Oral Anatomy:
The canine has 42 teeth. Of these 8 are considered strategic (4 canines and maxillary and mandibular carnassials (maxillary fourth premolars and mandibular first molars) and 12 aesthetic teeth (incisors). The other teeth are important of course, but these are considered critical.
The feline has 30 teeth. The critical teeth are the same as for the canine. The incisors in this breed are much smaller than the dog and are not generally treated except for extraction.
The important structures of the oral cavity in both species include the teeth, maxillary and mandibular bones, alveolar mucosa, attached gingiva, periodontal ligament, nerves and blood vessels of the teeth.
There are several modes of examination utilized in the oral cavity.

A. Visual
   i. Conscious: Depending on the patient, this can be quite limited or fairly detailed. You may be able to diagnose fractured teeth, caries, orthodontic problems, FORL’s, neoplastic masses, and some periodontal disease. However, this should not be relied upon to evaluate for periodontal disease on a continuing basis, especially in small and toy breeds.
   ii. Anesthetized: This is the only way to properly evaluate all oral pathology. Only when they are properly anesthetized, can the patient be fully examined. A full oral exam obviously should be done during any dental prophylaxis; however it is also a good idea to examine all anesthetized patients for dental pathology.

B. Periodontal probing: Periodontal probing is critical for evaluating periodontal disease. This is used to ferret out camouflaged periodontal disease. Periodontal probes will improve patient care as well as practice income.

C. Dental Radiology: This is becoming more and more common in veterinary dentistry. The amount of information that will be gained is invaluable. Any dental disease or therapy should be backed up with dental radiology. Help with interpretation is available at www.vetdentalrad.com.

D. Histopathology: Biopsy everything in the mouth that you are concerned about. ANYTHING that is abnormal should be biopsied no matter what your clinical judgement tells you. In my experience, approximately 1% of all benign appearing
lesions will actually be neoplastic. This can pertain to lesions you think are nothing or lesions you “know” are bad.

Detailed below are the most common diseases of the oral cavity in dogs and cats, with the exception of periodontal disease which is covered in the next article. This is not an exhaustive list and the reader is directed to texts such as *A color handbook of oral pathology* for more information.

**Malocclusions:** An occlusion that is not standard for the breed. It may be purely cosmetic or result in occlusal trauma.

There are several different potential etiologies for orthodontic problems, which are broken into two categories, genetic and non-genetic.

The most common cause of malocclusions is hereditary. This often results from the line breeding of domestic animals (pets) for a certain size or type of head, or other desired characteristics. In addition, malocclusions can result from the mating of parents with dissimilar jaw sizes causing an imbalance of maxilla and mandible. Stockard explained that malocclusions also result from the degree of expression of the *achondroplasia* gene within a patient. Additional genetic causes include tongue size, lip and cheek tension (or lack thereof), and presence of a cleft palate.

In general, jaw length malocclusions (class II, III, IV) are considered genetic and tooth discrepancies (class I) are considered non-genetic. A notable exception to this is mesioclusion of the maxillary canines seen in Shelties and Persians.

Non-genetic causes of malocclusions include local and systemic influences which may occur before or after birth. Local disturbances include trauma, early or delayed loss of primary teeth, cystic formation, or behavioral issues such as bruxism or abnormal chewing. Systemic disturbances include issues such as severe illness, nutritional disturbances, or endocrine diseases.

These patients often do not show any overt clinical signs other than the jaws or teeth being out of alignment. Depending on the class and severity of the problem, oral trauma may be present and can result in bleeding, oral pain, periodontal disease, traumatic pulpitis, and possibly oronasal fistula. A jaw length discrepancy will reveal an incorrect alignment of the mandible and maxilla. Other presentations can have normal jaw lengths with a tooth or teeth out of alignment.

**Management**

Therapy for malocclusions is relative to type and severity of the disease process. Options include:

- No therapy (if purely cosmetic)
- Extraction of the offending tooth or teeth
- Orthodontic correction using appliances
- Coronal amputation and vital pulp therapy

Remember that many orthodontic problems are hereditary and therefore the owner must receive genetic counseling prior to any orthodontic correction procedure. In addition, strictly cosmetic correction is not in the patient’s best interest. The pain associated with orthodontic adjustment, and the numerous anesthetics required, makes orthodontic therapy a disservice to the otherwise healthy patient. The practitioner should obtain a signed consent form for any orthodontic correction case in a non-altered patient.
Deciduous Malocclusions

In some cases, the patient may be genetically programmed for a normal bite and only temporarily maloccluded. In these cases, the alignment problem is typically mild. These temporary malocclusions occur when the maxilla and mandible grow at varying rates during development due to an independent jaw growth surge. In contrast, severe malocclusions within the deciduous dentition should be considered permanent.

In many cases, the deciduous dentition is trapped by a tooth or the soft tissues on the opposite arcade, which interferes with programmed jaw growth and subsequent self correction. This is called an adverse dental interlock.

Clinical Features

These patients most often come from breeders as these clients are likely to perform oral exams on young animals.

Oral exam will reveal that the mandible and maxilla do not rest in the correct occlusion. Any orthodontic presentation is possible; however class II (overshot), III (undershot), and base narrow are the most common presentations (see individual classes for full description of the malocclusions).

Depending on the class of malocclusion (especially class II and base narrow) palatine/gingival/lip/tooth trauma may occur. One major difference between adult and deciduous malocclusions is the anatomy of the teeth involved. The deciduous teeth (especially canines) are much sharper than the corresponding permanent tooth. Therefore, trauma and pain are more intense initially so are more likely to be clinically evident early in the course of disease. This may result in pain or bleeding as the presenting complaint. However, patients will commonly show no outward signs of distress. Occlusal trauma is traumatic and painful, regardless of the lack of clinical signs, therefore expedient therapy is mandated.

Management

If occlusal trauma is present, extraction of the offending deciduous teeth should be performed as quickly as possible to minimize the trauma and relieve the patient’s discomfort.

Even if there is no current occlusal trauma, selective extraction of the deciduous teeth should be performed to remove the adverse dental interlock and allow jaw movement. This is termed interceptive orthodontics.

Deciduous extractions should be performed as soon as the problem is noted (ideally at 4-8 weeks). This will allow the maximum amount of growth as well as relieve pain as expeditiously as possible.

Deciding which teeth to extract can be difficult. Obviously, any tooth that is creating trauma should be extracted. When performing pure interceptive orthodontics, the simple rule is to extract the teeth on the jaw that needs to grow. However, recent texts recommend extracting any deciduous tooth that is or is likely to become a hindrance to movement, while not extracting teeth that may be creating a favorable dental interlock.

Extractions of deciduous dentition are challenging as the roots are proportionally longer and thinner than permanent dentition. Deciduous tooth extraction must be performed very carefully and gently, with a great amount of patience.

Another reason to take great care during deciduous extractions is to avoid damaging the developing permanent tooth. Some veterinary dentists perform surgical extractions for deciduous canines to decrease the possibility of causing iatrogenic damage. However, others (including this author) prefer simple (closed) extractions for the majority of deciduous extraction cases due to decreased surgical time and trauma. Current literature recommends closed extractions in cases with significant root resorption and a surgical approach when the tooth appears intact.
Root fracture is a common occurrence during extraction attempts. If this occurs, every effort should be made to remove the piece(s). A retained root tip may become infected, or more commonly act as a foreign body and create significant inflammation. There are rarely any clinical signs associated with this, but the patient suffers regardless. Complete root tip removal is even more critical in the case of a malocclusion, as the root tip alone is sufficient to deflect the adult tooth from its normal eruptive path. Retained roots are best extracted utilizing a surgical approach. Dental radiographs should be exposed following extraction, to confirm complete removal of the deciduous tooth as well as document the continued presence and proper condition of the unerupted permanent teeth.

**Persistent deciduous teeth**
Persistent deciduous teeth are very common, especially in small and toy breed dogs. However, they can occur in any breed as well as cats. They create both orthodontic and periodontal problems if not treated promptly. It used to be believed that the persistent deciduous caused the permanent tooth to become maloccluded. Studies have shown, however, that it is the permanent tooth erupting incorrectly that causes the deciduous to be persistent.

It has been reported that orthodontic problems begin within two weeks of the permanent canines starting to erupt. This is due to the deciduous tooth being in the place that the adult wishes to occupy. The periodontal issues occur due to a disruption of the normal maturation of the periodontium. When there is a persistent deciduous tooth, one area of the periodontium is not attaching to the permanent, therefore the periodontal attachment in that location will not be normal. It has been reported that the damage begins within 48 hours of the permanent teeth starting to erupt!
Therefore, the adult tooth does not need to be completely erupted for these problems to occur, and they should be extracted as early as possible, do not wait until six months of age to perform the extractions along with neutering. In fact, we recommend that the owners of breeds prone to retain their teeth be instructed to watch for eruption of the permanent teeth and to present the patient for therapy as soon as this occurs.

**Supernummary teeth**
This occurs when there are extract teeth in the arcade. If there is room for these teeth and they are not causing undue crowding or occlusal trauma, no therapy is necessary. If they result in crowding, selective extractions are recommended to relieve the condition and allow for natural cleaning ability. Some veterinary dentists take this a step further and extract crowded rotated premolars in brachycephalic breeds.

**Fractured teeth**
The two main types of crown fracture seen in veterinary medicine are complicated and uncomplicated. Both types require therapy; however treatment for each is often different. The tooth crown is made up of 3 layers. The innermost layer is the pulp chamber (an extension of the root canal). It is filled with blood vessels and nerves that originate from the maxillary or mandibular artery and nerve. The outermost layer is called enamel. It is 97% inorganic material. It has no sensory ability; however it also has no ability to regenerate if lost. Between the pulp chamber and the enamel is dentin. Dentin makes up the majority of tooth structure in mature patients. Dentin is a living structure in that it has the ability to respond to stresses and has sensory ability. This
sensory ability is due to the fact that there are dentinal tubules which run at right angles to the root canal system ending at the dentinal-enamel junction (DEJ). There are 45,000 tubules per mm\(^2\) in coronal dentin. This means that a defect 1 cm in diameter will result in the exposure of 1,000,000 odontoblasts. The hydrodynamic mechanism of dentin hypersensitivity is the currently accepted explanation for pain associated with dentin exposure. Dentin exposure changes the fluid dynamics within the tubules. This change in fluid velocity is translated into electrical signals by the sensory fibers located within the tubules or subjacent odontoblast layer. These signals result in the sensation of pain (or sensitivity) within the tooth. It is rare for veterinary patients to show this discomfort, but occasionally anorexia will be the presenting complaint. Finally, the exposed dentinal tubules may act as a conduit for bacterial infection of the pulp, thus initiating endodontic disease. Over time, the tooth will respond to this exposure by laying down a layer of reparative dentin. There is no study that documents the time for an effective layer to be placed in veterinary patients. One human study found that reparative dentin is seldom found prior to 30 days following exposure of dentinal tubules and completion of formation is generally around 130 days. It is not known however, if this layer of reparative dentin is effective in decreasing tooth sensitivity.

All teeth with direct pulp exposure (complicated crown fractures) should be treated with endodontic or exodontic therapy; ignoring them is NOT an option. Prior to tooth necrosis, the viable nerve is excruciatingly painful. Following tooth death, the root canal system will act as a bacterial super-highway creating not only local infection, but also a bacteraemia which has been linked to more serious systemic diseases (see the article on periodontal disease for further information). The owners of these patients will be reluctant to pursue therapy as “It does not seem to bother the dog”. Fractured and/or infected teeth do bother the pet and they will act better following therapy. Veterinary patients are known for being stoic, and therefore lack of outward signs of oral pain should not be misinterpreted as a benign state. Therefore, you must be a patient advocate and recommend therapy. Uncomplicated crown fractures are also a very common finding on oral exam, particularly in large breed dogs. These fractures will result in direct dentinal exposure. The exposed dentinal tubules will create significant pain for the patient. The currently accepted means by which this sensitivity is created is via the theory of fluid dynamics. In addition, some of these teeth will become non-vital due to the traumatic incident, pulpal inflammation, or direct pulpal invasion via the dentinal tubules. For these reasons, it is recommended that these teeth be radiographed to ensure vitality. If the teeth are non-vital (evidenced by periapical rarefaction or a widened root canal) endodontic or exodontic therapy is required. If the teeth appear vital, the application of a bonded composite is recommended to decrease sensitivity (please see the article on composite bonding later in the issue for further information).

**“Worn teeth” (Abrasion/Attrition)**

Attrition is tooth loss due to tooth on tooth contact. Abrasion occurs secondary to chewing on foreign objects (tennis balls, rocks, cages, skin/hair). If this occurs rapidly, the pulp chamber may become exposed. In this situation the chamber can be entered and tooth pain/death will occur. Endodontic or exodontic therapy is mandated. If the wear occurs slowly, the body can lay down a layer of reparative dentin. This will protect the tooth and keep it vital. However, note that this is not effective in maintaining tooth vitality in all cases. Therefore, all worn teeth should be radiographed. If there is evidence of tooth death)widened root canal or periapical
rarefaction), endodontic or exodontic therapy is again mandated. If the radiograph is normal, a bonded sealant can be considered.

**Intrinsically stained teeth:** Endodontic disease is also manifested by intrinsic staining. This can appear as pink, purple, yellow, or grey. A study by Hale showed that only 40% of intrinsically stained teeth had radiographic signs of endodontic disease, however 92.7% are non-vital. Non-vital teeth lose their natural defence ability and are often infected via the bloodstream, which is known as anachorisis. Therefore, do not rely on radiographic appearance to determine vitality; all teeth should be definitively treated via root canal therapy or extraction.

**Caries:** True bacterial caries are rare in dogs and almost unheard of in cats. They are most common on the occlusal surface of the upper first molars, but can be seen on any tooth. In addition, the most common breed is a German Shepherd dog. Early lesions can mimic wear, and are best diagnosed by tactile feel of the defect with a sharp explorer. If it is sticky, like wax, it is likely a caries lesion. These lesions can progress into the endodontic system resulting in pain and infection (see fracture teeth above). Treatment options are restoration (composite or amalgam) or crown therapy (+/- endodontic therapy); or extraction.

**Enamel hypocalcification (hypoplasia)**
Enamel is a very thin (<1mm) material on the surface of tooth crowns. It is formed and deposited on the dentin by the enamel forming organ which consists of cells called ameloblasts. Enamel is only formed prior to tooth eruption and cannot be naturally repaired after eruption into the mouth.

Hypoplasia/hypocalcification results from disruption of the normal enamel development. Ameloblasts are very sensitive and minor injuries can result in enamel malformation.

The most common acquired cause of enamel hypocalcification of one or several teeth is trauma to the unerupted tooth. This may be due to any external trauma, but is most often associated with the extraction of a deciduous tooth. In traumatic cases, one or several adjacent teeth may be affected. Additional causes of this pattern are infection or inflammation from a deciduous tooth.

A severe systemic infectious or nutritional problem may also result in improper enamel production. In these cases, most or all of the teeth are affected, but only a small part of the crown, usually a horizontal circumferential strip. Canine distemper was a common cause of this condition in the past. Finally, enamel hypoplasia may result from a hereditary condition known as amelogenesis imperfecta. This condition is created by a decrease in the amount of enamel matrix applied to the teeth during. In these cases, nearly all teeth are involved on all surfaces.

Areas of enamel hypocalcification will generally appear stained a tan to dark brown (rarely black) color, and may appear pitted and rough. The tooth surface is hard however, as opposed to the soft/sticky surface of a caries lesion. The areas of weakened enamel are easily exfoliated which will expose the underlying dentin, resulting in staining.

Dentin exposure will result in significant discomfort for the patient (see uncomplicated crown fractures above).

The roughness of the teeth will also result in increased plaque and calculus retention, which in turn leads to early onset of periodontal disease.
For all of these reasons, prompt therapy of these teeth is critical to the health of the patient. Treatment is aimed at removing sensitivity, avoiding endodontic infection by occluding the dentinal tubules, and smoothing the tooth to decrease plaque accumulation. The most efficient and effective way to accomplish these goals is placement of a bonded composite restoration. If the damage is severe and the client is interested in a permanent correction, crown therapy can be performed. Alternatively, extraction may be performed; however this is not the recommended course of therapy if the root structure is normal with no evidence of endodontic infection.

**Feline Tooth resorption:** TRs are a very common malady. Reports vary as to their incidence, but approximately 60% of cats over 6 years of age have at least one, and those that have one typically have more. These lesions are caused by odontoclasts which are cells that are responsible for the normal remodelling of tooth structure. These cells are activated and do not down regulate, resulting in tooth destruction. There are currently two recognized forms of resorptive lesions, type 1 and type 2. Clinically, they appear very similar, as dental defects that are first noted at the gingival margin. However, advanced cases will show significant tooth destruction and may appear to be a fractured tooth. The best diagnostic tool for differentiating between types is dental radiology. With type 1 lesions, there is no replacement of the lost root structure by bone, whereas with type 2 there is generally marked replacement of the lost tooth structure.

Type 1 TRs are typically associated with inflammation such as L/P stomatitis or periodontal disease. In these cases, it is thought that the soft tissue inflammation has activated the odontoclasts. The inciting cause of class 1 lesions is a cemental defect. Odontoclasts move in and destroy the dentin, leading to secondary enamel loss and a resorption lacuna. The weakened crown will eventually fracture, and in these cases the root canal system stays intact resulting in continued pain and infection for the patient. Type 2 lesions are generally seen in otherwise healthy mouths; however the lesions will create local gingivitis. The etiology of type 2 TRs remains unproven. The two major current theories are abfraction injuries from eating hard food and excess vitamin D in the diet. Type 2 TRs show histological evidence of simultaneous repair of the defect by osteoblasts at the same time that tooth is being resorbed by odontoclasts.

Historically, restoration was a recommended therapy, however due to the progressive nature of the disease; extraction is now the treatment of choice. Extractions can be very difficult in these cases due to tooth weakening and ankylosis. Additionally, in some cases, there is little to no tooth structure remaining. In cases with significant weakening and or ankylosis, performing the extractions via a surgical approach is recommended to speed the procedure and decrease the incidence of fractured and retained roots (see extraction article).

Recently, crown amputation has been suggested as an acceptable treatment option for advanced type 2 lesions as it results in significantly less trauma and faster healing than complete extraction. This procedure, although widely accepted, is still controversial. Most veterinary dentists employ this technique, however in widely varying frequency. Veterinary dentists typically employ this treatment option only when there is significant or complete root replacement by bone. Unfortunately, the majority of general practitioners use this technique far too often. Crown amputation should only be performed on teeth with radiographically confirmed advanced type 2 TRs which show no peri-apical or periodontal bone loss. Crown amputation should not be performed on
teeth with: type 1 TRs, radiographic or clinical evidence of endodontic or periodontal pathology, inflammation, or infection; or in patients with L/P stomatitis. Those practitioners without dental radiology capability SHOULD NOT perform crown amputation. In these cases, the teeth should either be fully extracted or the patient referred to a facility with dental radiology.

Missing teeth
There are several reasons that teeth may be missing. These reasons include: congenitally missing, previously extracted, fractured (or extracted) with retained roots, or impacted. The first two scenarios do not require therapy, where as the latter two may necessitate intervention. Therefore, dental radiographs are indicated in all cases of “missing teeth”.

If dental radiographs reveal retained roots and evidence of inflammation or infection (clinical or radiographic), the teeth should be surgically extracted. If they are “quiet”, the owners should be informed and given the option of having the teeth surgically extracted.

Impacted teeth are defined as any tooth that has not erupted by its normal time. This is generally considered to be the time when the surrounding or contralateral teeth have already erupted. The most common cause of impaction is the presence of an overlying structure that interferes with normal eruption. These structures may be bone, soft tissue, or even tooth/teeth that interfere with the normal eruption path. The most common interference is an area of thick and firm gingiva called an operculum. Impactions occur most commonly in the maxillary cuspid and premolar teeth (especially PM1). They also occur most often in toy and small breeds as well as brachycephalic dogs.

These patients generally have no overt clinical signs other than a missing tooth in a young animal. Alternatively, there may be a persistent deciduous tooth present. On occasion, an unerupted tooth may lead to the development of a dentigerous cyst. The incidence of this is unknown in veterinary medicine; however pathologic changes were noted in 32.9% of cases in one human study. Consequently, the presenting complaint or oral examination finding may be a swelling in the area of a “missing” tooth.

A dentigerous cyst is a fluid filled structure which develops from the enamel forming organ, of an unerupted tooth. Small dentigerous cysts are generally asymptomatic, and often go undiagnosed without dental radiology. If clinical, these cysts will generally be seen as swellings in the area of a missing tooth in a young patient. Dentigerous cysts can become quite large and disfiguring, requiring major surgical correction. In addition, these cysts may become infected, resulting in acute swelling and pain. These cases are often misdiagnosed as abscesses. Finally, dentigerous cysts have reportedly to undergone neoplastic transformation. Dental radiographs are generally diagnostic, revealing a unilocular radiolucent area that is associated with the crown of an unerupted tooth. An aspirate obtained for fluid analysis and cytology will be supportive of a cyst. Definitive diagnosis can be achieved with histopathologic analysis of the cystic lining.

Prognosis for these lesions is excellent if diagnosis and treatment are achieved relatively early in the disease course. Surgical removal of the offending tooth and careful debridement of the cystic lining will prove curative. It is important to avoid leaving any of the cystic lining behind, as this could allow the cyst to reform. Early surgical intervention will result in the least invasive surgery possible.
Oral Neoplasia

The oral cavity is the fourth most common place to encounter neoplastic growths. The most common oral growths are the epulids (fibromatous and ossifying). These are benign overgrowths of the periodontal ligament (harmatomas). These can grow very large, but are not aggressive. Acanthomatous Ameloblastomas (epulids) are locally aggressive. They do not metastasize and are mildly aggressive locally. They respond well to local excision with ½ cm margins and enjoy a 90% control rate with radiation therapy.

Benign tumors are exceedingly rare in cats. By far the most common malignant oral tumor in cats is a squamous cell sarcoma. Fibrosarcomas are a distant second. Both of these tumors are typically seen in older cats, are locally aggressive, and are late to metastasize. The only therapeutic option at this point is early, aggressive surgery (2 cm surgical margins).

The above tumors are also seen in dogs. Their behavior and therapy is similar to cats, however these tumors respond better to radiation therapy in dogs. In dogs, the most common malignant tumor is a melanoma which is typically seen in older dark pigmented dogs. Melanomas are not only locally aggressive; they also metastasizes very early in the course of the disease. A combination of aggressive surgery, radiation therapy, and chemotherapy is the best way to treat this disease process. In addition, a vaccine has been recently released that shows promise as an adjunct therapy for this disease process.

Eosinophilic Granuloma Complex

The true etiology of these conditions is unknown; however a local accumulation of eosinophils is thought to initiate the inflammation and necrosis. The accumulation may result from a local (food) or systemic allergies; although these lesions have been seen in cases where allergic disease has been ruled out. Additional causes include a response to irritation, such as chronic grooming or traumatic malocclusion. There may also be a genetic predisposition.

Indolent Ulcers are the most common oral manifestation, and they will present as brownish-red lesions on the upper lip or around the maxillary canine teeth. Linear granulomas can be single or multiple; the most common sites are the lips, gingiva, palate and tongue. They are generally non-painful, but can become secondarily infected. The typical presentation is a raised, lobulated yellow-pink mass; however, they can also appear ulcerative causing severe damage to the oral mucosa and underlying bone. This may lead to severe periodontal loss, pathologic fractures, or oronasal fistulas.

Histopathology should be performed to confirm the diagnosis. Following confirmation of the diagnosis, a thorough allergy evaluation should be conducted including food trial, flea treatment, +/- allergy testing.

The acute disease process is best treated with systemic corticosteroids; however corticosteroids should NOT be used for long term disease control due to the significant systemic side effects. The typical initial protocol is prednisone 2 mg/kg q 12 hours for 3-4 weeks. Additional options include intralesional triamcinolone (3 mg weekly) or methyl prednisolone injections. Antibiotic therapy is required occasionally to induce remission and/or treat secondary infection. There are also cases that appear to respond to antibiotic therapy alone. Therefore, we initially treat mild cases with antibiotics alone and more severe cases with a combination of antibiotics and corticosteroids.
Many cases remain idiopathic, requiring lifelong therapy; options for this include antibiotics and cyclosporine. Fewer side effects may be expected with cyclosporine in comparison to steroids. Cyclosporine is currently not approved in cats and there are reports of opportunistic fungal and fatal protozoal infections associated with its chronic use. Use the lowest effective dose, and perform regular therapeutic levels and routine blood testing.

Caudal Stomatitis
This is another relatively recent disease process in cats that is frustrating us at present. The best description is a severe immune mediated reaction to dental tissues, but we really don’t know. Some feel that this may actually be a group of disease processes that look the same clinically which is why they can be very frustrating to treat. The history will generally include anorexia, drooling, gagging, and pain during mastication. Physical exam will typically include a thin pet with unkempt fur. The oral exam will reveal severe stomatitis usually over all teeth. The inflammation will most commonly be worse on cheek teeth than canines and incisors. However, faucitis is the key clinical finding. Severe hyperplastic inflammation to the gingiva can result from periodontal disease, however faucitis will not be present. A pre-operative blood panel will generally show a marked elevation in globulins (Polyclonal gammopathy) and total protein. Histopathology is recommended but not required. There have been a few cases with the classic look that were created by another pathology (fungal, Pemphigus). In this case full mouth extractions would be ineffective. Recently bartonella has been implicated as a possible cause of stomatitis. This is due to the high incidence of bartonella in the domestic feline population. Stomatitis is one clinical sign of bartonella infection; however it is not a typical cause. The other major sign is lymphadenopathy. If you see severe lymphadenopathy with stomatitis, consider testing prior to therapy. Most veterinary dentists do not really think that this is the cause of the vast majority of cases. Treatment is zithromax for 21 consecutive days. In multi-cat households the patient must be isolated or all patients treated. The results are questionable at present and therapy is pricey.

Medical Therapy: Most medical therapies will work for a while, however in general resistance will start within a year or less. In addition, most therapies have side effects worse than the disease process in and of itself. In general, medical therapy is very frustrating to the practitioner and client. Corticosteroids are the mainstay of most medical therapy today. It is generally very effective at first and is relatively inexpensive for the client. In my experience, injectable (depomedrol 10 mg IM) is much more effective than oral preparations in my experience. However, they will typically loose effectiveness after a year or so requiring higher and higher doses at shorter increments. This generally results in significant deleterious effects. About 10% of stomatitis cases we treat are already diabetic!!! Antibiotics are safer than steroids but much less effective, especially in long term therapy. They are generally disappointing in their success. Metronidazole and clindamycin are the mainstays of therapy; however Clavamox and amoxicillin can be used as well. Metronidazole may be the antibiotic of choice due to its anti-inflammatory effect. Other immune suppressive such as Imuran, Cytoxan, Gold Salts, Cyclosporine have been used. However, they are all very expensive with numerous adverse side effects (mylosuppression). Cyclosporine is currently the most commonly prescribed immune modulatory drug (other than steroids) for this disease process. However, its chronic use
is very expensive and has been implicated in severe fungal and protozoal infections. Starting dose is 5-10 mg/kg. You need to dose for a trough level of about 500 ng/ml on regular basis. In most dentists opinion it is only really effective AFTER teeth are removed. However, it has shown promise in resistant cases.

Laser therapy is not proven at all, most clients and RDVM’s are very unhappy with the long term results. It is very expensive and short term relief only.

**Surgical Therapy:** Extraction is currently the ONLY effective long term treatment for this disease process in cats. In our experience, the sooner this is done, the better that cats do both post-operatively as well as long term.

For extractions to be successful, the teeth must be COMPLETELY removed. Therefore post-operative radiographic confirmation of complete extraction of the tooth roots is recommended. Following the insurance of complete removal of the teeth, perform aveloplasty to remove the periodontal ligament and smooth rough bony edges. This is typically performed do this with a rough diamond bur.

Studies report a 60% success rate when all teeth caudal to the canines are extracted, however our experience has not been as good. However, whole mouth extractions have a success rate of approximately **90-95%** for clinical remission. Slight faucitis may remain, but pets are comfortable. In addition, the rare cases that don’t completely respond are generally much more responsive to medical therapy.

If there is NO inflammation to the canines or incisors (which is rare), then the owner is given the option of leaving the canines. However, if these are inflamed, all teeth should be extracted. If the teeth are ankylosed, complete root pulverization may be necessary. 60-70% of the strength for the rostral mandible is contained in the canine tooth roots. Extracting both mandibular canines at the same time has resulted in jaw fracture on occasion. Therefore, consider extracting all but 2 ipsilateral canines on the first visit. Often this will be sufficient and cats will never have the last two teeth extracted.

Why leave a maxillary canine if the goal is to decrease the inflammation? This is because about 1/3 of cats who have maxillary canines extracted surgically will develop lip trauma from the mandibular canine. In many instances, this has necessitated further therapy in a cat where the stomatitis was resolved.