Complications of Complicated Surgical Extractions
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Veterinary dentistry is unlike other fields of veterinary medicine in that the majority of pathology may not be able to be diagnosed, or even evaluated properly, until the patient is under anesthesia. Many clients are unaware of the status (or sometimes even presence!) of their pet’s teeth until there is overt pathology present. At that time, many dogs and cats present to their veterinarian for ‘just a dental cleaning’ and their owners are shocked to find surgery is needed to treat their pet’s oral disease, and that much more than a professional dental cleaning is necessary. The pet is typically middle-aged, has not had a professional dental cleaning before, does not receive oral homecare, and the owners are noticing oral malodor, spontaneous exfoliation of teeth, draining tracts, pain when eating, dysphagia etc. Thankfully oral disease in pets is at a much higher degree of awareness than it has been in the past, and we as a profession are trying to achieve actual preventive procedures (true prophylaxis) in veterinary dentistry.

This lecture focuses on surgical extractions – the ultimate, final treatment for periodontal disease, endodontic disease, tooth resorption etc. We will also discuss some alternatives to extraction and give examples. Like any surgical procedure, surgical extraction of teeth has potential complications. Factors that contribute to complications are degree of pathology present, breed-specific anatomy, age of the pet, concurrent trauma, concurrent neoplasia or previous treatment/radiation of the area and of course compliance (specifically non-compliance by both the pet and the owner).

Complications include: fractured roots, fractured jaw, retained tooth roots, osteomyelitis, orbital trauma, oronasal fistula formation, hemorrhage, and damage to permanent tooth buds, among others. There are several book chapters that cover potential complications of extractions (please see below for additional reading).

The clinician should be comfortable performing routine surgical extractions on a cadaver prior to performing them in a live patient. There are many wetlabs offered around the country that can aid in attainment of proficiency in surgical extractions. Several key tenets important to successful surgical extractions are: proper preparation of the tooth which involves proper exposure, alveolectomy and sectioning (where applicable). Additionally, correct materials (sharp instruments, correct size carbide or cutting burs on a high speed handpiece, etc.) and controlled, carefully placed force are essential. Patience (above all else!) and tension-free closure as well as postoperative compliance (both client and patient) are also vital.

This lecture is presented as Morbidity & Mortality Rounds, in which select surgical cases are presented with associated complications. Each case is discussed and then recommendations and learning points are given.

Suggested additional reading
There are many oral diseases unique to the pediatric patient. Topics that will be covered include persistent deciduous teeth, malocclusion, fractured teeth, cleft lip and palate, hypodontia and oligodontia, impacted teeth, neoplasia and others.

Prior to treatment of any oral disease(s) diagnosed on conscious oral examination, the puppy or kitten should have a complete general physical examination as well as attainment and review of full medical history; if medical history is unavailable, a detailed history of the puppy or kitten’s behaviors/appetite/attitude/energy level etc. should be obtained from the owner. Breed predilection for congenital diseases should be understood by the clinician and any pertinent history or examination findings pertaining to them should be addressed. Necessary wellness and prevention treatments should be performed. Preanesthetic bloodwork (complete blood count, serum chemistry profile) should also be performed.

In order to understand abnormal oral findings, the clinician must first understand normal. Below is the dental formula for dogs and cats, both deciduous and permanent dentition. In addition, the normal eruption times for dogs and cats, deciduous and permanent dentition are provided.

**Dental formula**

<table>
<thead>
<tr>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canine</td>
<td>Feline</td>
</tr>
<tr>
<td>Deciduous (primary, baby)</td>
<td>2x 3/3, 1/1, 3/3, 0/0 = 28</td>
</tr>
<tr>
<td>Adult (permanent)</td>
<td>2x 3/3, 1/1, 4/4, 2/3 = 42</td>
</tr>
</tbody>
</table>

**Eruption times**

<table>
<thead>
<tr>
<th></th>
<th>Deciduous</th>
<th>Permanent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incisors</td>
<td>3 - 5 weeks</td>
<td>3 - 5 months</td>
</tr>
<tr>
<td>Canines</td>
<td>3 - 6 weeks</td>
<td>4 - 6 months</td>
</tr>
<tr>
<td>Premolars</td>
<td>3 - 10 weeks</td>
<td>4 - 6 months</td>
</tr>
<tr>
<td>Molars</td>
<td>NONE</td>
<td>4 - 7 months</td>
</tr>
</tbody>
</table>

*Eruption times are approximate, ranges are provided due to considerable breed and individual variation.

The crowns of the teeth should be smooth, white, translucent and similar in color to adjacent dentition. The gingiva should be light pink, have a ‘knife-edge’ gingival margin and may have stippling in areas. The mucosa should be light pink or should have normal dark pigmentation. The mucosa should be flexible, malleable, smooth and soft.

**Persistent deciduous teeth**

There should only be one tooth per position in the mouth; that is, there should never be two teeth of the same type in the same place at the same time. A deciduous tooth is persistent as soon as the permanent tooth begins to erupt into the mouth. A deciduous tooth can be persistent secondary to an incorrect eruption path of the permanent tooth (most commonly) or a missing permanent successor. This may be genetic in origin. The most common breeds with persistent deciduous teeth are small and toy breed dogs, but persistent deciduous teeth can occur in any dog breed as well as cats. Persistent deciduous teeth can cause malocclusion and predispose to periodontal disease, thus extraction of the persistent deciduous teeth is recommended as soon as possible. Extraction of persistent deciduous canine teeth can be performed closed (using a ‘blade technique’ which will be discussed in lecture) or surgically depending on clinician preference.

**Malocclusion**

Every animal is entitled to a comfortable, functional occlusion. Normal occlusion involves several different indices. The first is the incisor teeth: the mandibular incisor teeth are positioned caudal to the corresponding maxillary incisor teeth, and the cusps of the
mandibular incisor teeth rest on the enamel cingulum of the maxillary incisor teeth. The mandibular canine teeth occlude in the maxillary diastema equidistant between the maxillary canine tooth and third incisor tooth. The maxillary and mandibular premolar teeth interdigitate in a ‘pinking shear’ relationship in which the mandibular premolar tooth is located equidistant between the maxillary premolar teeth of the same and previous numbers; the mandibular premolars are lingual to the maxillary premolars. Finally, the carnassial teeth occlude such that the maxillary fourth premolar tooth is buccal to the space between the mandibular fourth premolar and first molar teeth. Deviation from these normal positions is a malocclusion. A class I malocclusion is when there is malposition of one or more teeth, but the maxillary and mandibular dental arches are in a normal relationship. A class II malocclusion is mandibular distoclusion. A class III malocclusion is mandibular mesioclusion. A class IV malocclusion is maxillomandibular asymmetry, which can occur in a rostrocaudal, side-to-side or dorsoventral direction.

Is a malocclusion genetic? Potentially. Jaw length malocclusions are considered genetic and class I malocclusions are typically considered nongenetic (with the exception of mesioverted maxillary canine teeth in Shetland Sheepdogs and Persian cats). The maxillae and mandibles grow independently and can have growth surges resulting in mild, temporary disproportionate relationships. A very mild malocclusion may represent one of these growth surges, however a more moderate to severe malocclusion likely requires intervention.

Selective extraction of deciduous teeth to relieve a dental interlock or traumatic occlusion is called interceptive orthodontics. This should be performed as soon as possible, ideally as soon as the malocclusion is diagnosed. Relieving the malocclusion can help the animal achieve its full genetic potential while also providing comfort and relief from traumatic occlusion.

Treatment of various permanent tooth malocclusions are discussed in the lecture and include orthodontic movement using incline planes to treat linguoverted mandibular canine teeth (‘base narrow’ mandibular canine teeth), and crown reduction/vital pulp therapy of linguoverted mandibular canine teeth. Less severely linguoverted mandibular canine teeth may be amenable to gingival wedge resection and/or crown extension. It is not uncommon for linguoverted mandibular canine teeth to occur simultaneously with mesioverted maxillary canine teeth and orthodontic movement of these teeth is discussed using a combination of masel chains and incline planes. Malocclusion treatment includes a thorough discussion with the owners explaining each procedure option, any complications, follow-up/monitoring and prognosis. The potential for genetic heritability should be discussed, neutering should be encouraged, breeding of the pet should be discouraged and the goal of comfort and function should be stressed.

**Fractured teeth**

Fractured deciduous canine teeth (and incisor or premolar teeth) with pulp exposure should be extracted as soon as possible. Complicated crown fractured (pulp exposed) immature permanent teeth can be treated with vital pulp therapy if the tooth is treated within 48 hours; teeth with pulp exposure of > 48 hours have been shown to have decreased success rates. Vital pulp therapy is usually performed on young, immature permanent teeth (patients < 18-24 months of age typically), as these teeth are poor candidates for root canal therapy due to their thin dentinal walls and wide pulp cavities. Vital pulp therapy involves the removal of the coronal-most portion of the exposed pulp and placement of materials to promote healing of the pulp and restoration of the crown. Teeth treated with vital pulp therapy require periodic radiographic monitoring for the life of the patient to monitor for vitality (‘success’).

**Cleft lip and palate**

A primary cleft palate involves the lip and palate rostral to the palatine fissures, a secondary cleft palate involves the structures caudal to the palatine fissures (i.e. caudal to the incisive bone) including the hard and soft palates. A primary cleft palate forms if the nasal prominences fail to merge with the maxillary prominences, whereas a secondary cleft palate forms if the palatine processes fail to fuse. Cleft surgery is usually delayed until the pet is 3-4 months of age, which allows for adequate nutrition as well as more tissue available for repair. If aspiration pneumonia is suspected, thoracic radiographs should be obtained and pneumonia treated prior to surgery. Additionally, owners should be counseled about the possible genetic heritability of clefts and breeding of their pet should be strongly discouraged. Surgical repair requires client and patient selection: the best chance of success is with the first surgical procedure, thus postoperative compliance by the client and patient is crucial. The pet will be required to wear an Elizabethan collar and not be allowed to chew on toys or have any objects in its mouth for at least 4 weeks. If the client is unable to ensure this compliance or the pet is unable to be controlled then surgery should be delayed until compliance can be achieved. There are various ways to treat secondary cleft palates and two of the most common are the modified von Langenbeck technique and the overlapping-flap technique for the hard palate, and a double-layer appositional technique for the soft palate. Images from select cases are shared in the lecture.

**Hypodontia and oligodontia**

Hypodontia is the term used to describe the congenital absence of 1 to 5 teeth; oligodontia is the term used to describe congenitally missing 6 or more teeth. Hypodontia is likely a genetic condition, and is common in small dog breeds, brachycephalic dog breeds and Chinese crested dogs. The most commonly missing teeth are the first and second premolars, incisors and mandibular third molar.
It is very uncommon to be missing a canine tooth or carnassial tooth – thus radiographic confirmation of missing teeth is essential!

**Impacted teeth**

Unerupted teeth are teeth that are either impacted (have a physical obstruction to eruption) or embedded (lack sufficient eruption force).\(^9\) The most common unerupted teeth in dogs are the mandibular first premolar teeth and the maxillary and mandibular canine teeth.\(^10\) Unerupted teeth should be extracted due to the potential for dentigerous cyst formation.\(^9,10\) If a cyst is present, then enucleation of the cyst and histopathology of the cystic lining should be performed; histopathology confirms dentigerous cyst formation and rules out more sinister disease processes, as malignant transformation has been reported previously.\(^9,10\)

**Neoplasia**

Oral tumors can be characterized as odontogenic or nonodontogenic. Odontogenic tumors arise from remnants of embryonic tissues; the vast majority of these tumors are benign, malignant odontogenic tumors are rare.\(^11\) Examples of odontogenic tumors are odontoma, peripheral odontogenic fibroma (previously called ‘epulis’), ameloblastoma and feline inductive odontogenic tumor. Complete resection of these masses is curative. Some odontogenic tumors, like the ameloblastoma and feline inductive odontogenic tumor, are very locally aggressive and should be completely excised with wide margins as soon as possible. Nonodontogenic tumors seen in pediatric patients include viral-induced papillomas, squamous cell carcinoma and others. Nonodontogenic tumors can be malignant or benign and must be treated accordingly.

**References**

This lecture focuses on the most common feline oral pathology with emphasis on clinically important facts and new research findings pertaining to each disease. It is critical to understand that clinical signs of oral disease may be very vague in the cat. Clinical signs include ptyalism, reluciveness, lethargy, hiding, growling or hissing when attempting to eat, running from the food bowl, weight loss, abnormal chewing/turning the head when eating/bruxism/dysphagia. Not uncommonly the only clinical sign noted is weight loss or oral malodor.

A complete medical history is important to aid in distinguishing primary oral disease from extraoral or metabolic disease. A thorough physical examination and preanesthetic work-up, including a complete blood count, serum chemistry profile, thyroid profile and urinalysis will help the clinician in determining if other diseases may be contributing to the cat’s clinical signs.

The importance of the conscious oral examination cannot be stressed enough – by performing a proper conscious oral examination with the client present, the clinician can identify and show the client overt pathology requiring treatment and thus more accurately plan the financial estimate as well as his or her surgical schedule for treatment. This transparency encourages client acceptance and trust. Of course, not all pathology can be visualized during a conscious oral examination – an anesthetized oral examination and radiographs are required to fully evaluate a cat’s dentition. This should be clearly expressed to the client prior to anesthesia and surgery, as inevitably more pathology will be found during anesthetized examination and radiographs. Several videos are used to demonstrate proper conscious oral examination techniques in the cat.

Common feline oral pathology discussed in this lecture will include periodontal disease, stomatitis, squamous cell carcinoma, eosinophilic granuloma complex and selected trauma cases. Tooth resorption, arguably one of the most common pathologies to occur in a cat’s mouth, is discussed in detail in a different lecture.

**Periodontal Disease**

By the age of 2, 80% of dogs and 70% of cats have some form of periodontal disease. Periodontal disease is inflammation and destruction or loss of the periodontium, which are the supporting structures of the teeth. The periodontium includes the gingiva, alveolar bone, periodontal ligament and cementum. Periodontal disease is described by stage, or degree of attachment loss. Stage 1 disease is gingivitis only, and this stage can be reversed. Stages 2 to 4 involve increasing amounts of alveolar bone loss or periodontal attachment loss: Stage 2 is < 25%, Stage 3 is 25 to 50% and Stage 4 is > 50%. The combination of anesthetized oral examination findings (periodontal probing + measurement of gingival recession which summates to total millimeters of attachment loss) and dental radiographic findings gives the % of alveolar bone loss. It is imperative that the clinician diagnose the stage of periodontal disease for each tooth in the mouth. This diagnosis list can be written under the radiographic findings, on the dental chart, and/or summarized in the medical record for each dental procedure. Following professional dental cleaning, teeth with stage 4 disease are either extracted or receive open root planning and bone grafting (where appropriate). Teeth with stage 3 disease must be critically examined in the feline patient – these teeth potentially can be maintained with open root planning and bone grafting (where appropriate), however frequently they are extracted. Teeth with stage 2 disease can receive closed root planing (if applicable). Periodontal disease is cyclical and can be progressive, however it does not need to progress; the institution of oral homecare (ideally daily tooth brushing), management of patient comorbidities and periodic professional dental cleanings could in theory prevent progression of periodontal disease. The cause of and progression of periodontal disease is unfortunately multifactorial: the combination of plaque bacterial virulence, host response, anatomical factors, maladaptive oral behaviors and systemic comorbidities (DM, stress etc.) among others all contribute to periodontal disease. Discussion with the client should include instructions regarding oral homecare as well as a plan for the next professional dental cleaning – each client/patient relationship is unique and client discussions should be realistic and as clear as possible to avoid miscommunication or unrealistic expectations of periodontal disease prevention.

Oral examination findings of periodontal disease in the feline patient include gingivitis, gingival recession, furcation exposure and mobility of teeth, however alveolar bone expansion (ABE) is also a common pathologic finding. ABE is the correct term used to describe the enlargement and proliferation of bone secondary to periodontal disease. ABE is identified as enlargement (‘swelling’) of bone surrounding the affected teeth, classically the maxillary canine teeth are involved (which have centered and labial expansion) but the mandibular canine teeth (which have mesial expansion +/- incisor teeth involvement) and premolar and molar teeth can also have ABE. ABE is always associated with periodontal disease; tooth resorption is frequently associated and supereruption and tooth extrusion may be noted as well.

**Stomatitis**

Feline stomatitis or feline chronic gingivostomatitis is an idiopathic, chronic, debilitating, aberrant, oral hyperimmune, inappropriate response to bacterial plaque and/or viral infections that occurs in cats. Stomatitis is inflammation and ulceration that extends
beyond the gingiva into the mucosa. Caudal stomatitis or caudal mucositis is the term used to describe inflammation lateral to the palatoglossal folds. Stomatitis can occur without caudal stomatitis. There can be varying degrees of stomatitis and caudal stomatitis, as well as involvement of the palate, tongue and buccal mucosa; specifically the degree of stomatitis may be mild to severe, as well as the degree of caudal stomatitis, and there may be ulceration and/or proliferation of other oral structures as well. The cause of stomatitis is unknown and is likely multifactorial. Stomatitis can occur at any age. There are several key factors to treating stomatitis: the first is correct diagnosis (identification of inflammation extending beyond the gingiva with or without caudal inflammation), the second is treatment by surgical extractions. The standard of care for the treatment of stomatitis is partial or full mouth extraction. A recent literature review succinctly describes the collective response of cats to partial or full mouth extraction: a complete remission rate of about 28% is suggested, a large portion of cats (~60-80%) are significantly improved followed surgical treatment, but may need some medical management postoperatively, however not necessarily long-term, and a refractory stomatitis rate of 6-20% is discussed. These findings are important as older studies typically describe greater percentages of remission rates or do not describe the likely need for some postoperative medical management following full mouth extraction. Logically surgical treatment should be performed as soon as possible to reduce the hyperimmune response and allow cessation of inflammation.

Medical management, following surgical treatment, involves pain management +/- immunomodulatory therapy. Pain management includes opioid medications as well as nonsteroidal anti-inflammatory medication or short-term use of steroid medication. Gabapentin or other adjunctive pain medication and additional supportive treatment (anti-nausea medication, appetite stimulation etc.) can be beneficial. Immunomodulatory therapy is typically used to treat refractory stomatitis. Refractory stomatitis is the group of cats that have little to no improvement following surgical and medical treatment. Neoral (microemulsified cyclosporine), carbon dioxide laser ablation, adipose-derived mesenchymal stem cells and recombinant feline interferon omega are all treatments for refractory stomatitis. A chart illustrating these immunomodulatory treatments is discussed during the lecture.

**Squamous cell carcinoma**

Feline oral squamous cell carcinoma (SCC) is the most common oral malignancy in cats. It unfortunately has a poor prognosis as most cases of SCC are advanced at the time of diagnosis. Feline oral SCC is very locally aggressive and invasive. There is no hallmark presentation of SCC: the lesion may be ulcerated, cavitated, or have a smooth gingival covering. The bone involved may be enlarged or there may be proliferative tissue present with spontaneous exfoliation of adjacent dentition. The mass may be located sublingually (this is the most common location), and the tongue may be firm and fixed with or without mucosal ulceration. Surgical treatment is the treatment of choice, and ideally complete, early, wide excision is performed. It is important for the clinician to remember: if any area is suspicious or does not ‘fit’ the rest of the mouth, then biopsy is always a good idea!

Oral SCC has been described to have a low metastatic rate; the majority of cases, however, are not adequately staged or they succumb to their primary tumor so long-term follow-up is lacking. A more recent study describes a higher, but still low, metastatic rate (31% mandibular lymph nodes, 10% pulmonary). The significance of this likely depends on the ability to control the primary tumor.

Following surgical resection oncology consultation is recommended. Accelerated chemoradiotherapy has been discussed as adjunctive treatment for SCC. Unfortunately SCC has a fast mitotic rate thus traditional radiation has been largely ineffective. An accelerated treatment, i.e. the total treatment is delivered over a shorter amount of time, improves the amount of cells responsive to therapy.

**Eosinophilic granuloma complex (EGC)**

EGC is included in this lecture as a lesion/complex of some frustration to the veterinary practitioner; we are going to focus mostly on the oral manifestation of this disease. EGC can occur in the mouth: as an eosinophilic ulcer (most frequently located on the maxillary labia), on the tongue or on the hard or soft palate. Though the labia is reported to usually not be painful, lesions on the tongue and palate are typically painful and can cause dysphagia and ptyalism. An advanced lesion is shared in this lecture – bleeding from the major palatine artery occurred secondary to erosion which necessitated ligation of this vessel.

There is no feline breed or age predisposition. There is a possible female predisposition but this has not be documented consistently. Cytology and/or histopathology and bacterial culture when needed are essential as they provide definitive diagnosis and rule out malignancy. In the oral cavity, histopathology is typically performed to obtain diagnosis. One study found that all cats (n=16) had cytologic evidence of infection, and that treatment with Clavamox for 21 days caused mean reduction in lesion size of 96.2%. Our current understanding of EGC is that it is a manifestation of feline allergic disease. The cornerstone of treatment is identification of an underling etiology. Insect bite allergies (most commonly flea!), food allergy, atopy, immunosuppression and viral infections have all been associated. Strict ectoparasite control (year-round monthly flea/tick/mite preventative as a parasite control trial) and elimination diet trials are common initial investigative treatments. When these fail, atopic dermatitis is considered most likely. Other differentials include idiopathic EGC or a disorder similar to canine atopic-like dermatitis.
References

There are many questions surrounding the disease process of tooth resorption. The main question is what causes tooth resorption, how do we prevent it and how do we treat it? Additionally, how often should we look for it and does tooth resorption really occur in both cats and dogs? This lecture will focus both on feline and canine tooth resorption.

There are many archaic terms used to describe tooth resorption, and these include cervical lesions, neck lesions and feline odontoclastic resorptive lesions (FORL). The term tooth resorption (TR) is the correct term and should be used when diagnosing these lesions.

In cats, we use the terms type and stage, based on the radiographic image of the tooth, to describe and diagnose tooth resorption.

In dogs, we diagnose the lesions as either external or internal and inflammatory or noninflammatory, based on radiographic findings. This naming system in dogs is similar to that in humans.

It is VERY IMPORTANT to correctly name (diagnosis) the type of tooth resorption – the type determines the treatment. If the lesion is diagnosed properly, then it justifies the treatment you performed. We will cover various radiographic images to help in the diagnosis/naming of each type of tooth resorption, both in cats and dogs.

It is important to note that ideally histopathology would be used to diagnose the exact type and stage of tooth resorption, however in most cases this is not practical in clinical practice and is considered academic. Radiographic findings, combined with clinical findings, are usually sufficient to diagnose most lesions.

Feline tooth resorption (TR)
TR is very common in cats. Literature supports that anywhere from 29 - 67% of feline patients will develop TR at some point in their lives, and the incidence of TR increases with age. TR is painful. TR is also progressive, the resorptive process cannot be stopped. Tooth resorption usually starts with enamel or cementum involvement (or furcation where dentin may be exposed in some cats) and progresses to marked loss of tooth structure. The mandibular third premolar teeth (307, 407, called the ‘sentinel teeth’) are the most common teeth with tooth resorption.

We aren’t sure why tooth resorption occurs. There doesn’t seem to be a relationship with diet, as increased Vitamin D and other diets haven’t been shown to cause tooth resorption. Domestication is also not a cause of tooth resorption, as wild and feral cats have been shown to have tooth resorption. Studies looking at the use of restoratives, bisphosphonates, etc. among other treatments to prevent tooth resorption have failed to prevent progression or new development of TR.

Please note that tooth resorption occurring secondary to endodontic disease [for example apical periodontitis i.e. external inflammatory resorption of the apex of a tooth secondary to a complicated crown fracture (pulp exposure)], internal inflammatory resorption, or finally inflammatory tooth resorption secondary to periodontal disease should not be confused with Types 1 and 2 tooth resorption.

There is a recent article discussing suggested etiology of TR in cats. Based on the article, Type 1 and Type 2 are suggested to both be external resorption and Type 1 is inflammatory resorption, commonly associated with periodontal disease, and Type 2 is noninflammatory, similar to replacement resorption. Thus Type 1 is potentially associated with periodontal disease and Type 2 is truly idiopathic. TR, in particular Type 2, cannot be prevented.

Treatment of feline tooth resorption depends on the type and stage. TR can be difficult to diagnose on conscious oral examination, especially in the early stages of stage 1-2. Full mouth radiographs are recommended during each professional dental cleaning, as anesthetized oral examination and radiographic findings are crucial to the proper diagnosis of TR. Below is the classification of TR, as described on the American Veterinary Dental College website:

- Type 1: focal or multifocal radiolucency, tooth has normal radiopacity and normal periodontal ligament space.
- Type 2: decreased radiopacity of the tooth and narrowing or loss of the periodontal ligament space.
Type 3: both Type 1 and Type 2 present.

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Type 1 teeth are surgically extracted. Type 2 teeth are crown amputated. Type 3 teeth are named by the TR of each root (ex. mesial root is Type 1, it is extracted, distal root is Type 2, it is crown amputated). The Stage of TR describes the progression or extent of the disease, ideally this would be determined microscopically but more realistically it is determined based on radiographic evaluation. Stage 1: enamel or cementum involved. Stage 2: dentin involved. Stage 3: pulp involved. Stage 4 is extensive involvement of tooth structure and it is divided into 3 parts: 4a: both crown and roots equally affected; 4b: crown primarily; 4c: roots primarily. The final stage is Stage 5: only remnants of tooth structure remain and there may be complete gingival covering.

**Canine tooth resorption**

Tooth resorption is very common in dogs. Literature supports an incidence of 43.5 – 53.6% of tooth resorption in dogs. Similar to cats, tooth resorption occurs with higher incidence in older dogs. Tooth resorption also seems to be more common in larger breed dogs.

Tooth resorption in dogs is best classified using a classification or naming system similar to that used in humans. Canine tooth resorption is classified as either inflammatory or noninflammatory and either external or internal. Again, similar to cats, these lesions are named by radiographic findings. Ideally they would be distinguished histologically, but since this is rarely clinically feasible, radiographic findings are typically sufficient.

There are 6 (yes, 6!) types of tooth resorption recognized in dogs. They include: external surface resorption, external replacement resorption, external inflammatory resorption, external cervical root surface resorption, internal inflammatory resorption and internal surface resorption. The most common types of canine tooth resorption are external replacement resorption and external inflammatory resorption. We are going to discuss each type, review typical radiographic appearance of each type and discuss causes and treatment for each.

One of the most common types of TR in dogs is external replacement resorption. This type of tooth resorption has been known as ‘idiopathic canine tooth resorption’ or ‘old dog tooth resorption’. This tooth resorption is noninflammatory and radiographically appears as loss of periodontal ligament and replacement of roots with alveolar bone. External replacement resorption is believed to be the result of necrosis of the periodontal ligament fibers. It is likely progressive. Clinically, teeth with this type of tooth resorption usually appear normal, as the majority of the resorption occurs in the roots, and the dog is asymptomatic. Periodic radiographic monitoring is recommended if there is only radicular (root) involvement and the teeth otherwise appear vital radiographically and clinically. As the resorption progresses the crown eventually/likely will become involved, at this point the dog may become symptomatic. These teeth can then likely be treated with crown amputation (as they seem to be similar to Type 2 tooth resorption in cats) though it must be noted that this treatment in dogs has not been published in the literature yet!

The second most common type of TR in dogs is external inflammatory resorption. This type of resorption is secondary to endodontic (pulp necrosis) disease or periodontal disease or both. Teeth with external inflammatory resorption typically have pulp exposure from crown fracture or have advanced stage periodontal disease. Radiographically the apices have irregular resorption and typically there are concurrent lesions of endodontic origin present (periapical lucency, loss of lamina dura etc). These teeth are painful and require treatment: root canal therapy or extraction are usually performed depending on the cause and tooth involved.

External cervical root surface resorption is the next inflammatory type of tooth resorption. These lesions radiographically appear as a resorptive defect in the cervical portion of the tooth. They have an unknown etiology and are painful. The lesions are progressive and aggressive. They appear to potentially be similar to Type 1 tooth resorption in cats (though this hasn’t been published yet!), and extraction is the treatment.

External surface resorption occurs secondary to a traumatic event. In people the resorption is considered a reversible finding, however in dogs this may be the initial stages of what may progress to external replacement resorption. The lesion is located laterally on a root margin, and the ligament space is recreated in this area. As long as the tooth is vital no treatment is needed, other than periodic radiographic monitoring.

Internal inflammatory resorption occurs secondary to pulpitis or chronic endodontic disease. It is radiographically seen as an oval enlargement in the root canal/pulp cavity usually in the coronal 1/3. Treatment is root canal therapy or extraction.

Internal surface resorption occurs secondary to a traumatic event. It is radiographically seen as an oval enlargement in the root canal/pulp cavity, usually in the apical 1/3. These are areas of active revascularization. No treatment is needed as usually the resorption is transient and self-limiting.

The last type of tooth resorption that must be mentioned is unclassified TR. These are teeth that radiographically do not fit into one of the above described categories. These teeth are likely described as unclassified due to the limitations of conventional radiography in localizing resorption lesions.
Interestingly, there is literature that discusses oral tumors potentially causing tooth resorption. A recent study discusses that oral tumors may cause external inflammatory resorption at teeth at tumor sites and sites distant from the oral tumor. Oral tumors are classified as either odontogenic, meaning they arise from remnants of embryonic tissues, or nonodontogenic, meaning they arise from tissues not associated with development (differentiated cell types typically). The majority of odontogenic tumors are benign, whereas nonodontogenic tumors can be benign or malignant. The recent study showed that dogs with nonodontogenic tumors (most were malignant) had tooth resorption at tumor sites more often than did dogs with benign odontogenic tumors. The most common type of tooth resorption associated with tumor sites was external inflammatory resorption, specifically tumor sites that were nonodontogenic in origin or canine acanthomatous ameloblastoma (which is an aggressive, benign odontogenic tumor). Resorption occurs due to tumor associated inflammation, pressure and ischemia and increased osteoclastic activity. Resorption patterns were also noted to be different at tumor sites with benign tumors vs. malignant tumors. Benign tumors have been reported to have smooth resorption of associated teeth whereas malignant tumors have been reported to have irregular, ‘spike-like’ and more destructive resorption of associated dentition. Additional interesting findings were that dogs with oral tumors of any type were more likely to have tooth resorption at distant sites that control dogs – the tooth resorption was typically external surface resorption and external inflammatory resorption. If the dog had a nonodontogenic tumor the resorption at distant sites was more likely to be external inflammatory resorption. Finally, cats have also been reported to have increased odds for root resorption in teeth at sites distant from oral tumors. This finding is interesting as well, but it must be remembered that tooth resorption in cats is extremely common and that the most common type of oral tumor in cats is squamous cell carcinoma, which is very aggressive. In summary, full mouth radiographs of both canine and feline patients are of invaluable diagnostic worth – the more we critically evaluate the radiographs of our patients, the more pathology we will find (and the better medicine we will practice!). The landmark studies by Verstraete, Kass and Terpak provided our initial insight into just how much pathology we may find – in dogs alone, radiographs of clinically normal teeth yielded incidental or clinically important findings in 41.7% and 27.8% of patients!

References