THE MANY FACES OF ORAL DISEASE
"It's What the Vet Doesn't Know Which Will Bite Them"

Ira R. Luskin Dr. med.vet. Fellow AVD, Diplomate AVDC /EVDC
Animal Dental Centers of Annapolis, Columbia, Towson Maryland & York Pennsylvania
Animal Dental Training Center of Baltimore Maryland

Veterinary dentistry comprises a substantial portion of small animal private practice activity. Since greater than 75% of small animals present with oral disease, its recognition and appropriate treatment can make a substantial difference in the health and longevity of the pet and the client's satisfaction. Over the past several years, research has indicated that maintenance of oral health decreases the incidence of systemic disease and this can be of major economic consequence to the pet owner. Our clients through their own dental problems are very aware of their pet's oral needs and expect from their veterinary team appropriate professional dental advice and oral therapeutic solutions.

Understanding normal oral anatomy helps in the identification of pathological conditions and guides us in the therapy and to formulate a prognosis. The key to solving the diagnostic puzzle often relies on a good oral exam and evaluating all the oral cavity structures and comparing to the contralateral side..

CHRONIC GINGIVO STOMATITIS

In the past this condition has gone by many names most commonly addressed as LPGS (Lymphocytic Plasmacytic Gingivitis Stomatitis. It is presently called Chronic GingivoStomatitis (CGS) This condition routinely affects specific areas of the oral cavity and therefore it's naming is where the lesions occur in the oral geography: **Alveolar mucositis** or the mucosa overlying the alveolar process and extending from the mucogingival junction without obvious demarcation to the vestibular sulcus and the floor of the mouth. **Sublingual mucositis** involves inflammation of the mucosa on the floor of the mouth. **Labial /buccal mucositis**; involves inflammation of the lip/cheek. **Caudal mucositis** which involves the mucosa of the caudal oral cavity, bordered medially by the palatoglossal folds and fauces and dorsally the the hard and soft palate **Glossitis** involves the dorsal and /or ventral tongue surface , **Cheilitis** involves the mucosa of the lip and mucocutaneous junction. Stomatitis is inflammation of the mucosal lining of any of the structures in the mouth.

The etiology is currently unknown. Calicivirus, FIV, FeLV might be modifying agents more than causative ones. Often Calicivirus is seen with animals exhibiting CGS signs. It is unclear Therefore there are no tests or specific signs. Lymphocytes and plasmacytes are seen in all chronically inflamed tissue. Biopsies have limited value since 90% of feline gingival tissue contains identical histologic lesions: epithelial hyperplasia alternating with ulceration and intense subepithelial bands of plasma cells are intermingled with smaller number of neutrophils. Biochemical profiles rarely shows more than a hypergammalobulinemia indicating more chronicity than an actual pathologic entity.

In the treatment of this disease it is first important to differentiate it from the more common entities of gingivitis and periodontitis. The misconception here is that all oral inflammation has a tendency to be classified as CGS or will develop into CGS. Therefore gingivitis and periodontitis must be separated from the entity of mucositis. Characteristic of the first two entities is

inflammation of the free and attached gingiva, bone recession and/or the presence of tooth resorption. After a thorough cleaning, dental radiographs are performed. Depending on the extent of gingival recession and inflammation often the affected teeth need to be extracted and in addition, the opposing teeth on the ipsilateral quadrant should be extracted. The reasoning behind this is that these remaining teeth do not have the mechanical ability to rid themselves of calculus accumulation which will only speed up the process of plaque and calculus accumulation. Plaque will form within 14 hours after any dental cleaning. In order to properly control gingivitis and periodontal disease it is necessary to put the symptoms into remission and to establish proper home care to avoid further loss of teeth and patient suffering.

Unlike gingivitis which solely affects the free gingival margin and the gingiva, CGS (Chronic Gingivitis Stomatitis) affects the mucosa. Animals that are affected often present with acute symptoms: Most notable is severe halitosis, ptyalism, facial shyness, oral bleeding, weight loss, vocalization.

If mucositis affecting any area of the mouth is seen, the first step is to recognize it as such. Do not start antibiotics and steroids before a thorough oral evaluation and radiographs. The client must be informed that if any medications are initially used that this will only be a short benefit and will only delay the appropriate treatment.

It is this authors contention that once an animal is identified with CGS the earlier that a total mouth extraction with ridge reduction is performed the less likely the cat will have residual post operative inflammation. This is often encountered when surgical therapy is delayed for months or years and the animal is overtreated with antibiotics and steroids.

Refractory stomatitis often is seen in long term steroidal use. Usually there is some resolution of the lesions but often focal areas of inflammation remain. The clinician at this time has a few options available for treatment:

Depending on the associated symptoms of pain and dysphagia, the cats can be started on a low dose of prednisone (5 mg/cat) given daily for a week and than tapered down to every other day for 2 weeks. During this time the cat should be started on an esterified fatty acid like 1-TDC (TetraDeconol Complex) This is a monounsaturated fatty acid which can prevent gingival inflammation by blocking the inflammatory cascade. It has a high affinity to white blood cells and therefore is seen at the site of inflammatory tissue. It is used in both animals and people with periodontal disease and arthritis. It's action is against the leukotrene B4 (LTB4) which with prostaglandin E2 (PGE2) and inflammatory cytokines are part of the the Arachidonic Acid (AA) metabolism responsible for inflammatory changes of periodontal disease. The 1-TDC reduces chronic inflammation by reducing the release of the LTB4from stimulated neutrophils and interleukin 1 from monocytes. It inhibits inflammatory cell infiltration and osteoclastic activity. It has it's greatest affect when applied directly to the gingiva. Alternatively it will be absorbed transcutaneously in the ear pinna.

In severe refractory cases, after starting the cat on oral prednisolone, concomitant use of a cyclosporine which inhibits T cell activation can be started. Depending on the type of cyclosporine, Novartis's product "Neoral" a microemulsion can be started at 2.5 mg /kg b.i.d.. Alternatively Atopica which requires a higher dosing of 7.5mg-10 mg /kg s.i.d. (every 24 hours) can be used. It is important that after one month that a trough level (just before the next dosing of the medication) is drawn. Preferable a value of ~400ng/ml needs to be achieved in order to be therapeutic. Often the inflammation is still present but the animal is more comfortable in its eating and behavior. It is this author's experience that Cyclosporine will need to be used 6-8

months before the dose can be tapered. After the first month the prednisone is tapered and than discontinued.

TOOTH RESORPTION AND CROWN AMPUTATION

Tooth resorption in both dogs and cats is very common. In cats it is postulated to affect anywhere from 50 to 65% of the adult population. In it's early stages the lesions are extremely painful. The disease in the past has gone under many names: "Cat Cavities", "Cervical Neck Lesions" 'FORL's or Feline Odontoclastic Resorptive Lesions". These were an attempt to classify often what the lesions appeared as or where on the tooth they were present. Since often these lesions start at the "neck" of the tooth between the crown and the root the word cervical neck lesions were used. The fact that the teeth had holes in them which often were filled with granulation like tissue the word "cavities" were used. The reality is that there are different radiographic presentations. Often the tooth defect starts subgingivally and extends to the periodontal sulcus. But on the other hand when dealing with the canine teeth one can see radiographic changes that affect the roots and the periodontal spaces only. The term cavity is inappropriate since there isn't evidence of carious or decay lesions. Histologically what is evident is the presence of Odontoclasts. These are cells that normally reside in the periodontal spaces. Their activation and the subsequent tooth substance removal are unclear. It would appear that the lesions therefore originate in the cementum and invade into the dentin. From there the lesion progresses either down (apically) or up (coronally) The enamel subsequently chips off exposing the underlying dentin.

Classifications

The clinical classifications of tooth resorption is based on both the gross appearance and radiographic characteristics of the lesions.

Type 1 tooth resorption is often characterized by gingivitis or periodontal gingival and bone recession. It will start at the cervical region of the tooth at the intersect of the enamel with cementum. Commonly seen are defects or cavity like lesions in the crown with gingival tissue growing into the teeth. Radiographs will indicate changes of tooth density in both the crowns and roots. Very visible is the periodontal space around the roots.

Type 2 tooth resorption is often seen starting apically on the root and working it's way towards the crown of the tooth . The main characteristic is loss of the periodontal space surrounding the root. Replacement bone is seen frequently growing into the area of where the root is resorbing. Type 1 and 2 can be seen often together in an individual multirooted tooth.

Further classification of the tooth resorption is according to the stage or degree of the lesion affecting the tooth substance. The following stages are defined:

Stage 1 (TR1): Mild dental hard tissue loss of either the cementum or cementum and enamel. These on exploring are very shallow defects.

Stage 2 (TR2): Moderate dental hard tissue loss of either the cementum or cementum and enamel. The lesions do not extend into the pulpal cavity.

Stage 3 (TR3): Deep dental hard tissue loss of both the loss of enamel, cementum and dentin as the lesion extends into the pulp cavity and root canal. The tooth integrity is intact.

Stage 4 (TR4): Extensive dental hard tissue loss of all the structures above with the loss of the tooth integrity. This is further subdivided into subcategories.

(TR4a): Both the crown and root are equally affected
(TR4b): Crown is more severely affected than the root
(TR4c): Root is more severely affected than the crown

Stage 5 (TR5): Tooth remnants of dental hard tissue are visible only as irregular radiopacities, and the gingiva covers over where the teeth use to be.

Treatment

It is often helpful to divide the disease into three categories in regards to treatment.

1. Radiographic evidence only of root resorption Type 1 or 2 and stage TR4c with no evidence of crown changes nor pain to the animal does not need to be treated immediately. This is quite common in tooth resorptions seen in the canine teeth. Conservative monitoring with radiographic reevaluation in 6 months is important. Alternatively the teeth can preemptively be extracted.
2. If the tooth clinically is painful but there is minimal evidence radiographically of changes to the hard tissue Type 1 Stage 1 (TR1) the teeth need to be extracted. These are teeth that often have aggravated inflamed gingiva. The most common teeth initially to show this are the mandibular P3's (307,407) The surgical extraction techniques need to be followed which involve: opening an apical reposition flap of gingiva and mucosa, buccal cortical bone reduction over the individual roots with a #2 round bur in a high-speed handpiece, periodontal space widening with a highspeed ¼ round bur, sectioning the multi-rooted teeth and extraction of the tooth individual roots with a winged tip elevator. Closure of the flap is performed.
3. If the tooth is clinically painful and there is radiographic evidence of all stages I-4 but has no associated periodontal disease and there is a type 2 resorption (no evidence of periodontal ligaments) a **Crown Amputation** can be performed. This again is only used with Type 2 resorption not where there is both Type 1 and Type 2 present. In this type of procedure the crown of the teeth and the coronal 2-3 mm of root are removed. This leaves intentionally the resorbing roots behind. The procedure involves a creation of a modified gingival flap exposing all of the crown and the crestal bone. A high-speed #2 bur is used with water coolant and the crown is obliterated and a small portion of the tooth subcrestal is removed. The area is flushed and the flap is closed. Again this is not to be used in cats with periodontal disease or stomatitis but only with teeth that are in the Type 2 stage.

DECIDUOUS TEETH AND MALOCCLUSIONS

The adult teeth normally erupt in a specific relationship to the deciduous teeth. The maxillary and mandibular adult incisors erupt palatal and lingual to their deciduous oounterparts. The maxillary canine erupts mesial to the primary canine and the mandibular canine will erupt lingual to the primary canine. The premolars 2-4 both maxillary and mandibular will erupt palatal and lingual to their deciduous premolars.

Tooth eruption is aided by osteoclasts which resorb alveolar bone allowing a pathway for teeth to erupt. There are many factors which affect the time and the pathway of the eruption process. Among these are: Nutrition, breed genetics, head configuration & trauma which can dramatically affect the position and the timeliness of dental eruption.

Normally the process of primary teeth erupting, resorbing and then adult teeth erupting is a fluid process. However it is critical for the practitioner to evaluate the animal's dentition at defined

junctures in time. Usually at 6-8 weeks when all the deciduous teeth should be present, followed at 12-16 weeks when the adult incisors are erupting and finally at 20 weeks when the adult canines and most of premolar and molars are visible. It is during these times that veterinarians can intervene to effect a more normal tooth eruption pattern by removal of the deciduous teeth or non-essential adult teeth.

The following problems are often encountered in young puppies and kittens:

Delayed eruption of deciduous teeth: This can be caused by the presence of a dense overlying fibrous gingival tissue. In addition the delayed eruption can be caused by traumatic displacement of the deciduous and adult tooth long axis. Although this can affect all breeds the small breeds are more predisposed to the problem. If the practitioner by 8 weeks of age does not see the primary teeth they must alert the owner that there will be an adult tooth eruption problem. The surgical action should be taken around 5 months of age. At this time it would be expected that the completion of the adult incisors has taken place. Depending on how many teeth are affected, the solution is to create an operculectomy or an elliptical hole over the deciduous and adult tooth. If the primary tooth is present radiographically then it should be extracted. If there are multiple teeth that are affected, then an apical reposition flap should be performed. The gingiva along the length of the dental arch is incised and manipulated off the crestal bone both buccally and lingually (palatally) The fibrous tissues and/or deciduous teeth if present are removed prior to loosely repositioning the gingiva on the now partially exposed adult teeth. This will allow the still capable of erupting adult teeth to occur.

Retained or persistent deciduous teeth can block the normal eruption of the adult teeth. As previously mentioned if the primary teeth do not undergo resorption, their roots will serve as guides to the adult tooth eruption. That means in the case of the mandibular canines the adults will erupt more linguoverted and the maxillary adult canines will erupt more mesioverted. In the first instance of linguoversion, the adults will traumatize the mucoperiosteum of the roof of the mouth on eruption. This can lead to an oro-nasal fistula if not corrected. Depending on where the lower canines contact they can cause attrition of the upper canine teeth or worse traumatize them with subsequent death from irreversible pulpitis. If there is persistence of the maxillary deciduous canines, the adults will erupt more mesial (rostral) and narrow the interdental space between the lateral incisor and maxillary canine. With this dimininished space, the mandibular adult canines will be forced lingually thereby traumatizing again the hard palate. Alternatively these malerupted mandibular canines can force the maxillary lateral incisors labial thereby traumatizing the lip.

In addition to adult teeth malerupting, any malerupted mandibular deciduous canines that are linguoverted can create an impediment to forward or rostral mandibular jaw growth. This soft tissue (between the primary canine crown and the mucoperiosteum of the roof of the mouth) interlock, prevents this mandible from growing forwards.

Therapy:

All malpositioned, fractured, or persistent deciduous teeth need to be extracted. There should never be both the deciduous and its permanent replacement occupying the same space at the same time. Leaving a fractured primary canine, leads to ingress of bacteria. This bacterial invasion of the pulp chamber continues apically and potentially can cause an osteomyelitis around the apex of the deciduous tooth. Since the adult tooth bud is close in proximity to the primary tooth apex, it can be damaged by descending infections.

This is especially true when the young animal's are coprophagic and introduce E. Coli bacteria in the feces into the pulp chamber of the fractured crowns.

Extractions of all deciduous teeth should be carefully performed to prevent damage to the adjacent adult teeth. After severing the gingival attachments with a #11 blade, a winged elevator is placed in the appropriate position. This is especially necessary when the adult canine is not visible. The placement of the elevator to extract the maxillary fractured or persistent deciduous canine should be palatal. Stay away from the mesial surface where the adult tooth will erupt. In extracting the mandibular linguoverted deciduous canine, placement of the winged elevator should be caudal (distal) or rostral (mesial) on the tooth to be extracted thereby avoiding the lingual surface of the tooth where the adult mandibular canine will erupt. If the primary teeth fracture it is very important to perform a surgical extraction to remove the root remnant. These roots will guide the adult teeth in incorrect positions therefore they need to be removed. Knowledge of the topographic anatomy prevents any damage to the adult unerupted or partially erupted teeth.