveness of the drug. Care should be taken to always calculate the patient’s total toxic dose and to stay below that amount. These local anesthetic agents should not be administered intravenously as profound side effects such as bradycardia, respiratory distress and seizures can occur.

**What you will need**
- 25 or 27 gauge X 5/8” or 1 1/2” needle
- 1 cc to 3 cc syringe
- Bupivacaine
- Skull to visualized anatomical landmarks

**Technique**
- Drug is drawn into syringe
- Change to new needle
- Insertion of needle through the mucosa at selected site
- Syringe is aspirated to check for blood, if no blood, then inject slowly.
- Apply digital pressure for 30-60 seconds to diffuse medication

**Infraorbital (Rostral Maxillary) Block**

This nerve block provides analgesia to the ipsilateral bone, soft tissue, and teeth from the third premolar to the incisors. Care should be used in brachycephalic breeds and cats to avoid advancement of the needle into the ocular globe.
Technique
Palpate the foramen dorsal to the apex of the distal root of the maxillary third premolar. The neurovascular bundle can also be palpated in this area. The needle is held parallel to the maxillary bone and inserted in a rostral to caudal direction into the foramen. If resistance is met the needle should be redirected. Use above technique for injection.

Maxillary nerve block (caudal maxillary block)
This nerve block provides analgesia to the ipsilateral bone, soft tissue, and teeth from the first molar to the incisors. Please use caution, as this block can be potentially traumatic to the ocular area.

Technique
The foramen is located dorsal to the maxillary first molar. This needle is inserted 3-5 mm vertically, with the bevel pointed rostral (toward the nose) into the soft mucosa, just beyond the bony ledge located near the second molar. Use above technique for injection.

Middle mental nerve block
This block provides analgesia to the ipsilateral bone, soft tissue and teeth from the mandibular 2nd premolar to the incisors.

Technique
The foramen can be palpated ventrally from the distal root of the mandibular 2nd premolar and just caudal to the frenulum. Retract the frenulum ventrally while advancing the needle caudally with a slight ventral angle. In cats the foramen is rarely palpable and is located caudal to the apex of the mandibular canine. Use above technique for injection.

Mandibular (inferior alveolar) nerve block
This nerve block provides analgesia to the ipsilateral bone, soft tissue and teeth of the injected mandible. This block can either be performed extra-orally or intra-oral

Technique
• The foramen is located on the lingual aspect of the caudal mandible approximately 2/3 the distance between the last molar and the angular process. For the extra-oral technique the mandibular notch is palpated (just rostral to the angular process), the needle is inserted vertically on the lingual aspect of the mandible. An imaginary line from the lateral canthus of the eye to the ventral aspect of the mandible can also be used to verify placement. Use above technique for injection.
• Local and “Splash” Blocks: are nerve blocks that can be utilized for small areas, such as the gingiva for an oral mass or into the alveolus after an extraction has been performed. Tissues with inflammation and infection have altered pH levels which render analgesic agents ineffective.

Regional and local blocks are tasks that many technicians can perform with training and practice. They are relatively inexpensive and provide one puzzle piece in the multi-modal pain management approach. This will allow us to provide supreme patient care for our beloved friends.
Queen LaTeetha’s Magic Wand:  
A Guide to Hand Instrumentation  
Candice Hoerner, CVT, VTS (Dentistry)  
Big Sky Veterinary Dentistry Education  
Columbia Falls, MT

Veterinary Dentistry requires specific instruments to accurately diagnose and successfully treat periodontal disease. The main types of hand and power instruments utilized in the complete periodontal cleaning are as follows:

**Explorers**

The most commonly found explorer is a shepherd’s hook, usually made of stainless steel spring wire for greater tactile sensitivity. This instrument is used for detection of supragingival and subgingival calculus, necrotic cementum, exposed pulp chamber, tooth resorptive lesions, furcation involvement and cavities.

**Probes**

An essential tool for determining presence of periodontal disease by measuring pocket depth and attachment loss. Usually a double ended instrument combined with an explorer at one end. There are various bands or notches, usually at 1 mm intervals, which aid in accurate measurement of pocket depths.

**Scalers**

This instrument is used for supragingival (above the gum line) scaling ONLY! Can be gently used in the developmental groove of the upper fourth premolar as well as removal of tartar and plaque on the crown. The triangular shape with 3 sharp sides and a sharp tip can be very damaging if used subgingivally. Two common types are the sickle (curved) and jacquette (straight).

**Curettes**

Due to the delicate design of this instrument, with one to two sharp edges (Gracey vs. universal), a rounded back and toe, and half-moon cross section, this is the instrument of choice for subgingival scaling, curettage and root planing. The rounded back is inserted subgingivally in a "closed" position, then rotated up to the "open" position to engage the cutting edge, and then a pull stroke down towards the crown.

**Power scalers**

There are various types of power scalers available today. It is important to note that each type works differently. As the operator it is your responsibility to know the units specifications and to be able to adjust power and water settings as needed. The most common type of ultrasonic power scalers used in veterinary dentistry are piezoelectric and magnetostrictive. Piezoelectric ultrasonic scalers use crystals in the handpiece to create a vibration. The frequency of these units range from 25,000 to 45,000 cycles per second with approximately 0.2mm back and forth motion at the tip. The working tip is approximately 3 mm. Magnetostrictive scalers are either the metal strip/stack or a ferroceramic rod. The frequency of the vibration in these units range from 18,000 to 42,000 cycles per second. All sides of the working tip are active, approximately 10-12mm. There are multiple types of tips available such as subgingival-beavertail, periodontal or a universal tip. Once the tip has been worn it is necessary to replace or it will be ineffective. The operator should be aware of what type they are using and adjust power and water settings as needed.

Use of proper technique is vital when using power scalers. A light grasp on the handpiece in which someone could walk up behind you and pull the handpiece away without resistance is appropriate. The remaining cord can also be wrapped around another finger to relieve weight and fatigue on the operator.

A gentle pressure is applied with the lateral surface of the working tip, parallel to the tooth surface, and moved across the tooth and briefly subgingivally, with a wide sweeping motion, spending 10-15 seconds per tooth. Too much pressure or time can cause thermal damage to the tooth, excessive etching of the enamel, and the handpiece will be ineffective. Too light of pressure will not effectively remove the calculus. The working tip should NEVER be place perpendicular to the tooth surface (or used in the developmental groove of the upper 4th premolar!) as this is the most active part of the tip and can generate the most heat and vibration in this position causing severe damage to the tooth surface. Power scalers should not be used on restorations as they can cause them to be dislodged. Restorations should be gently scaled with hand instruments only.

**Low-speed/Polishing handpiece**

Polishing of the tooth surface is necessary to smooth the micro-etching of the enamel and remove traces of plaque left after scaling. A smooth tooth surface will not allow retention of plaque particles like a rough surface would. The low-speed handpiece on air driven units are generally used for polishing teeth with a soft prophy angle or prophy cups. There are two common types of prophy angles; a standard circulating one that rotates 360 degrees and an oscillating angle that moves back and forth 90 degrees. Oscillating prophy
angles are preferred as they do not generate as much heat as circulating ones. Prophy paste is used to return the tooth to a smooth surface and to reduce friction while polishing. Selecting the appropriate prophy paste is important as it is not recommended to use paste containing fluoride on restorations or one that is too coarse that could potentially damage the enamel on the tooth. Many prophy pastes will also cause artifact on radiographs if not rinsed away completely. Polishing should be performed at approximately 5,000 rpm with a fine grit or flour pumice prophy paste. Enough pressure should be applied to the handpiece to obtain a slight flare to the prophy cup and to quickly cover the tooth surface and gently pass underneath the gingival margin. Subgingival polishing is just as important as coronal polishing so as not to leave rough areas subgingivally that can lead to further attachment loss. This can be done in approximately 3-5 seconds per tooth. Thermal damage to the tooth may occur if too much pressure, too high of rpm and not enough paste are used.

**Sharpening**

The greatest reason for decreased instrument life is incorrect sharpening techniques. There are two methods: stationary stone-moving instrument or moving stone-stationary instrument. Both methods are correct, however the one that is most comfortable to the operator is the most effective. Before sharpening it is always best to determine if the instrument truly needs to be sharpened. An acrylic rod can be used to engage the cutting edge of the instrument and if the instrument "grabs" then the instrument is still sharp. If the instrument is dull it will not engage the acrylic rod. Additionally, a visual inspection of the cutting edge by holding it in the light can determine if the instrument needs sharpening. A dull edge will reflect the light, a sharp one will not. There are different types of sharpening stones. The most common is the Arkansas stone for regular sharpening, an India stone for recontouring dull instruments, and a conical stone for removing flashings on the face of the instruments. All of these require a small amount of sharpening oil.

The stationary stone-moving instrument technique requires the stone be placed flat on a work surface. A small amount of oil is added to the stone. The instrument is held firmly against the stone surface with the blade facing the operator. Position the curette face to form a 90 degree angle to the stone and then "tip" the blade away to a 100-110 degree angle. Perform 3-4 pull strokes. Then adjust the angle to maintain a 45 degree angle for rounding the toe of a curette.

The stationary instrument-moving stone technique can be performed on the edge of a counter with the instrument blade facing the operator. Position the stone on the lateral surface and form a 90 degree angle. Then adjust the stone to a 100-110 degree (1 o’clock) angle. Perform 3-4 down strokes then decrease the angle to 45 degrees when rounding the toe of the curette.

The conical stone can then be swiped across the face of the instrument to remove any flashings.

**Ergonomics**

A modified pen-grasp is used for proper instrument grip. The Triangle of Forces utilizes the thumb, index and middle fingers and forms a triangle to grip (not pinch) the instrument handle. The ring and pinky fingers act as a fulcrum. A rocking wrist motion along with an oblique /pull stroke generates the effective working stroke. Improper instrument grasp can lead to ineffectual removal of deposits as well as repetitive motion injuries common in dental professionals.

In addition to proper instrument grip it is vital to have a few essential items to support the operators’ longevity. Proper lighting, magnification and seating can aid the operator in maintaining an upright and relaxed position. Use of magnification loupes as well as additional head lamps are very useful and worth the investment. A saddle seat style stool can allow the operator to sit in a more upright position while allowing the legs to drop into a more ergonomic position that allows better blood flow to the lower leg. Additionally certain stretches and exercises for the hands and arms can help maintain optimal hand strength and reduce fatigue.

In order to properly perform periodontal cleanings in veterinary patients, the dental technician must have a good understanding of how the instruments are utilized. All periodontal cleanings require various instruments and techniques and in a qualified technician’s hands can provide an exceptional service to our patients.
A complete periodontal cleaning is required to remove the "disease" elements of periodontitis. This includes removal of bacterial laden supra(above) gingival and sub(below) gingiva calculus and plaque. The intent of professional dental cleaning is to prevent periodontitis, but most patients already have significant disease. The terms “prophy”, “prophylaxis”, “dental” are misused as very rarely are we actually preventing periodontitis. Dental procedures must be performed by a licensed veterinarian, a credentialed technician, or a trained veterinary assistant under the supervision of a veterinarian in accordance with state or provincial practice acts. Practice acts vary from jurisdiction to jurisdiction, and the veterinarian must be familiar with those laws. Surgical extractions are to be performed only by trained, licensed veterinarians.

In 2013 AAHA published the Dental Care Guidelines for Dogs and Cats, which are guidelines for the practice of companion animal veterinary dentistry and include the following steps.

**Admission**

Upon admission of the patient for its procedure, client contact information and consent for the procedure is obtained. This allows the client to be reached if any oral pathology is found while the patient is under anesthesia. No additional procedures should be performed without informed consent of the owner. Any patient history should be identified as well as a brief discussion of future homecare may be warranted if time permits.

**Anesthesia**

The appropriate anesthesia protocol and fluid support is decided upon based on the pre-anesthetic examination, age of the patient, laboratory results, general health, etc. as well as anticipated duration and pain level beyond a "routine" cleaning. A protocol for multimodal pain control should be considered ahead of time and could include use of an opioid for pre-anesthetic sedation which can decrease the amount of inhalant anesthetic agent used. A CRI can be considered for some patients as this also can decrease the amount of inhalant required. This combined with intra-operative pain medication of local and regional blocks and post-operative drugs are ideal for a multi-modal approach from the beginning.

**Patient positioning, monitoring & care**

After the patient has been given an anesthesia induction agent, an endotracheal tube with a properly inflated cuff is placed to prevent fluid or debris from entering the trachea and lungs, and the patient is maintained under general anesthesia. Gauze pads can then be placed in the oropharynx for additional protection if suction is not available. These gauze pads need to be changed frequently throughout the procedure and always removed before the patient is recovered from anesthesia.

For most procedures it is adequate to position the animal in lateral recumbency. Virtually all of the cleaning, polishing, radiography etc. can be accomplished in this position. Dorsal recumbency is another accepted position as the animal is placed into position only once and is not moved for the entire procedure. As with any new positioning technique it will take some time to practice and everyone has their preference.

The patient is then connected to appropriate monitors including blood pressure, pulse oximeter, electrocardiogram etc. Patient parameters are checked every 10-15 minutes and should be noted on an anesthesia worksheet of some type. Patient body temperature is especially important in the dentistry patient as they rapidly become wet from the water spray, and this can drop very quickly if initial steps are not taken to keep the patient warm. The patient is placed on a hot water circulating blanket or a type of forced hot air device. Towels can be placed under the patients head and around the body but must be changed to keep the patient as dry as possible.

**Oral exam**

Before you begin your periodontal cleaning a brief oral exam is performed including the tongue, soft and hard palate, pharynx, tonsils, buccal mucosa, lymph nodes and the extra-oral facial features as well as determining the occlusion of the animal. Any deviation of the normal occlusion pattern, in which the lower incisors occlude palatally to the upper incisors and the upper and lower premolars and molars occlude in an interdigitating, shearing relationship with the maxillary teeth buccal to the mandibular teeth are noted.

The amount of calculus, plaque, gingivitis, pocket formation and furcation exposure are graded prior to the beginning of cleaning and can be repeated several times during the procedure. This is noted on the dental chart which should include a diagram of all the teeth in buccal, occlusal, and lingual views so that different pathology can be properly charted at each site. Dental charting should be uniform within the clinic and have an abbreviation key available for reference. The AVDC website has all the dental abbreviation recommendations and this can be adapted to your clinics’ needs.
Gross calculus removal
To begin your cleaning the heavy calculus is removed with tartar removal forceps or regular extraction forceps. The tips of the forceps are carefully placed around the tooth just coronal to the gum line. Care must be taken to avoid the delicate gingival tissue, as it can become trapped in the forceps and damaged severely. The handles are then squeezed until the thicker layers of tartar “crack” off of the tooth. It is not necessary to remove every piece of tartar with the forceps as this will be done with the use of power and hand instruments.

Radiographs
Dental radiographs are a very useful tool in veterinary dentistry today. Full mouth radiographs are a preferred method due to the discovery of pathology that may have gone undetected. Radiographs are taken of any teeth with pockets greater than 2-3 mm, furcation exposure, mobility, fractures, or oral masses. It is also important to take radiographs of any “missing” teeth as it is common to find retained tooth roots or embedded teeth if there is no previous record of extractions at that site. Dental radiographs can be taken with dental film or a digital sensor. Digital radiography has sped up the process time of traditional film and provides excellent detail in a short amount of time as well as being less expensive in the long run. After the radiographs have been taken a treatment plan can be discussed with the client.

Power scaling
The remainder of the calculus and plaque is removed with power equipment and hand instruments. The three types of power scalers are piezoelectric, ultrasonic and air driven. The rotational vibration and water spray cavitation at the head of the scaler is what removes the additional calculus and adhered plaque from the tooth surfaces. The type of power scaler does affect how to use the direct sides of the scaler tip and are in the operator manual of individual units. This should be known prior to use of the equipment. A general systematic approach is used to clean each tooth in each quadrant. You should clean in the same pattern every time so as to decrease your chances of missing any surface. The scaler tip is then placed near the gingival margin and is moved across the tooth in a broad sweeping motion as you move coronally. Try not to inadvertently "lift up" and replace the tip any more than necessary as this can cause pitting of the enamel. Also do not spend more time than is necessary to clean the tooth as thermal damage can occur with prolonged contact. Specific power scaler tips are used for subgingival and supragingival scaling. A universal tip can be used above and below the gum line and is the best all-around tip for general use.

Hand instrumentation
Power scaling is always followed by hand instrumentation as a way to "check" for deposits of calculus and plaque left behind. Hand scalers are always used above the gum line on the crown of the tooth. Scalers generally have 2 sharp cutting edges and a pointed tip. These sharp edges, if used under the gum line, can cause severe damage to the gingival tissues. The most obvious areas that require hand scaling are the developmental groove of the upper 4th premolar and other fissures. The hand scaler tip can be used at a direct angle to the tooth whereas the power scaler is never allowed to be used at a right angle to the tooth! The curette comes in many different styles and angles but because it has a rounded end and backside it can be safely used under the gum line. The curette is a more fragile instrument and should not be used as a scaler! Regardless of whether you are using a scaler or a curette it is important to know which part of the instrument is doing the work. The “toe” (curette) or “tip” (scaler) is the very point of the instrument, then the cutting edge and the face. The toe and rounded back of the curette can be gently inserted under the gum line. It is then tilted up at approximately 15-20 degrees until the cutting edge is engaged and then in a quick long pull stroke of the instrument as it is moved coronally. This is repeated until the tooth is smooth. To remove and debris in the subgingival soft tissue the curette is reversed and the cutting edge is moved along the gingival tissue. This is basic root planing and subgingival curettage.

Detection of missed calculus and plaque
After all calculus and plaque has been removed a disclosing solution can be applied to the teeth to identify anything that has been missed. This is generally a red colored solution and many find it to be too messy. The air-water syringe can be used to air dry the tooth and look for any debris that may have a chalky white appearance. The air can be directed under the gum line to look for any additional debris that may be hiding.

Repeat of oral exam & plan for further treatments
The oral exam can then be repeated to include further grading of gingivitis, measuring of periodontal pockets, checking for tooth mobility, missing teeth, furcation exposure, fractured or traumatized teeth or any other abnormalities. A plan for further diagnostics and treatment can then be established.

Polishing & final rinse
After further procedures are completed the teeth can be polished using the slow speed handpiece and a generous amount fine grit polishing paste. The rpm of the handpiece should not exceed 5,000 as this can thermally affect the tooth. Time spent on each tooth
should be kept to a minimum for this reason as well. The oral cavity is then rinsed and dried and any additional debris, fluid and blood is removed. The oropharynx gauze pads must be removed, and the patients faced is cleaned and dried. The patient is then recovered from anesthesia and temperature is continually monitored as well as the need for additional pain medication. Once the animal is extubated and safe in his cage, cleanup and instrument care can take place.

**Discharge & follow up**
Discharge of the animal usually includes the patients’ post-op instructions, radiographs and photographs and should be printed and explained to the client. Clients are advised of any dietary needs, post-op medications and recheck exams. A two week follow up appointment is ideal to evaluate healing of the oral cavity after treatment and allows the client to focus on homecare recommendations. This way they are not bombarded with too much information on the same day as the procedure. It is also recommended to have a six month complimentary oral exam to re-evaluate the patient and address any concerns. Clients should also be aware of the advantages of regular home care. These recommendations should include daily tooth brushing, oral rinses, dental diets and approved dental chews. Home care should be tailored to the individual patients’ lifestyle and temperament as well as how willing the client is to follow through. Recommend brushing only the teeth they want to save, and see what kind of response you get!!!
Kidney failure is one of the most common diseases that affects feline patients. With the advances of treatment options and the understanding of kidney disease, feline patients are living longer and more productive lives after being diagnosed with kidney disease. Due to the allowed length of the proceedings notes, treatment of kidney disease for both the dog and cat will be discussed in the proceeding notes titled “The Dog and the Kidney”.

**Physiology**

The kidneys filter approximately 20% of the body’s blood. The kidneys are responsible for a myriad of tasks including: regulation of water and electrolyte balances, excretion of metabolic waste products and foreign chemicals, regulation of arterial pressure, regulation of acid-base balance, regulation of calcium excretion, metabolism of certain minerals, production of the active form of vitamin D, glucose synthesis and erythropoietin production. In a normal pet, the kidneys account for almost all the erythropoietin that is secreted into circulation. Erythropoietin stimulates the production of red blood cells.

Each bean-shaped kidney contain millions of functional units called nephrons, which filter blood through the glomerular filtration (GF) process and produce urine. Each nephron is a long tubule which consists of five main parts: glomerular capsule, proximal convoluted tubule, loop of Henle, distal convoluted tubule and collecting duct. As blood enters into the glomerular capsule, high pressures force fluid and small molecules out while larger particles (blood cells, plasma and protein molecules) are retained in the blood and continue through.

The proximal convoluted tubule reabsorbs 65% of the filtered sodium, chloride, potassium and bicarbonate. It also filters almost all of the glucose and amino acids. The proximal convoluted tubule concentrates nitrogenous waste which helps to create urea. Certain drugs and toxins are secreted into the filtrate and carried into the bladder via urine. This is why certain drugs, like penicillins, work well for treating bladder infections.

The loop of Henle helps to regulate the concentration and volume of the urine by removing excessive amounts of water. It reabsorbs considerable amount of calcium, bicarbonate and magnesium into the ascending loop of Henle. It maintains a balance with the body’s extracellular fluid (ECF) so if an animal is dehydrated less water will be absorbed and if it is overhydrated more water will be absorbed.

The distal convoluted tubule is responsible for making the some of the final adjustments to the fluid by reabsorbing even more sodium, secreting potassium and regulating the acid/base balance. Lastly, the collecting duct is responsible for collecting the fluid and making the very final adjustments so that it maintains a complete balance with the ECF. This is where the concentrated urine is collected.

**Renal failure**

Renal failure is divided into two forms: acute or chronic forms. Either form may be due to a large number of medical problems. Both are hallmarked by an elevated serum creatinine and blood urea nitrogen (BUN), known as azotemia. When the kidneys malfunction, they cannot filter properly which leads to abnormal fluid levels in the body, deranged acid/base levels, hematuria, anemia, isosthenuria (urine specific gravity of 1.008 - 1.012), and abnormal levels of potassium, calcium and phosphate.

Uremia is a term used to describe most of the clinical signs and biochemical findings that occur with renal failure. The most common uremic complication is the appearance of gastrointestinal (GI) symptoms (nausea, anorexia, vomiting, diarrhea). Gastrointestinal signs can escalate to include hemorrhage of the GI tract, ulcers in the mouth and necrosis of the tongue due to the increase of gastric acid juice because up to 40% of gastrin is metabolized within the kidneys. Other signs of uremia include polyuria, polydipsia (due to impaired urine concentrating ability), hypertension and anemia.

The goal with any kidney disease is to decrease the level of azotemia, therefore decreasing the uremic signs. During treatment, kidney values should be monitored and treatment should continue so long as improvement is seen or a plateau is reached. An average hospital stay is between 3 to 6 days.

**Acute kidney injury**

Acute kidney injury (AKI) results from a dramatic decrease in GF rate because of either prerenal, intrinsic renal or postrenal causes. Many causes of AKI are reversible pending diagnosis and treatment is made early.

Prerenal azotemia is not caused by primary kidney disease, but rather by a decrease in cardiac output resulting in inadequate blood supply to the kidneys. When mean arterial pressure (MAP) starts to fall below 60 mmHg, perfusion to the kidneys becomes compromised. Prerenal kidney azotemia is reversible because it is not associated with morphologic damage to the kidneys. It is marked by mild azotemia with BUN levels less than 80 mg/dl and serum creatinine levels less than 4 mg/dl. Any patient with renal
failure, including prerenal azotemia, should not be administered nonsteroidal anti-inflammatory drugs (NSAIDS) or angiotensin-converting enzymes (ACE) inhibitors because they can cause a worsening of the azotemia by further decompensating GF.

**Intrinsic damage** to the kidneys occurs from damage to the renal parenchyma, specifically to the vasculature, glomeruli, tubular epithelium and interstitium of the kidney. In cats, the most common causes occur from toxins, infectious diseases and ischemic causes (such as heat stroke, pancreatitis and disseminated intravascular coagulation). The kidney is particular vulnerable to toxins because of its high rate of blood filtration.

Some of the most common nephrotoxic drugs/chemicals include ethylene glycol, gentamicin (and other antimicrobials), cholecalciferol (vitamin D) and lilies. While not as common as in the dog, ethylene glycol toxicity it is widely talked about because of its common availability in such products as antifreeze, cleaners, cosmetics and flavoring extracts. In cats, the lethal dose of 95% ethylene glycol is 1.4 to 4 ml/kg. Ethylene glycol causes a toxic effect by forming oxalate which binds to plasma calcium and forms calcium oxalate crystals in the renal tubules. The calcium oxalate crystals clog the tubules leading to AKI.

Lilies are a common toxicity in cats and while the principle toxic factor is still unknown, it is known that all parts of the plant are toxic including pollen. After initial ingestion of the plant, cats may exhibit gastrointestinal signs such as nausea and vomiting. Signs develop within 12 hours, but the plant may still have effects on the body for 2 to 5 days after ingestion. Though the exact amount needed to produce a toxic effect is unknown, it is known that even a single bite can cause symptoms, which is why any cat exposed to a lily plant should be treated as if it were going to suffer nephrotoxic effects.

One of the most common diseases that causes AKI in cats is pyelonephritis. Pyelonephritis (inflammation of the kidneys) most commonly occurs secondary from a lower urinary tract infection. Clinical signs may include fever, vomiting, anorexia and abdominal pain (generally when palpating the kidneys). Most commonly, Escherichia coli (E. coli) is the organism that is isolated. Antibiotics that are specific to the organism isolated should be administered as a treatment along with treating the AKI symptoms.

**Postrenal** causes occur from obstruction or rupture of the urinary tract system. Urethral obstruction is most commonly seen in young to middle-aged male cats. Cats that have had feline lower urinary tract disease (FLUTD) have an increased risk to becoming obstructed. In cats that have been obstructed for a long period of time, AKI can occur. When a cat becomes obstructed, pressure within the urethra and urinary bladder will be transmitted up the ureters to the nephrons. Eventually the pressure starts to alter the GF pressure until the rate is zero. Early detection and treatment is imperative in order to correct the azotemia. Approximately 25% of cats that obstruct have a complete resolve of their azotemia in 2 to 5 days. Another 40% retain mild azotemia and are successfully managed with medical treatment.

Obstruction may also occur because of bilateral or unilateral obstruction of a kidney from nephroliths or ureteroliths (most commonly calcium oxalate). Cats with unilateral ureteral obstruction may be asymptomatic and symptoms may occur only when the kidney becomes enlarged due to hydrenephrosis. Bilateral ureteral obstruction will result in more symptoms including azotemia, vomiting and anorexia. Hematuria may occur.

**Chronic kidney disease (CKD)**

Cats, unlike dogs, generally can live many years with CKD. Roughly 30% of felines over 15 years will experience CKD. The causes of CKD are numerous and can be congenital, familial or acquired. Congenital causes are often suspected based on the age of the cat, breed and family history. Polycystic kidney disease (PKD) is more common in Persians and is inherited as a autosomal dominant trait. PKD is characterized by the presence of multiple fluid-filled cysts (hence "polycystic") which can result in the enlargement of the kidneys. The cysts generally develop at an early age (as early as 7 weeks), but signs of renal failure may not occur until the cat is middle aged (between 7-8 years). It’s important to note that not all cats with PKD will develop kidney failure. For breeders of at-risk breeds, genetic testing is available for cats older than 8 weeks old (Veterinary Genetics Laboratory, University of California-Davis: www.vgl.ucdavis.edu). Treatment is limited to dealing with the symptoms of renal disease.

Amyloidosis is a common familial disease found in Abyssinians, Oriental Shorthairs and Siameses. Amyloidosis occurs when protein is lost from an increase permeability of the glomerular membrane due to the abnormal deposit of the amyloid protein. Amyloidosis can occur rapidly causing renal failure to develop within one year of diagnosis. In other cases the effects on the kidneys is mild and cats may live without the disease ever being detected. Symptoms include poor hair coat, weight loss, polydipsia, polyuria, and anorexia. Proteinuria is a variable finding and may not reflect the severity of the disease. In order to diagnosis amyloidosis a renal biopsy must be obtained. Treatment is limited to dealing with the symptoms of renal disease.

Acquired CKD can result from any disease process that injures the kidneys to a point where the nephrons can no longer function appropriately. Some common diseases that lead to CKD include: feline infectious peritonitis, neoplasia (renal lymphosarcoma), hyperthyroidism, glomerulonephritis and chronic tubulointerstitial nephritis.

Feline infectious peritonitis (FIP) affects the kidney, liver, mesenteric lymph nodes, central nervous system, and eyes. Besides renal failure symptoms, fever, lethargy, anorexia and weight loss may be present. In order to diagnosis FIP, a fine needle aspiration or biopsy of the enlarged kidney must be obtained.
If left untreated, hyperthyroidism may cause renal failure. Thyroid hormones help support GF rate by increasing renal blood flow. Hyperthyroidism generally results in systemic hypertension which could be transmitted to the glomeruli, causing glomerular hypertension and glomerular hyperfiltration. Unfortunately, when treating the thyroid disease, the hypertension will dramatically decrease causing a decrease in renal blood flow and GF rate. Studies have reported that 14% of hyperthyroid cats have pre-existing renal disease, while approximately 30% of hyperthyroid cats become azotemic after therapy of hyperthyroidism.

Lymphosarcoma (LSA) is the most common renal neoplasm of the cat. It usually affects both kidneys. Approximately 50% of cats with renal LSA are feline leukemia positive. Treatment is aimed at dealing with the kidney disease and using conventional chemotherapy. Unfortunately, prognosis is poor.

Glomerulonephritis appears less common in the cat than in the dog and it is generally classified as idiopathic. There are generally two types of glomerulonephritis: classical nephrotic syndrome and chronic renal failure. With classic nephrotic syndrome edema, ascites, proteinuria, and hypoaalbuminemia are often present. Azotemia may or may not be present. Laboratory findings usually include proteinuria, hypoaalbuminemia, hypercholesterolemia, and nonregenerative anemia. A biopsy must be performed in order to make a diagnosis. Cats that have edema and ascites without azotemia can be treated with loop diuretics (furosemide) and prednisolone. Enalapril is not only used to treat the hypertension, but also has been found to have additional beneficial effects such as reducing the proteinuria and slowing the rate of disease progression.

Chronic tubulointerstitial nephritis (CTIN) is the number one finding in CKD cats (approximately 70%). Chronic tubulointerstitial nephritis occurs gradually over years and results in renal tubules atrophy and interstitial fibrosis which results in decreased renal function. The causes of CTIN are numerous and, despite a thorough work up, no cause may be determined. Failure to identify the cause is likely due to the numerous diseases that cause similar changes to the kidneys. In 2012 researchers from Hong Kong isolated a paramyxovirus known as feline morbillivirus from domestic cats. Approximately 12.3% of the cats tested using PCR methods were positive for morbillivirus. They then looked at what other common diseases these same cats were associated with. Ultimately they were able to isolate the virus from the kidneys. In conclusion approximately 7 out of 12 cats that had the morbillivirus also had tubulointerstitial nephritis.

Conclusion
Because causes can be numerous, it is important cats with kidney failure receive a complete diagnostic work up in order to diagnose the underlying cause. Kidney failure is not a death sentence for cats. With advances in treatments many cats live long and productive lives with both AKI and CKD.

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Though not as common as in the feline, dogs can certainly experience kidney failure for a variety of reasons. Due to the length permitted for the notes, physiology of the kidneys and renal failure in general is discussed in “The Cat & The Kidney”.

**Acute kidney injury**
Acute kidney injury (AKI) results from a dramatic decrease in GF rate because of either prerenal, intrinsic renal or postrenal causes. Many causes of AKI are reversible, pending diagnosis and treatment is made early.

**Prerenal azotemia** is not caused by primary kidney disease, but rather by a decrease in cardiac output resulting in inadequate blood supply to the kidneys. When mean arterial pressure (MAP) starts to fall below 60 mmHg, perfusion to the kidneys becomes compromised. Prerenal kidney azotemia is reversible because it is not associated with morphologic damage to the kidneys.

**Intrinsic damage** causes include from toxins, infectious diseases and ischemic causes.

In the dog ethylene glycol toxicity is the most common nephrotoxic drug likely because of its wide availability. Ethylene glycol causes a toxic effect by forming oxalate which binds to plasma calcium and forms calcium oxalate crystals in the renal tubules. The calcium oxalate crystals clog the tubules leading to AKI. Mortality rates are between 50-70%. Besides treating the AKI, dogs are usually given fomepizole (Antizol-Vet®), a synthetic alcohol dehydrogenase which helps to absorb the ethylene glycol.

Vitamin D (cholecalciferol) toxicity is more common in the dog than in the cat. Cholecalciferol can be found in some rodent poisons, vitamin supplements and psoriasis creams. AKI occurs because cholecalciferol is metabolized to calcitriol which, in turn, increases intestinal, bone and renal absorption/resorption of calcium. The toxic effects are due to the hypercalcemia and hyperphosphatemia. Initial detoxification should occur followed by treatment for AKI, hypercalcemia and hyperphosphatemia.

Raisin and grape toxicity in dogs has been well documented since 1999. The exact toxic substance in the grape is still unknown. The amount of ingested raisins and grapes needed to produce nephrotoxic effect varies from 3 to 57 g/kg. Within 12 to 24 hours of ingestion dogs develop vomiting, anorexia, diarrhea, abdominal pain and AKI.

Leptospirosis is a zoonotic disease that infects dogs. Transmission occurs through contact with urine, bite wounds or ingestion of infected tissues and has a 1 week incubation period. There are two forms: acute or chronic. Symptoms with the acute form include lethargy, anorexia, shivering, and vomiting. If it progresses, uremic symptoms become present. Approximately 70% to 85% of dogs survive with treatment. For those that do survive chronic renal and liver dysfunction are common. Treatment is aimed at treating the AKI signs and antibiotics (doxycycline).

Lyme disease is caused by the spirochete bacterium Borrelia burgdorferi and is transmitted by the Ixodes tick (deer tick). Many dogs carry Lyme disease without ever being symptomatic. Those that become sick often present with signs that include lameness (shifting leg), fever, lethargy and anorexia. In dogs that have chronic ongoing Lyme disease Lyme nephritis/nephropathy can occur. Prognosis is guarded to poor for dogs that develop AKI. Besides treating the AKI symptoms, doxycycline is the preferred antibiotic used.

**Chronic kidney disease**
The average age of dogs that develop chronic kidney disease (CKD) is around 7 years. The causes of CKD are numerous and can be congenital, familial or acquired.

Congenital causes are often suspected based on the age of the dog, breed and family history. Polycystic kidney disease (PKD) is more common in cats, but West Highland White Terriers, Bull Terriers and Cairn Terriers can carry a recessive trait to developing PKD. PKD is characterized by the presence of multiple fluid-filled cysts which can result in the enlargement of the kidneys. Treatment is limited to dealing with the symptoms of renal disease.

Amyloidosis is an uncommon familial disease found mainly in Shar-pei (Shar-pei Fever) and is the most common cause of CKD in the breed. Amyloidosis occurs when protein is lost from an increase permeability of the glomerular membrane due to the abnormal deposit of amyloid protein. Amyloidosis can occur rapidly causing renal failure to develop within one year of diagnosis. Symptoms include poor hair coat, weight loss and anorexia. In the case of Shar-pei Fever signs may include an intermittent fever lasting 24 to 36 hours and, as the disease progresses, signs of renal and liver failure may occur. In order to diagnosis amyloidosis a renal biopsy must be obtained. Treatment is limited to dealing with the symptoms of renal disease.

Fanconi syndrome is a disease that is inherited in the Basenji, but can also be acquired. Acquired causes include heavy metal intoxication (lead, copper, mercury), amyloidosis, neoplasia (multiple myeloma), hyperparathyroidism and vitamin D deficiency. Fanconi syndrome is a disease where the proximal tubule function of the kidney is affected, which results in decreased reabsorption of electrolytes and nutrients. Glucose will “spill” into the urine while the body’s blood glucose is normal. Fanconi syndrome dogs are resistant to ADH which causes them to develop nephrogenic diabetes insipidus.
Glomerulonephritis occurs in roughly 27% of dogs diagnosed with CKD and it is generally classified as idiopathic. There are generally two types of glomerulonephritis: classical nephrotic syndrome and chronic renal failure. A biopsy must be performed in order to make a diagnosis.

Chronic tubulointerstitial nephritis (CTIN) is the number one finding in CKD dogs (over 50%). Chronic tubulointerstitial nephritis occurs gradually over years and results in renal tubules atrophy and interstitial fibrosis resulting in decreased renal function. Often, the causes are numerous and, despite a thorough workup, no cause may be determined. Failure to identify the cause is likely due to the numerous diseases that cause similar changes to the kidneys.

Treatment of kidney disease
In general, all kidney disease is treated the same, with the exception of a few additional therapies depending on the disease. Fluid therapy, monitoring acid-base and electrolytes, ensuring appropriate nutrition and monitoring for anemia and hypertension is important in every kidney failure patient.

Fluid therapy
The gold standard is diuresis. It’s important to account for any body water deficits as well as any ongoing losses such as vomiting and diarrhea. Initial solutions are generally isotonic crystalloids. If a patient has cardiac disease or hypernatremia, low sodium fluids should be used (such as 0.45% NaCl). Once a patient is rehydrated, they should produce 1 to 2 mL/kg/hr of urine. Ideally urine output should be monitored to ensure that fluid therapy is adequate. The most accurate way of monitoring urine production is by placing an indwelling urinary catheter. In the case of oliguria, additional fluids or treatment may be required. Furosemide, dopamine and other osmotic diuretics (mannitol) can be used to increase urine production.

Acid-base/Electrolytes
Acid-base status and electrolytes must be constantly monitored. Metabolic acidosis and hyperkalemia are common in oliguric AKI patients and is commonly seen in feline urethral obstruction patients. In severely hyperkalemic patients brady cardia, peaked T waves, loss of P waves and life threatening cardiac arrhythmias can be seen. In such severe cases, several treatments can be initiated to help deal with the hyperkalemia.

Chronic renal disease feline patients are more likely to suffer hypokalemic effects because the potassium is closely regulated by the kidneys. Dogs rarely experience hypokalemia, but more commonly, they experience metabolic acidosis and hyperkalemia during AKI. Signs of hypokalemia typically include anorexia, polyuria, vomiting and weakness. In serum potassium levels less than 2.5 mEq/L, neuromuscular signs can occur which include a reluctance to move, a stiff gait, ventroflexion of the neck, and tremors. Intravenously administration of potassium, at a constant rate infusion, can be used to correct initial hypokalemia. Once hydration is adequate, cats rarely require oral supplements at home.

Hyperphosphatemia is commonly observed in CKD patients because the kidney plays an important role in excreting phosphorus. Typically hyperphosphatemia does not produce clinic signs, but it can lead to the progression of secondary hyperparathyroidism which can lead to death. Secondary hyperparathyroidism can lead to muscle weakness and central nervous system disturbances. Typically calcitriol (the most active metabolite of vitamin D) is used to treat secondary hyperparathyroidism because it inhibits parathyroid gland growth (which is a task vitamin D receptor does in the parathyroid gland).

Another common finding in chronic renal disease patients is hypocalcemia. Roughly 26% of CKD cats suffer this electrolyte disturbance. Intravenous calcium gluconate may initially be administered to help correct any serious hypocalcemia and then oral calcium supplements may be used after. There are many oral calcium based products that can be administered for the added phosphorus-binding effects. Serum calcium and phosphorus levels should be monitored every 2 weeks initially and then as needed.

Hypertension
Kidneys that are suffering from acute or chronic disease are more at risk because they lose the ability to autoregulate renal blood flow and glomerular filtration rate. These changes are transmitted directly to the glomerular capsule which results in glomerular hypertension. Ultimately all patients with hypertension should receive an antihypertensive drug to help prevent further damage to the kidney and other organs. The choice of which drug(s) to use is dependent on the degree of hypertension, the presence of target organ damage, the available routes of administration and the underlying disease.

Anemia
Often times, patients with CKD suffer from moderate to severe anemia. There are a myriad of reasons for patients to suffer anemia. Red blood cell transfusion is rarely recommended because of the decreased life span of red blood cells with uremic patients. As the patient’s kidney values decrease, the anemia starts to resolve. Red blood cell transfusion is only recommended for patients undergoing surgery. Recombinant Human Erythropoietin and darbepoetin are both commercially available and is used to treat CKD anemic patients. Depending on the dose, it can take 2 to 8 weeks for the hematocrit to rise to low normal.
Early detection of renal disease
IDEXX created a test in 2014 that detects renal changes earlier than other testing methods. It worked by testing SDMA (a methylated form of the amino acid arginine). SDMA releases into circulation when protein is broken down. It resides in almost every cell in the body. It is eliminated only by the kidneys. Therefore if the kidneys have any disease the filtering of SDMA does not occur as well and the biomarker increases. IDEXX reports SDMA can be used to detect CKD an average of 9 months earlier in dogs and 17 months sooner in cats.

What does this mean? Because treatment in pets with renal disease is limited to nutrition, SQ fluids and treatment of GI symptoms, treatment is unlikely to be altered with such an early diagnosis. Many times the pets with increases in SDMA are not exhibiting GI symptoms. No studies have been published on if earlier intervention with SQ fluids and nutrition increases life expectancy longer than average. In both dogs and cats increases in SDMA should prompt veterinarians to determine the cause of the increase.

Renal transplant/Intermittent hemodialysis
More than 90% of cats survive past one year and most survive to three years. The success rate of canine renal transplants is not as great as in cats. The University of California at Davis only has about a 40% success rate while other universities have discontinued the program due to the poor prognosis. Clients who wish to pursue this treatment option must be prepared to pay over $13,000 and adopt the dog/cat who donates the kidney. The actual renal transplant is long, roughly 4 hours, and complications include bleeding, hypertension, embolism, infection and, ultimately, rejection of the new kidney. Lifelong medication and monitoring is required. If kidney failure is manageable, transplant surgery is not recommended.

Intermittent hemodialysis has been a successful treatment in managing renal failure in cats and dogs. Its purpose is to correct the effects associated with uremia by filtering the blood across an artificial “kidney” membrane outside the patient’s body. Due to its expensive price tag, complications, and limited facilities performing the treatment, it is commonly reserved for pets suffering from AKI. Complications include neurologic (caused by disequilibrium from shifting osmotic gradients), gastrointestinal (vomiting, nausea) and hypotension during the treatment. In 2000 it was reported that 60% of cats with AKI undergoing intermittent hemodialysis survived. It is unknown how many would have died without treatment.

Continuous renal replacement therapy (CRRT)
As the name implies CRRT relies on continuous gradual blood purification. The patient’s blood is filtered until the kidney function returns to normal. CRRT is almost always used for acute kidney injury, but can be used for a toxin ingestion as well to help diuresis the body. The patient’s blood is passed through a filtration circuit tubing in a machine to a semipermeable membrane where waste products and water are removed. Replacement fluid is added to the blood and is returned to the patient.

In human nursing literature for CRRT it states that the benefits are: the ease of systems used to treat AKI patients; enables higher doses of therapy to be delivered consistent with current clinical literature; allows for 24 hour therapy; allows for better hemodynamic stability; allows for volume reduction allowing for fluids & nutrition; and allows for cytokine removal. While complications are less than that of intermittent hemodialysis the technician staff must constantly monitor these patients. Coagulation disorders can occur so clotting times need to be constantly monitored. Hypotension can be a problem as well likely due to the large amount of blood needed for the CRRT unit (50-84mls) as well as the reduction of blood volume. Patients weighing as little as 2.4kg have been successfully treated with CRRT leaving one to believe there may be no size restriction. Certainly more research must be done in veterinary medicine, but the limited research available has shown CRRT to be very effective and safer than intermittent hemodialysis.

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Powerful Management: Understanding How to be a Servant Leader
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The concept of servant leadership is not a novel idea in the least, however it seems that most new managers are more focused on passing down tasks and being the “boss” versus leading through influence. This is a natural progression for all leaders, but it’s important that we take a growth mindset and strive to convert ourselves as “servants” to our teams. This conversion results in a servant first mindset versus a leader first approach.

The servant leader prioritizes the needs of their team members above their own, and ensures their growth & well-being, and ultimately molds them into leaders themselves. Certain behaviors lend themselves to a servant leadership approach. These include leaders who actively listen and desire to understand, exhibit acceptance as well as empathy, foresight and have a talent for persuasion, and visualization of future improvements.

Leaders with an ability to truly listen will create a team where employees feel their voices are being heard and the leaders of their organization support them and are loyal to their team. Listening is certainly an art, not only consisting of paying attention and sealing your lips, but also being cognizant of subtle communication from staff including their facial expressions and body language. Fine tuning the art of effective communication with our team members allows us to become more compassionate and tailor our approach to each individual vs a one-size-fits-all mentality.

When communicating with your team, be sure to ask questions and expand upon their ideas. Try to learn from team members versus judging or criticizing their approach. When you are engaged, and follow up with team members after these discussions, not only will they feel appreciated, but you may gain insight into alternative ways to approach a situation.

Acceptance and empathy comes from an ability to understand your team members’ perspectives and intentions beyond your own viewpoint and natural assumptions. With an open mind, one is better able to understand why a team member behaved in the way they did versus responding with frustration. This builds respect between the leader and team member, and is a good stress management technique for leaders as well.

Foresight utilizes past experiences to help plan out future decisions by appropriately evaluating the current situation and understanding potential consequences. Once a plan has been devised, an influential leader is able to persuade the key opinion leaders of a practice to come aboard. These team members can then more easily influence the rest of the team.

Visionary leaders and servant leaders tend to go hand in hand. A visionary approach involves converting a broad revelation into a specific, tangible business plan. This plan is broken up into smaller goals to achieve throughout the year, along with the delegated responsibilities of other team members. The larger goal is always in the framework of the visionary leader’s plan, but they take time to appreciate and celebrate smaller achievements with the team along the way. It is helpful to keep a journal dedicated to future concepts and projects you would like to implement over the next 5 or 10 years in the company. You can slowly integrate these concepts into the company’s year business plan, and adapt them into step by step plans.

Lastly, integrity is crucial to the success of a servant leader. They keep the team members’ own sense of moral compass in line with the values and mission of the company. When employees feel a supervisor has a strong sense of integrity, they trust them and allow themselves to be more vulnerable and open as they know their manager has their best interests at heart.
Content employees equals a successful practice. When you take the time to cultivate a positive culture in your practice, team members will be more efficient, more willing to come in on time and stay late when needed, and client satisfaction will also improve. High amounts of turnover and increased cost of training programs can all be associated with negative morale. It’s important for employers to have culture development take a priority in their practice and to keep it at the forefront of team discussions and feedback requests.

One of the first recommendations for repairing (or boosting) a practice’s positivity and sense of culture is to improve and open up communication. Schedule out one one meetings, as well as team meetings, with the team on a regular basis throughout the year. Preparing these as part of your schedule beforehand will ensure they don’t take a backseat to the busy flow of the hospital. In these meetings, include self-care check-ins and provide mental health resources when needed, such as an employee assistance program. Show interest and prioritize individual development in meetings, as well as provide the necessary resources financially to help them attend. Give your team members a voice in meetings and engage them in discussions regarding how management can improve their quality of life at work. In order to encourage honest feedback, it’s important to crackdown on any observed negative fallout from those in management after these discussions. Leaders should come in with a growth mindset, and be willing to hear feedback and find truth in even the most difficult responses. Equally important, team members need to see a change after opening themselves up and sharing their opinions. If nothing improves, they will find the exercise pointless and opt to not allow themselves to be as vulnerable and honest in future discussions.

Exercises and reminders of gratitude in the workplace help boost the overall positive culture within a practice. Encourage gratitude reminders in team meetings - this can be a quick exercise where each team member shares one thing they are grateful for at work. Gratefulness can be found in their career choice (I’m grateful I get to help animals each day/make a difference in the lives of our clients), their teammates (I’m grateful that Kelly is always willing to come in early/stay late when we need her), or even their benefits at work (the ability to take time off for CE/to not work weekends/etc.). This exercise retrains the brain to look out for more positivity on a daily basis.

“Millennials” or not, all humans by nature crave appreciation and praise. Create rewards systems that acknowledge hard work from employees. There are many methods to provide kudos to your team members including verbal praise via a team kudos board posted in a common area of the hospital, or through an email, or even through some HR software programs. It is also important to first perform a motivation and rewards assessment for all employees to determine how they want to receive this recognition. Some individuals may prefer that acknowledgements are not public, for example, and this should be respected, or otherwise will prove counterintuitive. Other forms of kudos may be via giftcards, team lunches, extra paid time off, or physical team awards/certificates. As a manager, it may be helpful to schedule time every week to ensure you allocate kudos, as this can easily slip away from us when the hospital becomes busy and we become more focused on fighting fires.

Team building is a great way to promote a positive culture within the workplace. This does not have to consist of the clichéd trust exercises, but can be something as simple as playing a trivia game together, attending a yoga class, or practicing meditation. The goal is to make the activity fun and engaging for team members so they get to know each other better and build a rapport to strengthen their work relationships. A great team building activity that also can help the community is to schedule volunteer events throughout the year where the team learns to work together in a different capacity outside of the hospital. This can foster positive, problem-solving relationships among team members.

Through open communication, regular encouragement and relationship building, team leaders can foster a positive, uplifting environment. This will make team members truly want to come into the practice every day and try to make a difference. Creating a positive culture does not happen overnight and does need to be regularly maintained and cared for, but will result in greater business success and a happier work atmosphere for all.
Creating a dynamic team starts with utilizing your current team members’ strengths and maximizing their contributions. When a practice has poor morale, and actively disengaged employees, it’s easy to think there is no turning the practice around and you need to start from scratch. However, there are ways to improve the practice by building on assets of team members, clarifying job descriptions and narrowing swim lanes, as well as allowing autonomy and creating a culture of trust and empowerment.

Take the time to learn more about your employees when you meet with them one-on-one. Find out their career goals, passions and motivators. A good tool to retrieve this information is through stay interviews with your employees on a regular basis. This will allow you to discover what drives them to do their best day to day, as well as which aspects of the job they love the most and which they loathe. The stay interview is a good opportunity to discuss what they want to learn more about, and their overall career goals (even if they don’t ultimately end with being at your practice, or in veterinary medicine). From the information gathered, you can learn more about what they can contribute to the practice as individuals and what would keep them happiest in their positions. Your team will also likely work together in a much more cohesive, peaceful manner when they are taking care of tasks that best fit their personality, work style and skillset.

Through this process, a manager should create clear, well defined job descriptions and expectations. Try to show employees the connection between their day to day tasks and the company’s overall goals and mission. This will create a more efficient team, as well as one that has more buy-in to the practice. Make sure the supervisors are not holding onto the majority of the tasks (resistance to delegate) or micromanaging the team. This creates frustration and ambivalence within the team. If the guidelines and goals have been communicated well, team members should be allowed to work autonomously without fear of failure. Trying to have your hands in every aspect of operations at all times will not mean mistakes are never made. Employees who do not feel trusted are more likely to feel anxious and stressed, and create more mistakes due to this. Also supervisors with too much on their plates will likely drop the ball more frequently. Mistakes are a natural part of human nature, so allow the possibility for them to occur and focus more on how you respond to them. Look at how you explained processes and trained your staff to see where you could have improved as a leader. Communicate the “why” of operations to help team members truly understand the meaning behind their work, and fewer errors will occur.

Encourage a collaborative environment among the team where ideas are openly shared and decisions are made together. Utilize personality tests and include the results in regular conversations with the team so they better understand how to work with their teammates and resolve conflicts. Team building exercises can help maximize teamwork and contribute to a fluid workflow. Communication building can help team members better understand how their coworkers prefer to discuss issues and concerns and allow them to articulate their needs in the best way possible day to day. As a leader, provide multiple avenues of communication with the team including team meetings, email, one-on-one meetings, instant messaging and even message boards/forums.

As goals are met, ensure that team members are recognized and rewarded. This recognition will encourage individuals to continue to maximize their performance and will keep them engaged. Be sure to perform motivation and recognition assessments so you can appropriately acknowledge team members’ work in the way they prefer, otherwise the act will prove counterintuitive. Remember as long as the team is making strides towards the company’s goals at a reasonable pace, this is a success and will keep the team content within their positions. Try not to focus too much on specific results being met by unrealistic deadlines, and give some leeway for improvement and changes to take place.
How to Survive in this Field as a Veterinary Technician
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Being a veterinary technician is tough. Not many survive. Studies show that veterinary technicians have an estimated 30-35% turnover rate (LinkedIn Career Outlook). The national average turnover rate is only 12-15%. The 30-35% of veterinary technicians that decide to leave the profession do so between 5-10 years after they started. This makes the workforce very young with most veterinary technicians being under 35 years of age. Staggering statistics. Why do so many veterinary technicians enter the workforce and leave so quickly? How does one survive in this field? More importantly, how does one thrive?

The failures of the profession
According to United States Bureau of Labor Statistics in May 2012 the average yearly veterinary technician salary was $34,000. That being said there is quite a large range of what a technician can make. Some make just minimum wage and others make six figure salaries.

The job, as a whole, is physically and mentally demanding. Because of the lack of public awareness of the profession the public tends to disregard a trained veterinary technician's skills and knowledge. The animal patients of the hospital may carry diseases and may be aggressive. The dynamic between veterinary doctor and technician can be difficult. The job requires employees to be able to lift 40-50 pounds, stand on their feet all day, bend down, kneel down and crouch in numerous yoga-like positions to conform to the size of the patient they are working on. Urine, feces and vomit occur on a daily basis and it is required to clean up all three on a routine basis.

The hours can be tough. Working weekends, nights, holidays, long hours are all part of most veterinary technician's job description. Being called in to cover or being on call is normal in the profession. It is a difficult profession to also be a parent of a child in because very few hospitals support "normal" hours for parents.

The success of the profession
There are plenty of jobs. In fact, according to United States Bureau of Labor Statistics, the profession of veterinary technology is expected to grow about 30% over the next 10 years. That is significantly higher than most professions. Salary is also expected to increase slightly higher than the rate of inflation.

The public awareness of what a veterinary technician is has increased dramatically over the past 10 years. Veterinarians are now being trained on how to utilize the skills of a veterinary technician and how to work with together with them as a team. This allows technicians to utilize their skills more.

The patients of the veterinary hospital are arguably the best patients of any medical profession. They cause veterinary technicians to make baby noises at them, smile and laugh over their antics.

What a veterinary technician can do is quite diverse. While most technicians work in a general practice, the field is one of the largest of any professions. A veterinary technician can choose which species to work with (lab, large, wildlife, small, exotic, zoo) as well as the specialty (radiology, emergency, anesthesia, surgery, behavior, etc). They can choose to work for large corporations as consultants, researchers or managers (pharmaceutical, pet food industry) to small hospitals (head technicians, specialty, general practice).

So how does one survive?
What is YOUR Passion? The amount of different areas that a veterinary technician can work in is vast.

If you are bored or feeling burnt out at your job ask yourself "what do I enjoy about this job?" Chances are you don't dislike all of it. There are likely some parts of it you really do enjoy. If you find yourself enjoying puppies perhaps you really love behavior science. If you already work in specialty medicine what area of it still gets you excited? Do you love when a pet needs a blood transfusion?

Taking Ownership Of Your Passion You despise running fecals, but you don't mind IV catheter placement. You find yourself fearing CPR, but you don't mind caring for critical pets. How do you do the thing you love the most? Most of the time what you love to do is a job in and of itself. Depending on how specific your passion is you may need to realize you might have to do some things that are not your passion so that you can do what you love most of the time. Knowing is half the battle and from there you can then formulate your battle plan.

The most common thing that a majority of burnt out technicians do when they have found a passion is to go in to that specific area of medicine. If you already work in a specialty you can get even more specific. If you work in an ER and are tired of vomiting and diarrhea, but you really find yourself loving when an animal needs a blood transfusion you can suggest having your own practice start a blood transfusion program. Running an in-house blood bank or even a successful blood donor program looks great on a resume.
Larger hospitals may already have an in-house blood banking program and may be looking for a new technician. You don't know what you may find until you start looking.

**Taking On A Specialty** In 1993 NAVTA created the Committee on Veterinary Technician Specialties (CVTS) and by 1994 NAVTA grants the first provisional specialty in veterinary technology to the Academy of Veterinary Emergency and Critical Care Technicians. The demand and need for specialties is because of the increase level of veterinary care to animals. Continuous improving of medicine lends itself to those that specialize in certain areas. The veterinarians were the first to specialize and there are currently over 20 specialties for veterinarians. Since 1994 technicians can specialize in over 11 specialties. https://www.navta.net/specialties/specialties. All specialties are defined by the title Veterinary Technician Specialist (VTS).

Much like board certification for veterinarians, each technician specialty has slightly different requirements. Generally it is a 1 year application process. The application usually requires letters of recommendation, X amount of hours in that specialty, X amount of cases and then X amount of professionally written case reports. The application process reviews whether you, as a technician, has a higher level of knowledge for that specialty. Reviewers of the applications are looking for your knowledge of WHY you did certain things, not just the fact you did them. What complications occur after an animal is shock, why did you give that drug, etc.

Unlike a veterinarian who wishes to specialize there are no technician residency programs. If your application is accepted you have a 6 month to 1 year self study. This is your time to study everything in that specialty. Then you sit for a board certifying exam. Some VTS organizations have hands on parts, some offer only a written exam. All of them occur within 1 day and are usually given only once a year. The exam asks difficult questions. It is meant to ensure those that pass the test understand pathophysiology, pharmacology and a higher level of knowledge then you received in your regular school education.

If you pass you earn the VTS title and you are considered at the forefront of your field. VTS technicians are consider leaders. While most earn a higher salary, not all employers honor obtaining a VTS with a raise. That being said a VTS opens more doors and opportunities. Being a VTS opens doors for speaking, publishing and teaching.

**Education**. By 1999 there were 80 programs in the USA that were AVMA accredited. In 2003 the number of accredited programs climbed to 103. In 2014 there are over 220 AVMA accredited veterinary technician schools in the USA. Education is available in a classroom or online. If you have already obtained a college education as a veterinary technician you may want to go back to school to further it. For example if you have an associate’s degree and are interested in management going back to school to obtain a bachelor’s degree in management may help you go for your passion. If you just wish to learn about a particular area (emergency, pathology, pharmacology) there are plenty of online courses or conferences which offer continuing education in just about anything you can think of. Increasing your knowledge definitely leads to more opportunities as well as increases in salary. Putting your continuing education on your resume shows potential employers that you are committed to your field and that you are current in medicine.

**Learning Never Stops** If you have been working in the profession and it’s been more than two years since you did any continuing education then you are already setting yourself up to fail. Medicine is a constantly evolving and updating field. In order to survive in this profession you must evolve with it. Failure to do so will cause you to fail for your patients and yourself. You likely went in to this profession because you loved pets and medicine. If you are finding yourself stagnant in your job it’s likely because you stopped learning. Learning not only increases your knowledge, but elevates the entire practice and the care to the pets you work with.

**Toxic Work Environment** Sometimes despite knowing your passion is simply not enough if you work in a toxic work environment. It’s possible you are not even aware that you are in a bad working environment or it’s possible you are the cause of it. When polled with the question “list something in your last job you did not enjoy” the number one answer was “gossip”. The second most written down answer was “laziness of fellow coworkers”.

No one likes gossip. Gossip occurs when an individual speaks about another individual when they are not present. There is “positive” gossip and “negative” gossip, but both forms can be harmful and not welcomed by most people. While technicians say they don’t like gossip in a work environment the reality is that most people will listen to, enjoy and even feed in to the gossip being given to them. A work environment where gossip overruns the practice is a toxic work environment. It breeds distrust, disrespect and dislike amongst coworkers. You will never survive in a toxic environment. If people around you are telling you negative thoughts day in and day out you will never excel. You will be filled with negativity, thoughts of why the practice is bad to work at and you will experience demotivation. Conversely, if you are the one providing the gossip then you are the one who is demotivating the rest of the staff.

There is no need for a “negative nanny” in a practice. That person is the worst of the gossipers. They are the one who does nothing but constantly tells others what’s wrong with everyone else. They will tell you why the day is so hectic. They will tell you about why that client was horrible and why the doctor made wrong the decision. This person sets up others to fail. It takes a strong person to tell that employee to ignore them when they start complaining.

If your work environment is toxic you have three options: Recognize and Ignore It, Express your Concerns to the Manager or Leave and Find a Healthier Environment. There are some people who recognize it’s a toxic environment and have the ability to ignore the gossip and negative issues, put blinders on and still thrive. You should always tell your manager or owner if you find
you yourself in a toxic environment. Be sure when you express your concern you don’t play the blame game. Sit down and express your concern about this individual dragging down the team as a whole. Express your concern about the overall team’s health. Lastly you can leave. Unfortunately there are some working environments that are simply toxic. It’s not that they can’t be fixed, but it’s that they cannot be fixed by you. This type of work environment requires aggressive help from management or the owner. If you have expressed your concern and nothing has changed sometimes it is best to move on.

Dress the Part The saying “dress of success” is a popular one for good reasons. You will not succeed in the field if no one takes you seriously. You work in medicine. You are a medical professional. Stains on your scrubs, holes in shoes, mismatched scrubs look unprofessional. How will clients ever take the veterinary technician profession seriously if they see a green scrub top that has bleach stains on it and blue scrub pants that have rips in the knees?

Dress in a way that elevates the profession. That includes when you are representing your profession at events. You are representing your profession. If you don’t look professional then how will veterinarians, management, front office staff view the profession? When you go to an interview you should dress appropriately. If you are coming from work and truly do not have time to change in to business attire apologize to the person interviewing you.

Outside Life You must have a healthy work-life balance. Yes, there will be days you get stuck late at work. Yes, there will be days you get called in. If you work on salary you may find times you work 50-60 hours a week. Go home, unwind and stop going online or on your smartphone to check on work! If you are in a management position be sure to set boundaries for your employees and the company. It should be normal to assume that not everyone is available 24/7. If your job asks you to be on call be sure to be compensated for it. Most states require hourly employees to be paid for on call if it is a requirement of the job. If you are salaried and it is a requirement be sure to set boundaries and make sure the request is reasonable. Above all else you must find time to go home, get away from work and live life...and hug your own pets!

Recognition of Stress Technicians work in stressful work environment. It is a labor intensive and emotionally charged profession. Technicians are constantly helping others. They help clients, pets and their coworkers. Unfortunately they often drop the ball when it comes to helping themselves. You cannot survive in this profession if you do not help yourself.

Burnout and compassion fatigue are two different things which may be causing you not to survive in this profession. Burnout is a cumulative process in which the individual slowly lacks empathy for a particular situation and is due to an increase in stress or workload. The individual often has feelings of anger and does not care about their work as much as they used to. They watch the clock and know exactly how many minutes are left in each shift. If they see a mess they walk over it rather than stopping to clean it up because they simply have stopped caring due to burnout. Compassion fatigue is an emotional strain from the consequences of traumatic events such as a stressful case or event. An individual experiencing compassion fatigue may have nightmares about a particular event, be more emotional or think about a particular event if something triggers it. Perhaps they poured their heart out over a tragic case of a young dog that was hit by a car. After a week of trying to save the dog it died. That technician can still show empathy to other patients, but may be more emotionally invested, cry if they see the same breed of dog or not want to work with a hit by car for some time. The two syndromes can be experienced together.

Recognition is the first step. Realizing that you need a vacation or a break from work for a few days is important. Talking to your manager, coworkers or a professional will help as well. If you have been in this business long enough you have a good chance of experiencing one or both of these things. Everyone has different coping mechanisms and it’s important to find yours.

Besides burnout and compassion fatigue the simple nature of the job can play as toll on an individual. Taking care of yourself while on the job is equally important to taking care of yourself after. Even if you work long shifts and the clinic is very busy you must stop to do the following: eat, stay hydrated, go to the bathroom and laugh. Failure to do these things will result in exhaustion and misery. It does not take long to do any of those things and taking a few minutes to do one of them will make you happier in your job. A happy technician equals better care to clients, pets and coworkers. Equally important is living a good lifestyle when you get home. Getting a good night’s sleep, eating well and working to stay healthy will keep you performing better at your job.

Surviving an Overnight Shift Since technicians have such a wide range of jobs they can do you may be one that has a schedule that requires overnight shifts. Overnight shifts are bad for your health. There are some people that do better with overnight shifts. These people are those that can actually sleep during the day. There are other people that can suffer through them. They don’t sleep well during the daytime, but they get enough sleep to function. Then there are some people who simply can’t work an overnight. If you fall in this group and recognize you can’t sleep at all during the day then it’s best to change jobs or ask for a different schedule. It’s simply dangerous working without sleep. If you are in the first two groups you must do you best to take care of yourself.

It’s not natural to work nights. Your body knows this. Overnight employees have a harder time losing weight, your kidneys actually don't regulate fluid balance as well so you may find yourself thirstier or having to urinate more frequently. Overnight employees have weakened immune systems. The lack of sleep can cause an overnight employee to fall asleep while driving or not be able to think as clearly which may lead to medical mistakes. Both of these can be deadly. Recognition of how your body handles an overnight shift is important.
Speak to your doctor about medication that may be beneficial to help you to sleep, eat healthy and find some rituals that work well for you. Black out shades, no caffeine before the end of your shift and white noise in the bedroom may all help with you sleeping during the day.

How have you survived?
I have survived by following all the advice I’ve already given you. I started off my first job in a general practice. I was very fortunate to join a high quality, nontoxic work environment. The owner promoted technicians and encouraged the staff to utilize their skills to their fullest. I wish for every new technician to find a healthy work environment for their first job. It was in this general practice that I found my passion. By year five I was bored with vaccines and fecals, but I found myself getting very excited when a hit by car arrive or a sick dog come in. I decided to push myself and write an article for a magazine and I was thrilled when it was published. Writers do not make any money. Writers write to push themselves and to provide an article to a publication they hope will get others equally excited about what they are writing about.

During my first 5 years as a technician I continued to educate myself. There was no CE budget at the hospital so I had to save up my own money so I could attend local conferences. There was no such thing as “online” learning at that time. I also started working part time at an emergency practice. It was then that I realized I needed to leave the comfort of my general practice and go full force into my passion. I joined my first emergency practice and knew it was where I belonged. I loved everything about the specialty and found myself wanting to learn everything there was.

I had written a few articles and decided that I wanted to try lecturing. I have no idea what possessed me to reach out to a very famous and popular veterinary technician speaker, but I am forever grateful for her advice to me. I asked how she became involved with speaking as I was interested in trying my hand at it. She told me what I still consider the best advice. “Do a case report at IVECCS.” She was right.

Presenting a case report is a great way for a new speaker to get into the speaking circuit and for them to see if speaking is something they really want to do. While the case report is meant to present an interesting case, it is actually meant to also showcase the speaker's skills. It’s a good way to introduce you to speaking, introduce yourself to some key people and also to challenge yourself. After presenting my case report at IVECCS doors started to slowly open. It was a lot of work for years, but I knew that speaking, publishing and working in emergency medicine were my passions.

I have survived because I have continued to push my self and because I continuous want to learn more. I found my passion and went after it. I have left jobs where it was a toxic work environment and I have worked hard to maintain a happy work-life balance. I’ve recognized when I'm stressed and found outlets for it. When I've become bored or complacent in my work and knowledge I have sought to find something that brings back my spark.

Conclusion
Survival is up to you and how much you want to be a veterinary technician. You may find some mentors along the way, but ultimately it’s up to you. Only you can decide if you want to survive and if you do, then go get what you want out of the profession. Your happiness as a veterinary technician is obtainable.