Below the gum - that's where we should be

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Periodontal diseases are one of the most common health problems in small animal patients affecting up to 90% of your patient population today! Typically, treatment comes very late in the course of the disease leading to numerous local and systemic complications. With its lack of clear outward clinical signs, its existence remains undiscovered by clients leading to those delays in treatment. Another contributing factor to the delay is the client's perception that oral health is linked to their pet's ability to eat. No doubt you've heard the comment 'Well, he's still eating?" Combine this with our own misunderstandings around dental disease and we have created the perfect storm for treatment to begin once the disease has taken hold.

Periodontal disease defined

Without a true understanding of what periodontal disease is, its contributing factors and initiating cause the veterinary healthcare team are unable to accurately address pet owner's concerns, develop and communicate treatment plans and discuss appropriate home care strategies. That understanding firstly starts with the anatomy, and in particular the Periodontium which is comprised of:

- **Gingiva:** which is divided into *free* gingiva and *attached* gingiva, which is further divided into the gingival epithelium (which is easily irritated, leading to gingivitis), and the junctional epithelium where the gingiva attaches to the tooth – this acts as a barrier to the apical periodontal structures.
- **Cementum:** this is the mineralised portion of the tooth and periodontium being approximately 45–50% mineralised and 50–55% connective tissue that receives its blood supply from the periodontal ligament – its organic tissue is composed primarily of Type 1 collagen - its function is to anchor the periodontal ligament to the tooth.
- **Periodontal ligament:** contains Sharpey's fibres primarily of Type 1 collagen that anchors the tooth to the alveolar bone, is actively involved in periodontal maintenance, and dampens occlusal forces, in effect acts as a shock absorber.
- **Alveolar bone:** the anchor for the Sharpey's fibres of the PDL holding the teeth in the maxillofacial skeleton. It is continuously remodelled by osteoblasts (cells responsible for bone production) and osteoclasts (cells responsible for bone removal) as it distributes occlusal forces as a result of mastication. The actions of these cells are regulated and balanced by the RANK / RANKL / OPG system. However, inflammatory cytokines in periodontitis change the balance and allow induction of osteoclasts and associated bone resorptive activity (Lobprise 2019).

Periodontitis is the *active inflammation* of the periodontium. Its initial *reversible* form is gingivitis which can progress to the chronic irreversible form, periodontitis characterised by alveolar bone resorption ('bone loss') and recession of the attached gingiva, destruction of the periodontal ligament and root cementum ('attachment loss').

Periodontitis is caused by the bacterial biofilm (plaque) and the associated inflammatory response (Lobprise 2019). Plaque is a biofilm – an organic matrix of salivary glycoproteins, oral bacteria, lipids, cellular debris and extracellular polysaccharides which adheres to the tooth surface. It is not a food residue, and it forms more readily during sleep. Periodontitis begins with:

- The accumulation of the pellicle on the crown of the tooth that occurs within seconds of the tooth being cleaned. Within hours gram positive oral bacteria colonise the pellicle and the plaque biofilm is formed – being established within 24 hours and reaching maturity, where anaerobic organisms can be supported, within days.
- Supragingival plaque influences and protects subgingival plaque, reducing oxygen available deeper in the plaque matrix, allowing proliferation of gram-negative anaerobic bacteria, some of which can actively invade

gingival tissues through virulence factors of collagenase and protease production, release endotoxins, impair neutrophils and activate the hosts inflammatory cytokines.
- The host inflammatory response is directly responsible for tissue damage and/or stimulates the osteoclastic bone resorption – substantially contributing to the destruction of the periodontium.

Contributing factors:
- Breed predilection to overcrowding of teeth or malocclusions; thinner gingiva and alveolar bone as in toy breeds; or predilection to aggressive periodontitis.
- Genetics affecting structure size, immune response, organ health and other body systems
- Overall poor health, underlying systemic problems (e.g. diabetes mellitus, immunosuppressive diseases or medications).
- Increasing age.
- Lack of effective homecare, reactive healthcare rather than a focus on prevention and wellness
- Abnormal chewing behaviours and/or dermatological problems that damage periodontium; nutritional components (e.g. deficiencies or excesses).
- Saliva quality and quantity, e.g. reduced salivary flow increases plaque development.
- Local trauma, e.g. malocclusion, bruxism (grinding), or foreign body (e.g. hair) in gingival sulcus.

Mechanical removal and disruption of plaque biofilm both professionally and through toothbrushing is necessary to treat and prevent periodontal disease. (Lobprise 2019).

Dental grading vs. periodontal disease staging

The dental grading system is used to drive a conversation with a pet owner whose pet has been identified as showing indicators that likely point to the pet suffering from periodontal disease (or another oral condition) that requires further investigation and treatment. Indices are used to quantify the findings of plaque attachment (only if disclosed through staining), calculus burden, severity of gingivitis, gingival recession and furcation exposure, diet and existing homecare (or lack thereof). It is non-diagnostic and gives us a scale to provide an estimate of fees in order for the pet owner to budget for the expense. It also provides us an opportunity to educate the client on the importance of dental care for their pet's well-being, and to discuss appropriate homecare recommendations in the long-term management of the patient's oral health.

Staging of periodontal disease differs in that it is the assessment of the extent of pathological lesions in the course of a disease that is likely to be progressive. It also tells us the extent to which periodontal disease affects the individual patient and involves a full radiographic evaluation and periodontal examination under anaesthesia. The level of horizontal or vertical bone loss is quantified along with the measurement of sulcus or pocket depths, gingival inflammation and recession, furcation exposure and tooth mobility. The stages of periodontal disease are described as follows:

Stage 0 periodontal disease (PD0)

- Clean crowns, gum margin is flat, pink and creates a scalloped edge against the teeth with no inflammation. The bone level (on radiographic examination) is just under the bulge of the crown (i.e. no bone loss).
- Clinically normal; gingival inflammation or periodontitis is not clinically evident.

Treatment for stage 0

- Regular professional teeth clean by veterinarian under anaesthesia.
- Homecare – brush all teeth daily.

Stage 1 periodontal disease (PD1)

- Slightly swollen and reddened gums – first sign of pain! Brushing teeth may now be painful – gingivitis only without attachment loss.
- No bone loss on radiographic examination – the height and architecture of the alveolar margin are normal.

Treatment for stage 1

- Regular professional teeth clean by veterinarian under anaesthesia
- Homecare – brush all teeth daily.

Stage 2 periodontal disease (PD2)

- Early periodontitis.
- Gingiva is swollen or you may not see significant swelling or redness.
- There is 0 to 25% bone loss on radiographic examination.
- Less than 25% of attachment loss or, at most, there is a stage 1 furcation involvement in multirooted teeth.
- There are early radiologic signs of periodontitis. The loss of periodontal attachment is less than 25% as measured either by probing of the clinical attachment level, or radiographic determination of the distance of the alveolar margin from the cementoenamel junction relative to the length of the root.

Treatment for state 2 periodontal disease

- Professional dental cleaning by veterinarian under anaesthesia ASAP – this is at the point where further. destruction and loss of bone can be prevented.

Stage 3 periodontal disease (PD3)

- Moderate periodontitis.
- Gingiva may still appear normal to mild inflammation or swelling.
- There is 25–50% of attachment loss as measured either by probing of the clinical attachment level, radiographic determination of the distance of the alveolar margin from the cementoenamel junction relative to the length of the root, or there is a stage 2 furcation involvement in multirooted teeth.

Treatment for stage 3 periodontal disease

- Extraction, or
- Advanced periodontal therapy by a veterinary dental specialist followed by dedicated home care.

Stage 4 periodontal disease (PD4)

- Advanced periodontitis.
- Gingival recession, inflammation, pain.
- There is more than 50% of attachment loss as measured either by probing of the clinical attachment level, or radiographic determination of the distance of the alveolar margin from the cementoenamel junction relative to the length of the root, or there is a stage 3 furcation involvement in multirooted teeth.

Treatment for stage 4 periodontal disease

- Extraction (AVDC 2024).

Subgingival professional cleaning

Now with a thorough understanding of the pathophysiology of periodontal disease it is clear that the *subgingival* cleaning is required in order to perform the proper treatment for this condition (gingivitis) or its management (periodontitis) and subsequent slowing of its progression, along with a more thorough and accurate assessment of the subgingival tissues.

Subgingival scaling is required to remove the plaque biofilm and its endotoxins, any formed calculus and other debris from the sulcus, thereby reducing the incidence of inflammatory response and allowing the gingival tissues an opportunity to heal. If not performed, the patient leaves the clinic un- or under-treated.

Subgingival scaling is performed using either ultrasonic scaler with appropriate tip and amplitude (power settings) or curettage using Curettes (requires specialised training). The manufacturer of your ultrasonic scaler

can provide you with information on the appropriate tips and their correct settings.

Polishing of the tooth's surface *subgingivally* is also required. Within the normal depth of the sulcus (0-3mm in dogs, <1mm in cats) enamel can be found – the cementoenamel junction is further below the junctional epithelium (attached at the base of the sulcus where no attachment loss has occurred). This area of enamel must be polished, as is the crown of the tooth, leaving a smooth surface to inhibit the fast accumulation of the plaque biofilm within the sulcus. When this area of the tooth is *not* polished, a rough surface remains encouraging plaque accumulation and a faster return to the disease state.

The final step in the subgingival cleaning is to flush all remaining prophy paste, calculus and plaque and other debris, and the endotoxins from within the sulcus. This provides improved patient comfort in recovery along with removal of stimulants to the immune system.

Early intervention

Early recognition and intervention is key to successful treatment of periodontal disease. However, advocacy for an oral health wellness program from an early age is pivotal in the prevention of periodontal disease. Far too often do we rely on the statistic that two-year-olds suffer from periodontal disease, then advocating for treatment once the disease is established. A more proactive approach is to identify those patients at highest risk of developing periodontal disease, and to intervene with an educational program and homecare routine at a young age that is aimed at plaque control to give that patient the best outcome for life. It just takes that first step.

References

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