ATTACHMENT LOSS: NOTES ON PERIODONTAL DISEASE

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Periodontal disease is the most common ailment of companion animals. Period. If you're not seeing it in over 80% of your patients, you're not taking enough dental radiographs! **Periodontal disease (PD)** is the pathologic attachment loss caused by dysfunction of the periodontium. Active inflammation and dormant disease are both still PD, but **periodontitis** is characterized by active inflammation of non-gingival tissues (i.e. the periodontal ligament and alveolar bone).

Anatomy of the periodontium in dogs and cats includes gingiva, the periodontal ligament, tooth cementum and alveolar bone. 360 degrees of attached, healthy gingiva is necessary as the tooth's primary defense against plaque. Intact gingiva with normal attachment to the tooth surface keeps the plaque bacteria mostly aerobic and exposed for brushing/chewing and other forms of mechanical removal. The periodontal ligament is comprised of stretchy collagen filaments that keep the tooth in the socket. They stretch in all directions (up, down, diagonally). They serve as shock absorbers, storers of stem cells, and they need to be broken down to successfully extract a tooth. Tooth cementum is the biological velcro that grows on the outside of the root. It needs to be clear of plaque and calculus, but retain its texture, for periodontal ligament fibers to attach. FInally, the alveolar bone needs to form a durable surface to protect the tooth roots. The lamina dura is the white line that is radiographically present in healthy periodontia and describes radiographically the alveolar surface to which the PDL is attached. Alveolar bone should start about 2mm below the cemento-enamel junction (also known as the tooth-root junction). Those 2mm is where the gingiva attaches to the tooth surface and form the sulcus. Remember that maxillary/mandibular bone remodels faster than anywhere else in the body, so changes for better or worse can happen quickly. *Failure of any of these components will cause periodontal disease. Periodontal disease will cause further failure of the periodontium. It's a vicious cycle.*

Causes of periodontal disease are multifactorial: genetics, oral flora, immune response, diet, tooth conformation, jaw conformation, occlusion, oral trauma, chewing activity, lack of daily brushing, changes in oral moisture levels…all of these factors contribute to periodontal health and disease. Most likely a combination of any or all of these, changing over time.

One example of a periodontal disease pathway is associated with plaque retention. Plaque starts as a layer of proteins and salivary products forming a thin film called a pellicle. The pellicle covers teeth within seconds after cleaning and becomes a bacterial breeding ground. It is populated first by mostly aerobic bacteria that can cause superficial inflammation (gingivitis). As the layers of plaque become thicker, growing numbers of anaerobes are present in the deep layers. Gingival inflammation is the individual's response to plaque, and those inflamed tissues become porous and get invaded by bacteria. Once in a relatively oxygen poor environment, anaerobes thrive, releasing LPS and collagenolytic products. Inflammation + bacterial collagenolytic enzymes + time = attachment loss. Anaerobic refuge within areas of attachment loss accelerates pocket formation and deepening.

Other ways that plaque can find its way past the gingival attachment include: incompetent gingiva around teeth create open doors for bacterial invasion, crowded teeth, persistent deciduous teeth, traumatized or missing gingiva, such as gingival clefts. Traumatized teeth can disrupt gingiva or infect the periodontium via the pulp, such as in cases of slab fractures and nonvital teeth (endo-perio lesions).

Defining the stages of periodontal disease takes a little critical thinking and some historical perspective. The American Veterinary Dental College has historically used (and still uses!) a human textbook reference to define the stages of periodontal disease in cats and dogs: *Wolf HF, Rateitschak EM, Rateitschak KH et al. Color atlas of dental medicine: periodontology, 3rd ed. Stuttgart: Georg Thieme Verlag, 2005.*

Stage 0 (PD0): No clinical evidence of gingival inflammation or periodontitis.
Stage 1 (PD1): gingivitis only with no attachment loss.
Stage 2 (PD2): early periodontitis; less than 25% attachment loss (clinical probing or radiographically); stage 1 furcation present**
Stage 3 (PD3): moderate periodontitis; 25-50% attachment loss: stage 2 furcation present**
Stage 4 (PD4): advanced periodontitis; greater than 50% attachment loss; stage 3 furcation present**

**Here's the deal with furcations: Yes, the AVDC does list grade 3 furcations as one of its definitions of grade 4 periodontal disease. This is based on human definitions during the infancy of the AVDC. Human teeth have root furcations at or around the 50% bone loss limit. Therefore, in humans, a tooth that has furcation exposure is already at 50% bone loss, and therefore grade 3 furcations and stage 4 periodontal disease necessarily co-exist. In our canine patients, a root furcation can be present in the 25% of root structure closest to the tooth crown. Therefore, grade 3 furcations can be present with stage 2 periodontal disease and the tooth should not be condemned based solely on the presence of a furcation, with some exceptions (maxillary fourth premolars and molars). Saving these teeth may require periodontal therapy or periodontal surgery such as a tunneling procedure.

Radiographic changes associated with periodontal disease are classified as horizontal vs. vertical bone loss. **Horizontal bone loss** is measured from the biologic height of the alveolar crest (typically 2mm apical to the cemento-enamel junction) toward the apex of the root. It is horizontal bone loss between/around roots or between two teeth. **Vertical bone loss** is NOT between roots or two teeth, it is into the bone down the length of a root. PD0 and PD1 show no radiographic changes. PD2 shows up to 25% bone loss down the length of the affected root. PD3 shows 25-50% bone loss, and PD4 shows more than 50% bone loss.

Clinical changes associated with periodontal disease can be seen, felt, heard and smelled. **Pocket formation** greater than 3-4mm probing depth in dogs or greater than 0.5mm probing depth in cats is measurable only under anesthesia. Horizontal bone loss leads to periodontal pocket formation between soft tissue and tooth root. Vertical bone loss leads to infrabony pocket formation that extends down between the tooth and the walls of infected bone. 1-, 2- or 3-walled pockets refer to how many sides of the infrabony pocket are still bone: 3 sides? 2 sides? 1 side? Remember that 1 side is the root surface. **Gingival clefts** are present when there is complete loss of gingival tissue along a tooth surface, extending all the way up to the mucogingival line. This is NOT a dental death sentence, as these can be treated by a specialist. **Tooth death** can result from periodontal infection that extends down to the apex of a tooth root can infect the pulp tissue of the tooth, leading to discoloration of the tooth crown (pulpitis) and acute dental pain, following by chronic bone pain. **Rhinitis and oronasal communication** result from periodontal infection that erodes the alveolar bone that separates the tooth from the nasal cavity. Inapparent oronasal fistulas and rhinitis can cause clinical signs like sneezing and unilateral, purulent nasal discharge. **Halitosis** is caused by hydrogen sulfide gases released by anaerobic bacteria that necessarily reside in oxygen poor environments, like deep pockets. **Pathologic jaw fractures** result when bone loss exceeds the structural integrity of the mandibles or maxilla. Any minor bump or fall can finish the job and separate the bone. Clinical signs include bruising, lacerations, bone exposure, malocclusions or inability to close the jaws, behavioral changes (head shyness, biting).

What about cats? Some cats don't follow the rules and exhibit inappropriate inflammatory responses to plaque and other normal oral processes, like tooth eruption. **Juvenile (aggressive) gingivitis** is thought to be associated with inflammation that persists following eruption of the permanent (adult) teeth. It can cause gingival enlargement or hyperplasia (which responds typically to gingivoplasty). Juvenile gingivitis can spontaneously regress to worsen to **rapidly progressive periodontitis.** This occurs in young cats with disproportionately advanced PD for their age, and yet inflammation is limited to periodontal structures (loss of bone, PDL and gingiva). Sometimes it can be difficult to distinguish **periodontal disease vs. stomatitis** during a conscious oral exam. Remember that tooth-associated inflammation is typically plaque-responsive inflammation. An exuberant immune-mediated response to plaque in areas that are not periodontium or even touching plaque-retentive surfaces (the teeth themselves) is stomatitis (Feline Chronic Gingivostomatitis or FCGS). If all the inflammation is around the teeth, it could be PD and not stomatitis. Take dental radiographs to look for bone loss! These processes are not mutually exclusive.

In conclusion, there is no dogmatic approach to treating periodontal disease once it has been diagnosed. But knowing what periodontal disease looks like in its early and advanced stages will help you create a plan for how to address your patients' disease. If you're not sure how to interpret your dental radiographs, please send them to a dentist for interpretation help! We are here for you.