

## Common Oral Pathology in the Dog and Cat

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It has been the author's experience that oral pathology, most notably periodontal disease, is the most common diagnosis in veterinary medicine. A thorough oral evaluation as part of a comprehensive patient history and physical examination is required for the diagnosis of oral disease. This presentation will focus on common pathologies of the oral cavity that are encountered on a daily basis in the general practice setting.

It is vital for the practitioner to have an understanding of normal anatomy of the oral cavity in order to recognize and diagnose the abnormal.

The four tissues of the tooth are enamel, dentin, cementum and pulp. **Enamel** is the hardest tissue of the body, but still very brittle. It is mostly inorganic (96%) and has no regenerative capacity when damaged. Enamel has a thickness of 0.1 – 1.0 mm in dogs and cats and is mostly impermeable. **Dentin** is the second hardest tissue of the body and is more elastic than enamel. Unlike enamel, it has a higher organic component (30%), has a regenerative/repairative capacity and is permeable. There are 30,000 – 40,000 fluid-filled dentin tubules per mm<sup>2</sup> of dentin that communicate between its outer surface and the pulp. **Cementum** is an organic/inorganic material that covers the outer root surface and seals the dentin tubules. It is anchored to surrounding alveolar bone by the periodontal ligament. **Pulp** is the very lifeblood of the tooth that consists of blood vessels, nerve fibers, lymphatics and various cellular components. Access to the pulp is limited to the apical delta of the root apex and possible scattered lateral canals. The pulp has a limited reparative capacity and has the most profound inflammatory response to damage of all the tissues of the tooth.

Pulp has an intimate association with the surrounding dentin, referred to as the **dentin-pulp complex**. Odontoblasts make up the outer cellular component of the pulp and produce different forms of dentin as long as a tooth remains vital. Primary dentin forms during tooth development and secondary dentin after eruption. When dentin is damaged and exposed, odontoblasts form tertiary (or reparative) dentin that is structurally unorganized with few dentin tubules and serves a protective function for the pulp. Each odontoblast has a process that extends into the fluid-filled dentin tubules. Each odontoblastic process has corresponding nerve fibers that course into the tubule. An osmotic gradient exists on the outer surface of dentin when it is damaged and exposed. Any outer stimulant to this exposed surface, whether heat, cold, fluids, carbohydrates, ... can change this osmotic gradient causing fluid shifts within the tubule that tug on the nerve fibers. This stimulation is registered as pain and explains the *hydrodynamic theory of dentin sensitivity*.

The **periodontium** is made up of tissues that surround the tooth. **Gingiva** covers the base of the crown and alveolar bone. It serves primarily a protective role, but is far from just a static shield of tissue. The gingival sulcus represents the initial battlefield between plaque bacteria and

the immune system's response. Its normal depth is 1-3 mm in dogs and < 0.5 mm in the cat. **Alveolar bone** surrounds the root and forms the alveolus (socket). The **periodontal ligament (PDL)** is a multipurpose connective tissue that forms the junction between the root's cementum surface and surrounding bone. The PDL is far more than just connective tissue. It contains nerve fibers, blood vessels and cellular components that give it a dynamic role in periodontal health and disease. **Cementum** is also considered part of the periodontium. See its description above for dental anatomy.

**Periodontal disease** is easily the most common disease in veterinary medicine. As it progresses through stages, attachment loss of the periodontium to the tooth causes local and potentially far reaching negative systemic ramifications.

**Stage 1 periodontal disease** is manifested as gingivitis that progresses to advanced gingivitis with edema and bleeding upon probing. It is the author's opinion that clients should be advised at this stage to have a complete oral health and assessment treatment (COHAT) performed for their pet. Gingivitis represents infection of the gingival tissues, though periodontitis has yet to take place. **Stage 2 periodontal disease** is the onset of periodontitis with infection extending beyond the gingiva. Pocket formation and root exposure begin at this stage with up to 25% of periodontium attachment being lost. **Stage 3 periodontal disease** is advanced periodontitis with 25-50% attachment loss. Furcation exposure begins at this time. **Stage 4 periodontal disease** represents an "end-stage" condition involving >50% attachment loss. Exfoliation of the tooth is often pending at this stage as the immune system is simply trying to rid the body of periodontal infection in the absence of treatment.

The onset of pocket formation in the early stages of periodontal disease should be treated as soon as possible with either closed or open root planing procedures based on pocket depth. Periodontal pockets < 4-6 mm can be treated with closed root planing, while those > 4-6 mm should be treated with more involved open root planing. It's worth noting that pocket depth is relative to the individual patient. A 4 mm pocket in a canine tooth of a 2 kg dog likely has proportionally far more attachment loss than a 6 mm pocket in the same tooth in a 35 kg dog. The "smaller" pocket in the smaller patient should probably be treated more aggressively with open root planing than the deeper pocket in the larger dog, which could likely be treated effectively with closed root planing. **Gingival hyperplasia** is a frequently encountered condition in which "pseudo-pockets" form secondary to gingival growth in a coronal direction. Periodontal disease is the most common cause of local gingival hyperplasia, but certain breeds, such as the Boxer, Doberman, Great Dane and Collie, are predisposed to generalized forms. Medications, such as anti-seizure, calcium channel blockers, and cyclosporine are often implicated in a generalized gingival hyperplasia. Regardless of the cause, pocket reduction is required. Left untreated, pocket formation will often allow extension of periodontal infection into the endodontic system of the tooth, causing a "perio-endo" lesion.

**Feline stomatitis** is a common condition with a histologic lymphoplasmacytic diagnosis. It differs from gingivitis/periodontitis in that caudal oral mucositis is visible. Finding a definitive underlying etiology has been elusive. Treatment most often involves extraction of teeth at least caudal to the canines, but most often requires full mouth extraction. Medical therapy with immunosuppressive agents, such as corticosteroids or cyclosporine, may still be required in 20-40% of cases treated with full mouth extraction.

**Tooth resorption lesions (TR)**, previously known as feline odontoclastic resorptive lesions (FORLs), are frequently encountered in the feline patient and involve noncarious odontoclastic destruction of the tooth. Staging TR is based on the severity of lesion, while the type of TR is based on its location. Typing the resorption is more important clinically. A Type 1 (TR1) involves a focal or multifocal lesion(s) where a clear distinction between root and bone exists. Type 2 (TR2) involves a loss of a clear distinction between the root and surrounding bone. A Type 3 (TR3) lesion involves both TR1 and TR2 lesions. It is very important that a clear diagnosis of the type of TR present be made both on visual exam and radiographically. The entire tooth needs to be extracted when a TR1 lesion is present. When a TR2 lesion is detected in a root(s) of a tooth and involves the majority of the root in the absence of signs of a periapical abscess, the extraction may be performed as crown amputation. This is one of the few clinical situations in veterinary dentistry where intentional root retention is permissible for an extraction procedure.

**Endodontic disease** is frequently encountered and has numerous etiologic pathways. Crown fractures are the most common etiology. **Uncomplicated crown fractures (UCF)** involve fractures that do not expose the pulp, while **complicated crown fractures (CCF)** involve pulp exposure. All crown fractures should be evaluated radiographically. Due to dentin permeability, an UCF will still result in some degree of pulpitis that may be reversible or irreversible. CCFs will always result in irreversible pulpitis unless quickly diagnosed and treated with vital pulp therapy. The veterinary dentist infrequently has the luxury of a prompt diagnosis and/or the option vital pulp therapy in this situation. Irreversible pulpitis will always result in a non-vital (dead) tooth. CCFs and UCFs that result in non-vital teeth should always be treated with either extraction or root canal therapy. Though somewhat controversial, UCFs that do not show radiographic signs of apical periodontitis and are periodontally sound, may be treated with a bonded sealant. These procedures seal exposed dentin tubules, thereby decreasing the chance of bacterial migration to the pulp and decreasing pain by alleviating stimulation of nerve fibers adjacent to odontoblastic processes. If nothing else, a sealant procedure will smooth the roughened fracture surface making it less plaque retentive.

**Intrinsically stained (discolored)** but teeth have been shown to be non-vital 92.7%. Etiologies include blunt trauma, malocclusion and anachoresis, though blood-borne infection is thought to be rare. Regardless of the cause, intrinsically stained teeth are most often non-vital and should be treated with either extraction or root canal therapy. Diagnosis is made by visual exam, radiographs and transillumination.

Dental radiography of non-vital teeth is an indispensable diagnostic tool, but limitations exist. A non-vital tooth may have a wide pulp canal and/or periapical lucency consistent with apical periodontitis. Quite often a non-vital tooth will have a perfectly normal radiograph. A common misconception is that a pulp canal will dilate in response to endodontic disease. Truth be told, the canal simply stops narrowing with endodontic disease. This may or may not be radiographically discernible based on the age of the patient and how long ago the loss of vitality took place.

True carious lesions take place in canine patients, though with less frequency than observed in humans. The maxillary and mandibular molars are most often affected. Caries may lead to irreversible pulpitis and should be treated with restorative procedures if there are no signs of the tooth losing vitality.

Neoplasia is a frequently encountered condition in canine and feline oral cavities. In dogs the oral cavity is the fifth most common site for tumor involvement after skin, mammary, digestive and hemolymphatic systems and represents 5.4% of all tumors. Malignant melanoma (MM) and squamous cell carcinoma (SCC) followed by fibrosarcoma (FS) are the most common malignancies.

In felines, the oral cavity is the third most common site after the hemolymphatic system and skin. Feline oral neoplasms represent 10% of all tumors with 89% of oral tumors being malignant. SCC is the predominant oral malignancy, most often involving the gingiva or the tongue. FS is the second most common malignant neoplasia. Oral melanoma is uncommon in the cat.

The malignancies noted above represent *non-odontogenic tumors*. *Odontogenic tumors* (OT) arise from remnants of embryonic tissues of the tooth forming apparatus. Much confusion has existed regarding the origin, nomenclature and classification of OT. The term “*epulis*” has contributed to this muddled history. A new classification of “*epulide*” growths has been suggested and may clarify clinical situations involving these frequently encountered neoplasms.

The term “epulis” is often applied to any expansile gingival lesion. It is purely descriptive and arose from the Greek term “epi-oulon,” which simply means “on the gum.” It gives us no additional information with regard to the histological or pathological nature of a lesion.

*Canine acanthomatous ameloblastoma* (CAA) replaced “acanthomatous epulis” since it has been recognized as a separate clinical entity without a definitive human equivalent. It is a frequently encountered oral neoplasm and arises from remnants of odontogenic epithelium located in the gingiva (rests of Serres). CAA is locally aggressive and invades bone, making en bloc excision of the tumor with an absolute minimum 1 cm margins the primary treatment modality. Radiation therapy has proven to be effective, but up to 18% of previously irradiated tumor sites may develop malignancies.

Neoplasms previously described as fibromatous and ossifying epulides have been reclassified as *peripheral odontogenic fibromas* (POF). POF is a slow growing neoplasm that is common in the dog and rare in the cat. Treatment options are dictated by the location and size of the tumor. Conservative soft tissue excision may be curative depending on the involvement and post-excision availability of attached gingiva. Incomplete excision will often result in recurrence. En bloc resection of the mass and underlying bone may be needed for definitive treatment in some cases.

Time and space does not allow a full discussion of oral cavity malignancies in the dog and cat. Regardless of the presumptive diagnosis, surgical excision should be preceded with incisional biopsies, dental radiographs and possibly computed tomography. It is a frequent temptation for the veterinary oral surgeon to approach an oral neoplasm with definitive intent at the initial presentation. The author believes it is far better to perform incisional biopsies at the initial presentation. Once a diagnosis is made, surgical excision with definitive intent can be attempted. Excisional biopsy samples are most often indicated to confirm the initial diagnosis and evaluate tissue margins.

“Every animal is entitled to a pain free and functional bite.” The author believes this should be the driving force behind the diagnosis and treatment of all malocclusions in our patients. The early diagnosis and initiation of treatment in the puppy or kitten with a malocclusion will most often extend the list of treatment options.

Base narrow mandibular canines are perhaps the most common malocclusions diagnosed. Numerous different treatment options exist, including coronal composite extensions, acrylic incline planes, gingivoplasty and coronal reduction with pulp caps. Treatment choice is based upon patient age and owner ability to comply with post-operative care.

Mandibular distoclusion and mesioclusion are commonly diagnosed. Treatment is often unneeded, but is based upon the presence of a traumatic occlusion. Are there any teeth impacting other teeth or soft tissues with a traumatic result?

Mesioverted canines (lance) may involve maxillary and/or mandibular canine teeth. The malocclusion is often traumatic and predisposes involved teeth to an early onset of periodontal disease. Orthodontic movement is often the treatment of choice when compared with extraction of a strategically important and vital tooth.

A deciduous tooth is considered persistent when its permanent successor begins eruption into the oral cavity. No two teeth should occupy the space normally occupied by one and will often result in displacement of the permanent tooth and a malocclusion. Extraction of the persistent deciduous tooth is most often indicated.

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