Veterinary Periodontal Disease

Introduction

Periodontal disease is the most common infectious disease of adult dogs. It is a progressive, cyclical inflammatory disease of the supporting structures of the teeth and is the main cause of dental disease and early tooth loss in dogs and cats. It affects over 87% of dogs and 70% of cats over three years of age.

By the end of this chapter you should be able to:

- ✓ Understand the aetiopathogenesis of periodontal disease
- ✓ Know the significance of untreated periodontal disease on both the mouth and body organs
- ✓ Treat and prevent common periodontal problems
- ✓ Understand the different pocket types that may present and select appropriate treatment for them.

Periodontal Tissues

Periodontal tissues include four defined structures: gingiva, cementum, alveolar bone, and the periodontal ligament.

The following landmarks are crucial to the understanding of the support structures of the tooth and the aetiopathogenesis of periodontal disease.

Gingiva

The gingiva is the only one of the four periodontal tissues normally seen in the mouth.

Attached Gingiva

The attached gingiva is keratinised to withstand the stress of ripping and tearing food. It tightly adheres to the underlying connective tissue with rete pegs.

Free Gingiva

The free gingiva surrounds the crown of the tooth.

Cementum

The cementum covers the dentin of the root surface of the tooth. It is histologically similar in structure to bone. It is thicker apically than coronally and is capable of both necrosis and some regeneration by cementoblasts. Both the periodontal ligament and gingiva anchor fibres into the cementum.

Alveolar Bone

The roots are encased in alveolar processes. The most dense bone lines the alveolus, and is occasionally seen radiographically as a white line called the lamina dura. Sight of an uninterrupted lamina dura is interpreted radiographically as a sign of good periodontal health.

Periodontal Ligament

The periodontal ligament is comprised of tough collagen fibre. It is very vascular and contains nerves, which include proprioceptive fibres as well as pain fibres (unlike the pulp).

Mucogingival Junction

The mucogingival junction is the margin between the oral mucosa and the attached gingiva. Its importance in periodontal disease is that its position remains consistent. This makes prognosis and treatment planning of periodontal disease easier.

Gingival Sulcus

The gingival sulcus is invisible to the veterinarian and is the space between the tooth surface and the internal epithelial surface of the free gingiva. The sulcus is bathed in crevicular fluid, which contains the elements of immunity and defence. In cats the depth should be 0.5mm to 1mm and in dogs from 0.5mm to 3mm.

Junctional Epithelium

The junctional epithelium, or epithelial attachment, joins the gingival tissues to the tooth using hemidesmosomes. Its presence is critical to the health of the three underlying periodontal tissues it protects. Consider this tissue to be the gatekeeper. Once it is breached by advancing disease it marks the change from gingivitis - which is reversible – to periodontitis, which is not.

Cemento-enamel Junction

The cemento-enamel junction is the line which demarcates the shiny enamel of the crown and the dull cementum of the root. It also denotes the most apical extent of the junctional epithelium. Under normal circumstances it is not visible to the naked eye.

Disease Process

Periodontal disease consists of cycles of active destruction (periodontitis) and dormancy leading to progressive breakdown of tooth attachment.

1. ***Plaque formation***

   Periodontal disease has a multi-factorial aetiology.

   There is variation in individual susceptibility depending on specific host responses. The primary aetiological agent is plaque, leading initially to gingivitis.

2. ***Deepening of sulcus***

   During active disease it is common for the sulcus to deepen as the junctional epithelium becomes inflamed.

   The tissues become oedematous and infiltrated with polymorphonuclear granulocytes.

3. ***Proliferation of subgingival plaque***

   The accumulation of the supragingival biofilm reduces the oxygen available to the plaque in the sulcus. As a result, there is a transition from aerobic or facultative anaerobic to an overwhelmingly anaerobic microflora within the subgingival area.

   As the junctional epithelium swells from oedema and begins to break down, the gingival sulcus is deepened and the three deepest periodontal tissues - periodontal ligament, alveolar bone and cementum - all risk being exposed to the disease process occurring in the tissues close to them.

4. Toxins lead to tissue damage

This virulent bacterial plaque population and the associated cytotoxin and endotoxin production can often cause direct tissue destruction.

The immune response of the host at this stage is critical. Individuals with a poor immune response will experience tissue destruction, deepening of the pocket and further attachment loss.

5. Continued attachment loss

As the plaque begins to age it mineralises and forms calculus, which adheres mainly to the buccal surfaces of teeth. Whilst calculus in itself does not cause periodontal disease, it can allow plaque to more rapidly colonise the tooth surface.

Also, when calculus is present subgingivally, proper healing is not possible. For this reason diligent removal of all calculus sub and supragingival is a prerequisite in any dental procedure.

6. Tooth loss

The junctional epithelium separates away from the tooth surface and, eventually, a periodontal pocket forms and deepens.

As more effusion takes place into the sulcus, the tissues move away from the tooth surface and the resultant space fills with calculus, necrotic cementum and other debris. When microbes get within 0.5mm of bone it starts to resorb by osteoclastic resorption.

Once approximately half the alveolar bone is lost the tooth becomes mobile and will be lost.

Staging Of Clinical Periodontal Disease

Introduction

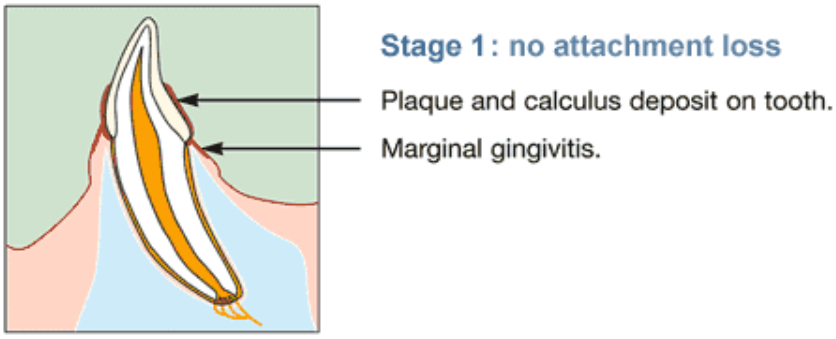
Staging of periodontal disease allows the clinician to determine how far the disease process has progressed for any individual tooth. Any staging refers to the tooth only and not the mouth in general. Assessment of degree of attachment loss can be estimated with a probe, but needs radiographs to confirm accurately.

Stage 0: Normal and Healthy Periodontium

Healthy periodontium should be pink (non-inflamed), except when pigmented, firmly attached to the underlying bone and with a sharp margin where the soft tissues meet the tooth.

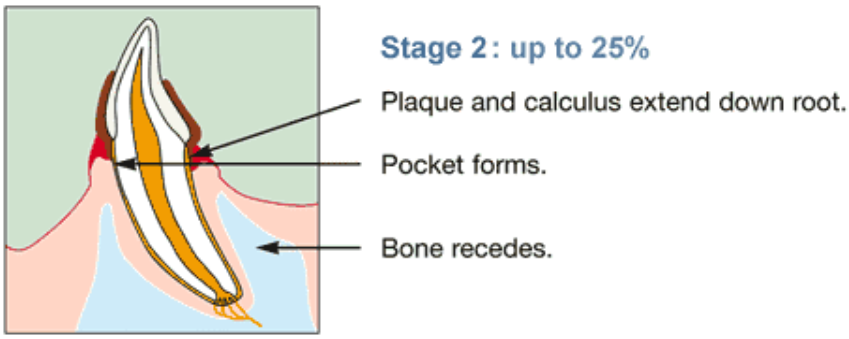
Radiographs should show good bone height to cemento-enamel junction, no pockets and possibly an uninterrupted lamina dura.

Stage 1: Gingivitis



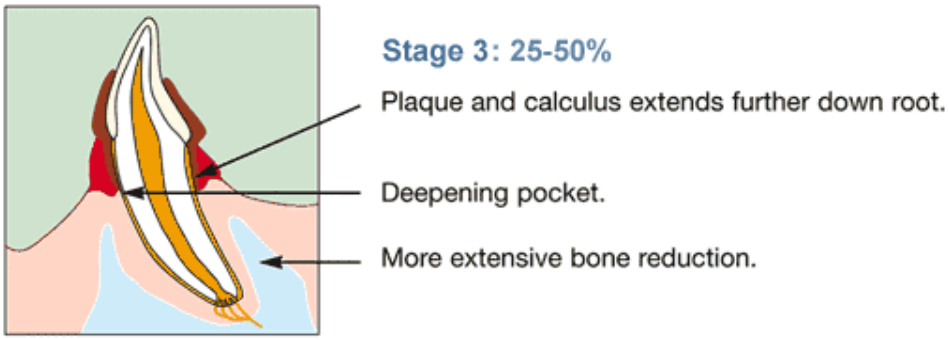
Gingivitis only, due to plaque deposition and lack of home care. Reversible by diligent homecare or professional scaling and polishing. There is no attachment loss at this stage. Without treatment and with an increase in quantity or virulence of bacteria, this will lead to stage 2.

Stage 2: Early Attachment Loss



Early disease defined by attachment loss of up to 25%. Sulcus deepened by disease with periodontal ligament (PL), alveolar bone crest and cementum all exposed to bacteria. These tissues recede away from the infection leading directly to attachment loss.

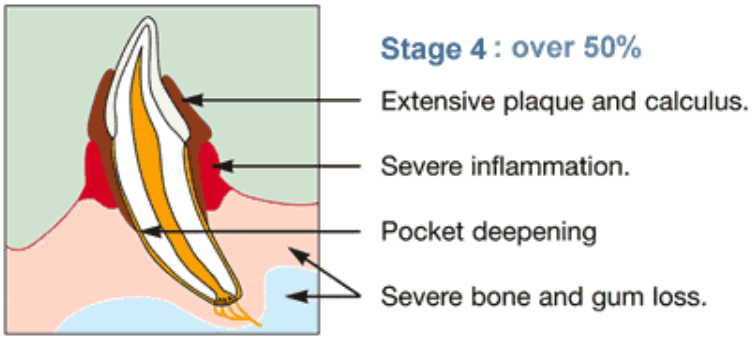
Stage 3: Moderate Attachment Loss



Defined by attachment loss of 25% to 50% verified by probing and radiographs. Note that gingival recession occurring at the same time as deepening pockets may not increase the probing depths. However, attachment loss is still significant.

Stage 4: Severe Attachment Loss

Attachment loss is now greater than 50%. If disease is generalised, horizontal bone loss is most likely, whereas localised lesions, such as the palatal aspect of the maxillary canines, may present infrabony pockets with vertical bone loss.



Predisposing Factors

Introduction

The factors that predispose various individuals to periodontal disease are many and varied.

They fit broadly into three categories:

- The susceptibility of the individual based on the bacterial challenge provided and the defence systems available to fight it
- The anatomical shape of the head and location of individual teeth
- External factors causing damage, such as abnormal chewing behaviour.

Breed

Overcrowding of teeth and malocclusions are more common in small breeds and brachycephalic dogs. This may cause up to 90^{o} rotation of teeth or overlapping of cusps leading to the entrapment of food and other debris. Mouth breathing in brachycephalics can also dry out the oral tissues.

Genetic factors are also responsible for types of periodontal disease in Greyhounds, Schnauzers, Maltese terriers and also Abysinnian and Somali cats.

Chewing Behaviour and Diet

Normal chewing cleans the teeth physiologically through abrasion and “scrub”. With natural diets, the action of the cheek teeth in breaking down tough and fibrous food to swallowable strips can also help to remove plaque.

Feeding soft, textured foods tends to reduce mechanical abrasion especially in the buccal surface of the caudal cheek teeth. Dry foods often offer more abrasion during feeding with a higher salivary flow.

Bored dogs may chew stones, fences and bones resulting in fractured teeth, gingival laceration and periodontal disease. Electric burns may result from chewing cable. Food, foreign body or hair chewing as a result of pruritic problems can lead to impaction in the sulcus, causing local tissue irritation.

Developmental Defects

Retention of deciduous teeth is common and can predispose to periodontal disease – amongst other problems - due to crowding. Micrognathia, cleft palate and polydontia are less common.

Disease

Any febrile disease of young animals, during enamel development of permanent teeth, leads to enamel damage by hypocalcification. The resultant rough surface and exposed dentine attracts plaque rapidly.

Any infection or systemic disease process (Hypothyroidism, FIV/FeLV, FCV, pemphigus, pregnancy, renal disease etc.) can contribute to poor oral cavity health, due to a reduction in the efficiency of the local immune system and/or poor wound repair and tissue regeneration.

Statistically significant relationships have been found between myocardial degeneration, kidney disease (both glomerular and interstital) and liver parenchymal disease.

Pocket Types

Introduction

Pocket development and tissue loss can broadly be classified into one of four categories on the basis of diagnostic probing and radiographs. It is important to be able to distinguish between these categories, as each may have a different prognosis and treatment plan.

Supra-bony Pocket

This pocket is common in dogs and cats. The periodontal tissues migrate apically and probing will reveal an increase in depth from the normal anatomical sulcus.

The entire pocket is coronal to the alveolar crestal bone and within soft tissue. Attachment loss will vary in severity but, in most cases, the pocket will be bound by necrotic cementum on the root surface and chronically inflamed gingiva.

Infra-bony Pocket

Infra-bony pockets are also common in dogs and cats. A pocket will form, as the periodontal tissues become diseased. The apical limit of the pocket is below and within the alveolar crestal bone. This is diagnosed by using the periodontal probe and radiographs.

Pseudo Pocket

The gingiva is abnormal and hyperplastic in the pseudopocket. Probing will reveal a sulcus depth greater than the normal. However, the bulk of the pocket will be above the normal gingival margin, due to migration of hyperplastic tissue coronally. Some of the pocket may also occur apically.

Focal or generalised gingival hyperplasia can occur, due to a chronic inflammatory response to plaque. Some breeds such as Boxers, Setters, Dobermans, Collies and Dalmatians show a familial tendency. Some drugs (e.g. epanutin) can also cause hyperplastic gingiva formation.

Gingival Recession

It is possible for the inflamed gingiva to recede apically. Once the microbes of periodontal disease come within 0.5mm of the bone crest it also recedes and all the tissues move apically in their normal relationship without

forming a pocket. The net result is exposure of the cemento-enamel junction and root surface but typically, no increase in sulcar depth and no pocket.

Periodontal Surgery

Introduction

Once a diagnosis has been made as to the type of pocket present, a treatment plan can be formulated.

In some instances extraction of the tooth will be the best treatment – particularly if attachment loss and mobility is advanced. In more moderate disease, there is a variety of treatment regimes available to the clinician. The importance of diligent and ongoing oral hygiene cannot be too highly stressed. Advanced periodontal treatment should not be performed if oral hygiene will be absent or cursory.

Aims

- To help eliminate pockets or pseudopockets
- To promote re-attachment of the tissues to the tooth
- To enable successful homecare by the owner.

Principles

Destroy the minimum of normal tissue and conserve gingiva, if at all possible.

The location of incisions and sutures are critical to successful flap management. Incisions should be made at the line angle of adjacent teeth. Whether the incision crosses the mucogingival junction will depend on the pocket depth present and the aim of the procedure. If it is not necessary to cross this line do not do so.

Tissue Healing

The soft tissues readily re-attach to the cementum once debridement has taken place. Re-attachment will eliminate the dead space of the pocket **but** this new attachment can be weaker than the original, depending on which of the following four tissue types repopulate the surface of the root most quickly.

Gingival Epithelium

If gingival epithelium attaches most quickly a long junctional epithelium results. This is not desirable, as it is very weak.

Gingival Connective Tissue

When gingival connective tissue connects first, root resorption often occurs.

Alveolar Bone

If alveolar bone cells arrive first, either root resorption or ankylosis with result.

Periodontal Ligament

When periodontal ligament connects most quickly, new attachment can occur. This option is the most desirable.

Materials

Barrier materials, special collars and grafts are used to slow down or exclude gingival epithelium or gingival connective tissue. These allow better access to the slower progenitor cells of the periodontal ligament and/or

the alveolar bone. Since soft tissues are faster to migrate than bone, the hope is that periodontal ligament will develop before any new bone.

Barrier Materials

- Possible barrier materials are made of Gore-Tex™ or Vicryl™
- Osseopromotive materials are used such as BoneGlass® (Consil™: Vetoquinol UK)
- Antibiotic based gel matrix product (Doxirobe™: Pharmacia)
- Chlorhexidine based material (Periochip™).

Bone Grafts

- Autogenous cancellous bone: maximum osteogenic potential. Can be time consuming to harvest
- Allografts of banked bone (e.g. freeze dried irradiated bone). Slower response than autografts, with no direct osteogenesis.

All of these products eliminate the dead-space and encourage new cells to migrate into the pocket, allowing it to heal and eliminate the pocket at the same time.

Indications for Surgery

Surgery is indicated when the pocket formation exceeds the ability of the body to eliminate the sulcar pathogens on a daily basis, to prevent progression of pocket depth.

Notes

- There can be bone loss without pocket formation, where the gingiva follows the bone and maintains the normal anatomy. Surgery is **not** indicated here
- A minimum of 1.5mm to 2mm attached gingiva must remain to protect alveolar bone.

Odontoplasty

Indications

Selective grinding and polishing of morphological anomalies (e.g. slab fractures of the crown) to make them less plaque retentive.

Contraindications

Do not overdo this procedure and remove excessive normal tooth tissue. Take care, as normal enamel in dogs and cats is very thin. Also, do not remove the enamel bulge. The enamel bulge is a protective structure, designed to deflect the food away from the periodontium during eating.

Method

Use 12 bladed flame (or barrel) diamond FG burs or a white stone FG bur with water coolant, followed by polishing with rubber cup and fluoride to reduce sensitivity.

Gingivectomy

Indications

Gingival hyperplasia and/or shallow suprabony pockets that do not extend below the muco-gingival line. At least 2mm attached gingiva should be retained at all times with this procedure.

Contraindications

Infrabony pockets and osseous thickening when attached gingiva is deficient or absent.

Method

1. Determine pocket depth with probe and create a bleeding point on the buccal surface.
2. Join bleeding points with no.15 or no.12 blade, electrosurgery loop on cut/coagulation (fully filtered) mode or a Kirkland knife.
3. Use a bevelled (obtuse) angle **at more than 90°** to the long axis of the tooth to "feather" the gingival margin. It is important to scallop between the teeth to preserve the interdental papilla.
4. Perform a light prophylaxis immediately and again two weeks post surgery.

Simple Flap Procedures

Simple flap procedures include the Simple Apically Repositioned Flap and the Modified Widman (Reverse Bevel) Flap.

Objective

To gain access to the deeper periodontal structures using a flap reflected from the root and alveolar surfaces.

Indications

- Active pockets over 4mm deep which are not responding to initial treatment
- Pockets beyond the mucogingival line with bone loss
- Pockets with marginal deformity.

Contraindications

Gingival enlargement by hyperplasia.

Method 1: Modified Widman

1. Use a reverse bevel incision at approximately 10 degrees to the long axis of the tooth. Incise around the tooth (teeth) approximately 1mm from the gingival margin. Scallop between teeth to preserve the interdental papilla.
2. Make releasing incisions at the line angle (imaginary line of roots) of the adjacent healthy teeth.
3. Reflect full gingival flap with periosteal elevator. A collar of tissue, which includes the sulcus epithelium, should remain attached to the tooth at this time.
4. Remove remaining collar of tissue with a curette.
5. Systematically plane the root and correct osseous defects.
6. Close labial or palatal flaps with interrupted sutures.

Method 2: Apically Repositioned Flap

1. Proceed as above, or via a sulcus incision, without leaving a collar of gingiva attached to the tooth.
2. Make releasing incisions at the line angle (imaginary line of roots) of the adjacent healthy teeth.
3. Reflect full gingival flap with periosteal elevator beyond the mucogingival junction (MGJ).

4. Root plane and curette exposed surface. Any diseased bone is recontoured with a diamond bur in a high speed handpiece or with a Weidelstead chisel.

5. Replace the flap further down the long axis of the root to eliminate pocket depth. This is known as apical repositioning. Note that the MGJ line will not be continuous with the adjacent teeth. The redundant tissue can be left to fibrose naturally. A barrier material can be employed to eliminate pockets within the bone before the flap is replaced.

Sliding Flap

Objective

To cover a root surface denuded by gingival defect or periodontal disease, and widen the zone of attached gingiva. This technique is commonly used to eliminate clefts in gingiva at carnassials or cuspid (canine) teeth.

Method

1. Identify an adjacent healthy donor site.

2. Prepare, clean and scarify the recipient site then create a flap **at least 2.5 times wider** than the defect on an adjacent site.

3. Make a mixed partial thickness/full thickness flap of epithelium and connective tissue. This requires leaving the periosteum on the bone in the area that will be left exposed (i.e. at the donor site).

4. Slide the flap laterally onto recipient site and fix with interrupted sutures (5/0 at 1.5mm intervals) to adjacent gingiva and alveolar mucosa.

5. An absorbable suspensory suture around recipient tooth will prevent the flap from slipping apically.

6. Apply a dressing of Tinct. Myrrh and Benzoin and flush the site twice daily with chlorhexidine (CHX Guard Solution™: St Jon VRx).

Free Gingival Graft

Indications

Gingival recession on single teeth or groups of teeth beyond the muco-gingival line where no donor site is adjacent.

Method

1. Make a template of original defect with tinfoil.

2. Harvest adequate tissue (greater than 2.5 times in width and length) from another site within attached gingiva that has sufficient width (e.g. below LM1 or upper canine).

3. Place graft on a swab saturated with saline.

4. Tack edges with Tissu-Glu™ (Ellman Veterinary) or superglue and suture with 5/0 absorbable suture with swaged cutting needle.

5. Push graft onto periosteum with finger pressure for 2 to 3 minutes to prevent blood clot formation underneath.

6. Apply a dressing of Tinct. Myrrh and Benzoin and flush the site twice daily with chlorhexidine (CHX Guard Solution™: St Jon VRx).

Key Points

- ✓ Periodontal disease affects over 70% of adults cats and 90% of adult dogs.
- ✓ Dental plaque is the main cause of gingivitis.
- ✓ Gingivitis is reversible. If not treated, it may proceed to periodontitis - which is not.
- ✓ Periodontal disease is a progressive condition that proceeds through periods of dormancy and active destruction of tissues – periodontitis.
- ✓ In general, there are four different pocket types that may present. You must be able to select appropriate treatment for each of them.

Further Reading

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