DEVELOPMENTAL DENTAL DISORDERS

Developmental dental disorders may be due to abnormalities in the differentiation of the dental lamina and the tooth germs (anomalies in number, size and shape) or to abnormalities in the for- mation of the dental hard tissues (anomalies in structure).

Anomalies in number, size and shape

Congenitally missing teeth

Congenital absence of teeth is common in the dog. Radiographs are required to determine if teeth missing on clinical examination are actually absent or unerupted. This is often of interest for the owner of a dog meant for the show ring. Absence of teeth can be aninherited abnor mality or can result from disturbances during the initial stages of tooth formation. The primary teeth give rise to the permanent tooth buds, so if there is no primary tooth the permanent counter- part will also be missing. It is possible, however, for the primary tooth to be present and the permanent counterpart absent.

In dogs anodontia (total absence of teeth) and oligodontia (congenital absence of many but not all teeth) are rare and can be associated with ectodermal dysplasia or occur in dogs with no apparent systemic problem or congenital syndrome . Hypodontia (absence of only a few teeth) is, however, a relatively com- mon finding in dogs. It is especially common in purebred and linebred dogs, as the genetic fault will have been perpetuated. In general, missing teeth are of no clinical significance other than that plaque accumulation may be more extensive as the cleaning of teeth associated with chewing is likely to be reduced.

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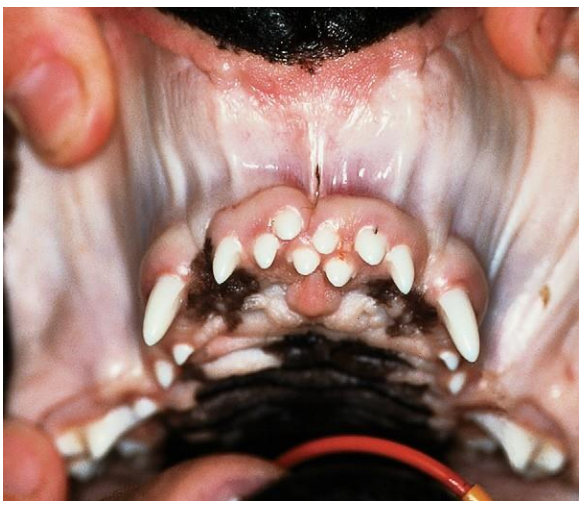
Supernumerary teeth

Supernumerary teeth are common in certain dog breeds. They are the result of either a genetic defect or a disturbance during tooth development. Supernumerary teeth that contribute to mal- occlusion or crowding should be extracted. Radiographic evaluation allows differentiation

between primary and permanent teeth. Primary teeth are smaller than their permanent counter- parts, with long, slender roots.



Supernumerary teeth. Supernumerary teeth commonly cause crowding and malocclusion. In this dog, the supernumerary permanent teeth were involved in an incisor malocclusion, resulting in excessive wear of the mandibular incisor teeth and gingival trauma in the upper incisor arch (radiographs were taken to elucidate whether the supernumerary teeth were primary or permanent). Treatment consisted of extraction of the upper incisor teeth that were grossly out of alignment and had abnormal occlusion with the mandibular incisor teeth.

Anomalies in structure

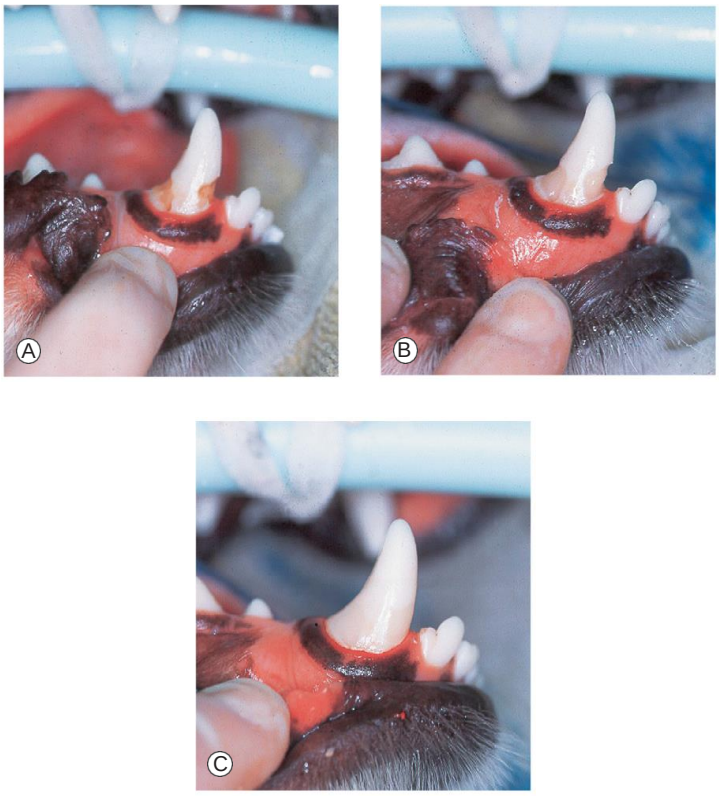
Enamel hypoplasia (dysplasia)

Enamel hypoplasia (dysplasia) may be defined as an incomplete or defective formation of the organic enamel matrix of teeth. The result is defective (soft, porous) enamel. It can be caused by local, systemic or hereditary factors. Depending on the cause, the condition can affect one or only a few teeth (localized form), or all teeth in the dentition (generalized form). It is essential to remember that enamel hypoplasia results only if the injury occurs during the formative stage of enamel development, i.e. during amelogenesis. Thus, the defect occurs before the tooth erupts into the oral cavity. Crown formation lasts from the 42nd day of gestation through to the 15th day postpartum for the primary teeth and from the second week through to the third month post- partum for the permanent teeth of dogs and cats. Depending on the time of the insult, enamel dysplasia will affect primary and/or permanent teeth.

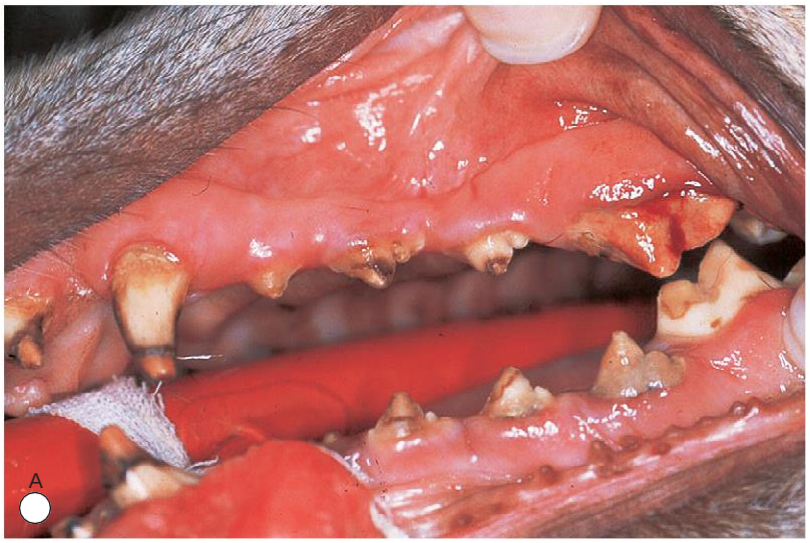
As already mentioned, enamel dysplasia may be caused by local, systemic or hereditary factors. Local factors include trauma to the developing crown, e.g. a blow to the face or an infection. Infection is often a consequence of a bite injury. Periapical disease of a primary tooth may cause enamel dysplasia in adjacent developing permanent teeth. Usually only one or a few teeth are affected. Systemic factors include nutritional deficiencies, febrile disorders, hypocalcemia and excessive intake of fluoride during the period of enamel formation. Usually most teeth are affected. Historically, enamel dysplasia in dogs occurred due to distemper infection. This is rare today as most dogs are vaccinated against distemper. Hereditary types of enamel dysplasia have been described in humans. The incidence in cats and dogs is unknown.

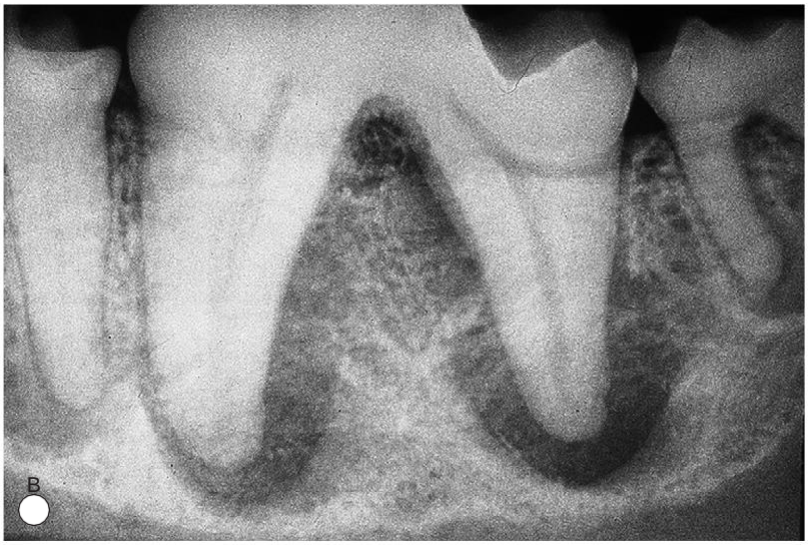
If the enamel dysplasia is the result of a local trauma or systemic pyrexia that resolves within a period time, only those areas undergoing active formation during the period of the insult will be affected. This is seen clinically as bands of dysplastic enamel encircling the crown, with areas of normal enamel else- where on the tooth. Banding is evident in both.

Poorly protected or exposed dentine is painful. These teeth do become less sensitive with increasing age of the animal since secondary dentine is laid down continuously by the pulp. Another consideration is that dysplastic enamel harbours dental plaque. In severe cases of generalized enamel hypoplasia, where the dentine is effectively exposed to the oral environment, chronic pulp disease and potentially periapical disease may occur due to pulpal irritation via the poorly protected or exposed dentine tubules.



Localized enamel dysplasia. A: Localized region of defective enamel of the right mandibular canine tooth. This was the only affected tooth in the dentition. This type of enamel dysplasia is likely to be the result of local trauma, e.g. blow to the face. Only the region of enamel undergoing active formation at the time of the trauma is defective, appearing as a band at the gingival third of the crown. The rest of the crown is covered by normal enamel. **B**: The defect has been debrided (discolored dysplastic enamel was removed with a round bur in a slow-speed hand piece with water cooling) and prepared to accept a restorative material. **C**: Completed restoration using a white filling material (compomer).

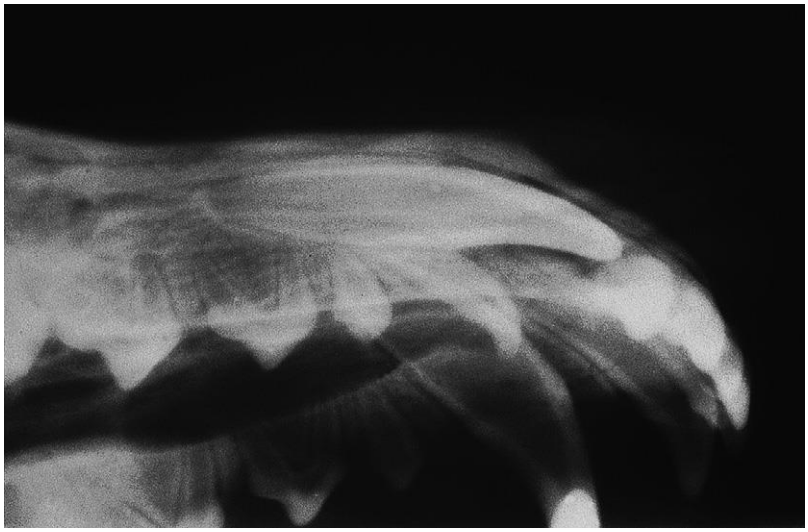




Generalized enamel dysplasia.
A: Enamel dysplasia affecting all teeth of the dentition. This type of enamel dysplasia is likely to be caused by systemic factors, e.g. pyrexia, at the time of active enamel development. Only the areas actively forming at the time of the insult will be affected as is seen by the obvious banding with areas of normal enamel elsewhere on the tooth.
B: A radiograph of the caudal left mandible of the same dog reveals pulp and periapical disease affecting the mandibular 4th premolar and the 1st and 2nd molars. The full mouth radiographic series showed that almost all teeth of the dentition had evidence of pulp and periapical pathology. The dog was referred to me because her teeth were discolored and the enamel had seemed to 'crumble' on ultrasonic scaling. She was 5 years old at the time of referral. Treatment consisted of extraction of all teeth except the incisors and canines as these were unaffected by pulp and periapical disease. Home care was recommended and annual radiographic examination was instituted. The dog was not amenable to toothbrushing, and further extractions due to pulp and periapical pathology have been performed.

DISORDERS OF ERUPTION AND SHEDDING

Unerupted teeth can be detected and evaluated by radiographic examination only. Embedded teeth are those that have failed to erupt and remain completely or partially covered by bone or soft tissue or both. Unerupted teeth may cause no pathology, in which case they do not require any treatment. If an obstruction to eruption can be clearly identified. The follicle of the unerupted tooth undergoes cystic transformation. The resultant follicular (dentigerous) cyst may cause extensive alveolar bone resorption as it increases in size. These cysts expand as an osmotic gradient develops between the cyst lumen and the surrounding tissues. The pressure of the expanding cyst stimulates resorp- tion of the bone. Follicular cysts can become large and cause extensive resorption of the surrounding alveolar bone. Consequently, unerupted teeth that are maintained require regular radiographic monitoring to identify development of a follicular cyst at an early stage. Treatment then consists of removing the unerupted tooth and its associated cyst. Some clinicians choose to extract unerupted teeth as a prophylactic measure.



Unerupted teeth. Unerupted teeth can only be detected and evaluated by radiographic examination. In this patient, the right permanent maxillary canine tooth has not erupted. The right primary maxillary canine tooth is persistent. The owner was not amenable to the regular radiographic evaluation indicated if the unerupted permanent tooth were to be maintained. The chosen treatment in this case therefore consisted of extracting (open/surgical technique) both the persistent primary canine and the unerupted permanent canine.

WEAR OF DENTAL HARD TISSUE

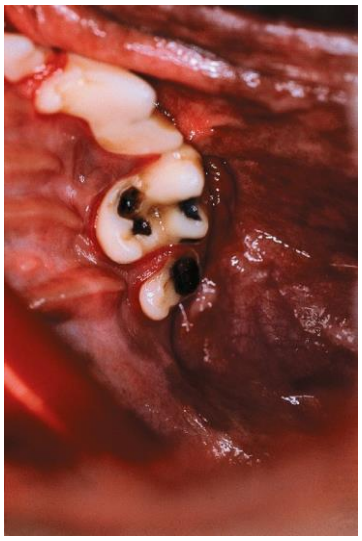
Attrition is the loss of tooth substance that results from wear that is produced by opposing teeth coming into contact with one another, i.e. teeth that have occlusal contact. Attrition is therefore also called occlusal wear. Incisal wear is the term used when describing attrition of the incisor region. There is progressive attrition with increasing age, resulting in the wearing away of the cusps and exposure of the dentine

Attrition. Attrition is the normal wear on tooth surfaces that are in occlusion. As the enamel is worn away, the dentine is exposed to the oral environment. The deposition of secondary dentine keeps pace with the loss of tooth substance and there is rarely pulpal exposure. The exposed dentine is yellow to brown and has a hard surface on exploration with a dental probe/explorer.

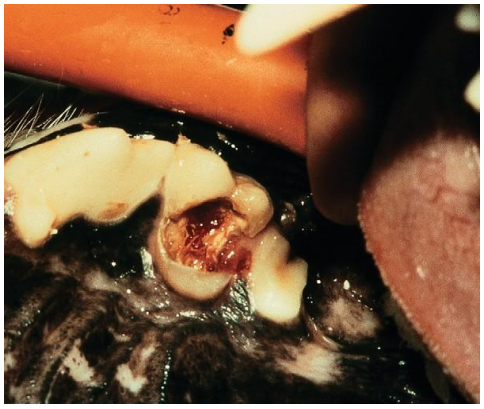
CARIES

Caries (dental decay) occurs in dogs. In our experience, medium and large breed dogs are more commonly affected and the lesions usually affect the teeth that have true occlusal tables, namely the molar teeth. Caries has not been described in cats.

While both periodontal disease and caries are caused by the accumulation of dental plaque on the tooth surfaces, the pathogenesis of the two diseases is completely different. Periodontal disease is a plaque-induced inflammation of the periodontium and caries is a plaque-induced destruction of the hard tissues of the tooth. Caries starts as an inorganic demineralization of the enamel. The demineralization occurs when plaque bacteria use fermentable carbohydrate (notably sugar) from the diet as a source of energy. The fermentation products are acidic and demineralize the enamel. Once the enamel has been destroyed, the process extends into the dentine. In the dentine, the process accelerates as an organic decay and will eventually involve the pulp causing pulpitis and possibly pulp necrosis and/or periapical pathology. Dental caries stimulates the formation of secondary dentine on the surface of the pulpal wall, which is directly beneath it (If the carious lesion is progressing slowly, the deposition of secondary dentine may keep pace with its advance and prevent exposure of the dental pulp.



Caries. The clinical appearance of dental caries affecting the left 1st and 2nd molars is depicted. The black areas were soft on exploration, with the explorer readily 'catching' in the tooth surface. Radiographs are indicated to assess the full extent of the lesions and select appropriate treatment, i.e. extraction or referral.



Caries. The carious lesion of the maxillary 1st molar depicted here has resulted in extensive loss of enamel and dentine and has exposed the pulp chamber to the oral cavity. The pink tissue seen in the centre of the occlusal table is inflamed and hyperplastic pulp tissue (pulp granuloma). Radiographs reveal that the dentine destruction has been so extensive that the furcation of the roots has been broached, i.e. the three roots are unconnected to the crown. Extraction is the only treatment possible for this tooth!

Caries can occur on any tooth surface. How- ever, the occlusal (grinding) surfaces of the molar teeth seem predisposed in dogs. Clinically, caries manifests as softened, often discolored (dark brown or black) areas in the enamel. A dental explorer will 'catch' in the softened carious tooth surface. A small enamel defect covers a large cavern of decayed dentine. Note that not all lesions are grossly discolored and all occlusal surfaces, whether discolored or not, should be meticulously examined with a dental explorer. If the explorer sticks in the tooth surface, then caries should be suspected and radiographs are indicated. Radiographically, radiolucent defects are seen in the affected area of the crown.

Radiographs will also give an indication of how close to the pulp chamber a caries lesion extends (the extent of secondary dentine formation and the amount and thickness of dentine that separates the pulp from the carious lesion), which allows selection of most appropriate treatment. Dis- colored areas that are hard and in which the explorer does not 'catch' are not caries – they could be exposed dentine due to attrition or stain.

Diagnosed caries requires treatment. The options are extraction or referral to a specialist for restoration (if the process involves the pulp tissue, endodontic therapy prior to restoration is required). If the process has resulted in gross loss of tooth substance at the time of diagnosis, then extraction is the only option. Fluoride enhances remineralization and makes the enamel more resistant to the acid dissolution that occurs with caries.

Periodontal disease

Introduction

Periodontal disease is the result of the inflam- matory response to dental plaque, i.e. oral bacteria, and is limited to the periodontium. It is the most common oral disease seen in dogs and

In addition to periodontal disease, a spectrum of inflammatory responses to agents other than plaque (e.g. toxic, viral and unknown) also occurs in the oral cavity. These generally affect the oral mucous membrane, but may also involve the periodontium. Inflammation of the oral mucosa is called stomatitis. Periodontal disease is a collective term for a number of plaque-induced inflammatory lesions that affect the periodontium. The term infection refers to the presence and multiplication of a micro- organism in body tissues. Periodontal disease is a unique infection in that it is not associated with a massive bacterial invasion of the tissues. Gingivitis is inflammation of the gingiva and is the earliest sign of disease. Individuals with untreated gingivitis *may* develop periodontitis. The inflammatory reactions in periodontitis result in destruction of the periodontal ligament and alveolar bone. The result of untreated periodontitis is ultimately exfoliation of the affected tooth. Thus, gingivitis is inflammation that is not associated with destruction (loss) of supporting tissue. It is reversible. In contrast, periodontitis is inflammation where the tooth has lost a variable degree of its support (attachment). It is irreversible. The salient features of gingivitis and periodontitis are depicted in diagrammatic form in Figure 9.1. Periodontal disease can cause discomfort to affected individuals. Moreover, there is strong circumstantial evidence that a focus of infection in the oral cavity may cause disease of distant organs. Consequently, prevention and treatment of periodontal disease is important for the general health of companion animals. It is not a cosmetic issue! Prevention of periodontal disease is detailed in Chapter 10. This chapter details etiology, pathogenesis, diagnosis and treatment. Successful management of periodontal disease relies on a comprehensive understanding of the etiology and pathogenesis of the disease.

ETIOLOGY

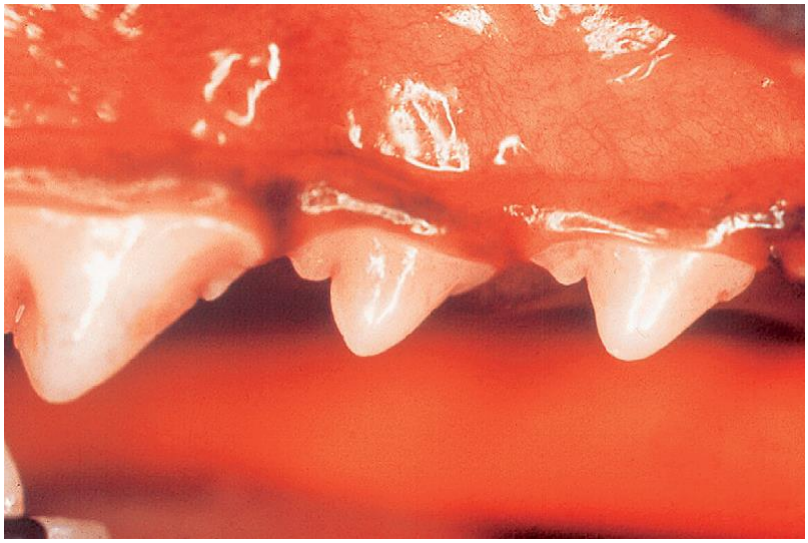
The *primary cause* of gingivitis and periodontitis is accumulation of dental plaque on the tooth surfaces. Contrary to common belief, calculus (tartar) is only a secondary etiologic factor.

Dental plaque

Dental plaque is a biofilm composed of aggregates of bacteria and their by-products, salivary components, oral debris and occasional epithelial and inflammatory cells. Plaque accumu- lation starts within minutes on a clean tooth surface. The initial accumulation of plaque occurs supragingivally but will extend into the sulcus and populate the subgingival region if left undisturbed. As demonstrated in a study where dogs were fed by intubation, the formation of dental plaque occurs whether food passes through the oral cavity or not, i.e. food debris does not attach to the teeth to form plaque. Supragingival plaque bacteria derive their main nutrients from dietary particles dissolved in saliva. Within the sulcus or pathologic periodontal pocket, the major nutritional source for bacterial metabolism comes from the periodontal tissues and blood.

Classic experiments have demonstrated that accumulation of plaque on the tooth surfaces reproducibly induces an inflammatory response in associated gingival tissues, and that removal of the plaque leads to disappearance of theclinical signs of this inflammation. At first, a direct relationship was assumed to exist between the total number of bacteria that accumulated on a tooth surface and the amplitude of the pathogenic effect. are already attached. As soon as a tooth becomes exposed to the oral cavity, its surfaces are covered by the pellicle (an amorphous coating of salivary proteins and glycoproteins). The pellicle alters the charge and free energy of the tooth surfaces, which increases the efficiency of bacterial adhesion. Specific bacteria such as Streptococcus sanguis and Actinomyces viscosus can adhere to the pellicle. These bacteria produce extracellular polysaccharides, which aggregate other bacteria that are not otherwise able to adhere.

The plaque associated with healthy gingiva is mainly comprised of aerobic and facultative anaerobic bacteria. As gingivitis develops, plaque extends subgingivally. Aerobes consume oxygen and a low redox potential is created, which makes the environment more suitable for growth of anaerobic species. The aerobic population does not decrease, but with increasing number of anaerobes, the aerobic/anaerobic ratio decreases. The subgingival flora associated with periodontitis is predominantly anaerobic and consists of Porphyromonas spp, Prevotella spp, Peptostreptococcus spp, Fusobacterium spp and spirochetes. High levels of Porphyromonas spp and spirochetes are consistently associated with progressive periodontitis in the dog. The bacterial flora of the normal feline gingival margin, as well as the bacteria found in subgingival plaque of cats with gingivitis and periodontitis, are similar to those found in humans and dogs.



Dental plaque. Dental plaque is a biofilm composed of aggregates of bacteria and their by- products, salivary components, oral debris and occasional epithelial and inflammatory cells. It starts accumulating within minutes on a clean tooth surface. Plaque may be difficult to see with the naked eye and the use of plaque- disclosing solutions (dyes that stain plaque) is recommended for visualization.

Dental calculus

Dental calculus is mineralized plaque. However, a layer of plaque always covers calculus. Both supragingival and subgingival plaque becomes mineralized. Supragingival calculus *per se* does not exert an irritant effect on the gingival tissues. In fact, it has been shown in monkeys that a normal attachment may be seen between the junctional epithelium and calculus if the calculus surface had been disinfected using chlorhexidine . It has also been shown that sterilized calculus may be encapsulated in connective tissue without causing marked inflammation or abscess formation. It has been speculated that calculus may exert a detrimental effect on the soft tissue owing to its rough surface. However, it has clearly been established that surface roughness alone does not initiate gingivitis. The main importance of calculus in periodontal disease thus seems to be its role as a plaque-retentive surface. This is supported by well-controlled animal and clinical studies that have shown that the removal of subgingival plaque on top of subgingival calculus will result in healing of periodontal lesions and the maintenance of healthy periodontal tissues.

PATHOGENESIS

The pathogenic mechanisms involved in peri- odontal disease include:

- Direct injury by plaque microorganisms and Indirect injury by plaque microorganisms via inflammation.

The microbiota in periodontal pockets is in a continual state of flux; periodontitis is a dynamic infection caused by a combination of bacterial vectors that change over time. As a result, the molecular events that trigger and sustain the inflammatory reactions constantly change. Many microbial products have little or no direct toxic effect on the host. However, they possess the potential to activate nonimmune and immune inflammatory reactions that cause the tissue damage. It is now well accepted that *it is the host's response to the plaque bacteria, rather than microbial virulence* per se *that directly causes the tissue damage*.

In gingivitis, the plaque-induced inflammation is limited to the soft tissue of the gingiva. Sulcus depths are normal (i.e. periodontal probing depths are 1–3 mm in the dog and 0.5–1.0 mm in the cat). As periodontitis occurs, the inflammatory destruction of the coronal part of the periodontal ligament allows apical migration of the epithelial attachment and the formation of a pathologic periodontal pocket (i.e. periodontal probing depths increase). If the inflammatory disease is permitted to progress, the crestal portion of the alveolar process begins to resorb. Alveolar bone destruction type and extent are diagnosed radiographically. The resorption may proceed apically on a horizontal level. Horizontal bone destruction is often accompanied by gingival recession, so periodontal pockets may not form. If there is no gingival recession, the periodontal pocket is supra-alveolar, i.e. above the level of the alveolar margin. The pattern of bone destruction may also proceed in a vertical direction along the root to form angular bony defects. The periodontal pocket is now intra- or subalveolar, i.e. below the level of the crestal bone.

Disease progression is generally an episodic occurrence rather than a continuous process. Tissue destruction occurs as acute bursts of disease activity followed by relatively quiescent periods. The acute burst is clinically characterized by rapid deepening of the periodontal pocket as periodontal ligament fibers and alveolar bone are destroyed by the inflammatory reactions. The quiescent phase is not associated with clinical or radiographic evidence of disease progression. However, complete healing does not occur during this quiescent phase, because subgingival plaque remains on the root surfaces and inflammation per- sists in the connective tissue. The inactive phase can last for extended periods.

Other conditions, such as physical or psycho- logic stress and malnutrition, may impair protective

responses such as the production of antioxidants and acute phase proteins, and can aggravate periodontitis but do not actually cause destructive tissue inflammation. A genetic pre- disposition to destructive inflammation of the periodontium may be important in some indi- viduals. In humans, a strong association has been observed between the severity of periodontitis and a specific genotype of the interleukin-1 (IL-1) gene cluster. Patients carrying this periodontitis-associated genotype (PAG) may demonstrate phenotypic differences, as indicated by elevated levels of IL-1β in gingival sulcular (crevicular) fluid. No similar data are available for the dog or cat.

Significance

Undisturbed plaque accumulation results in gingivitis. While some individuals with untreated gingivitis will develop periodontitis, not all untreated animals will do so. It cannot be predicted which individuals with gingivitis will develop periodontitis. However, animals in which clin- ically healthy gingivae are maintained will not develop periodontitis. Consequently, *the aim in periodontal disease prevention and treatment is to establish and maintain clinically healthy gingivae to prevent periodontitis.*

DIAGNOSIS

General considerations

Diagnosis of periodontal disease relies on clinical examination of the periodontium in the anesthetized animal. In addition, radiography is mandatory if there is evidence of periodontitis on clinical examination. It is essential to differ- entiate between gingivitis and periodontitis in order to institute appropriate treatment. In individuals with gingivitis, the aim is to restore the tissues to clinical health; in individuals with established periodontitis, the aim of therapy is to prevent progression of disease.

Oral examination and recording of findings are detailed in Chapter 6.

The following parameters need to be assessed and recorded for *each tooth* in *all patients*:

1. Gingivitis and gingival index
2. Periodontal probing depth (PPD)
3. Gingival recession (GR)
4. Furcation involvement
5. Mobility.

Periodontal probing depth, gingival recession, furcation involvement and mobility measure the extent of destruction of the periodontium, i.e. assess the presence and severity of periodontitis. I do not assess and record the extent of plaque and calculus accumulation in patients that are seen for the first time. These deposits will be removed during periodontal therapy. Instead, I assess and record plaque at follow-up visits to assess the efficacy of the homecare regimen that has been instituted. Plaque accumulation is visualized using a plaque disclosing solution and the teeth that have plaque at the gingival margin are noted and recorded. The amount of plaque is graded subjectively as mild, moderate or severe depending on the depth of staining achieved by the plaque disclosing solution.

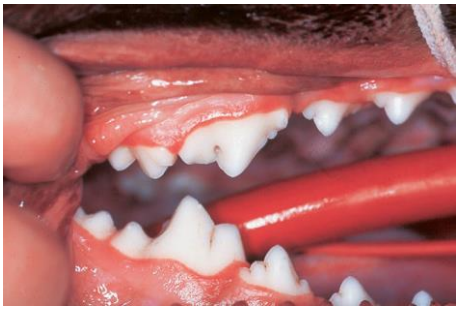
Gingivitis

Gingivitis is defined as a *reversible* plaque- induced inflammation limited to the gingiva (i.e. no loss of periodontal attachment).

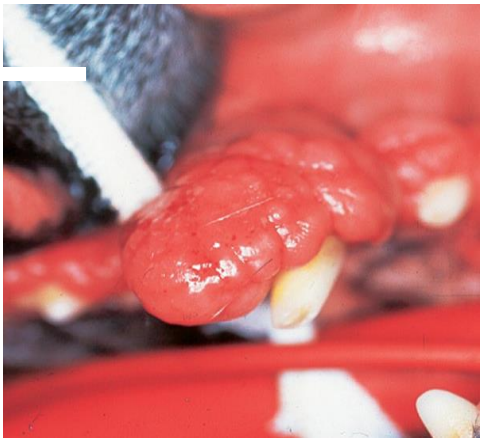
Clinical signs and diagnostic methods

Gingivitis manifests clinically as swelling, reddening and often bleeding of the gingival margin. It

may be accompanied by halitosis. It is diagnosed clinically by means of a combination of visual inspection and tactile examination. The presence and degree of gingival inflammation is assessed based on a combination of redness and swelling, as well as presence or absence of bleeding on gentle probing of the gingival sulcus. Various indices can be used to give a numerical value to the degree of gingival inflammation present. In the clinical situation, a simple bleeding index may be the most useful. Using this method the gingival sulcus of each tooth is gently probed at several points and given a score of 0 if there is no bleeding and a score of 1 if the probing elicits bleeding. The patient with uncomplicated gingivitis will have normal peri- odontal probing depths (1–3 mm in the dog and 0.5–1.0 mm in the cat) and show no evidence of gingival recession, furcation involvement or tooth mobility. Radiography is not mandatory if the clinical examination reveals no evidence of peri- odontal destruction, i.e. periodontitis.



Gingivitis. Gingivitis manifests clinically as swelling and reddening of the gingival margin.



Gingival hyperplasia. The hyperplastic gingival tissue almost covers the crowns, resulting in the formation of pseudopockets

Consequences to affected animal

Uncomplicated gingivitis is generally not associ- ated with discomfort or pain in humans. In fact, it is an insidious process and the patient may be unaware of its existence. The significance of gingivitis is that, if untreated, periodontitis may develop as described earlier.

Gingival hyperplasia does pose an additional concern. The hyperplastic gingiva alters the pos- ition of the gingival margin and results in a false or 'pseudo' pocket. It is called a pseudopocket because the increased periodontal probing depth is not due to destruction of periodontal ligament and alveolar bone with apical migration of the junctional epithelium, as in periodontitis. Instead, the increased periodontal probing depth is due to the overgrowth of the gingiva. The presence of hyperplastic gingiva compromises tooth cleaning and may predispose to periodontitis. Radio- graphy is mandatory for patients with gingival hyperplasia.

Periodontitis

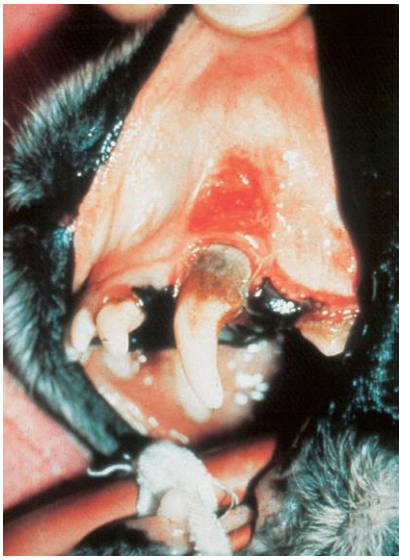
Individuals with untreated gingivitis may develop periodontitis. The inflammatory reactions in periodontitis result in destruction of the peri- odontal ligament and alveolar bone. The result of untreated periodontitis is eventually exfoliation of the affected tooth. It is important to remember that periodontitis is a site-specific disease, i.e. it may affect one or more sites of one or several teeth. Periodontitis can generally be considered irrevers- ible. The aim of treatment is thus to prevent develop- ment of new lesions at other sites and to prevent further tissue destruction at sites which are already affected.

Clinical signs

Halitosis is common and is often the first sign noted by the pet owner. Large amounts of dental deposits are usually present. These deposits need to be removed to allow a detailed examination of the periodontium. Ulcers affecting mucous membranes of lips and cheeks may be present in areas where these tissues are exposed to plaque- covered tooth surfaces.

Diagnostic methods

Tissue destruction in periodontitis is assessed by measuring periodontal probing depth, gingival recession, furcation involvement and degree of tooth mobility. In many cases, measuring or calculating the periodontal attachment level (PAL) is also useful. Periodontal probing depth (PPD) is not necessarily correlated with severity of attachment loss. Gingival hyperplasia may contribute to a deep pocket (or pseudopocket if there is no attachment loss), while gingival recession may result in the absence of a pocket but also minimal remaining attachment.



Gingival recession and mucous membrane ulceration. The periodontal ligament and alveolar bone on the labial aspect of the left upper canine has been destroyed. The gingival margin has receded. Periodontal probing depth is 1 mm, i.e. there is no pathological pocket. A mucous membrane ulcer has developed on the lip surface that is in contact with the plaque-covered tooth surface. While uncomplicated periodontitis is not associated with severe discomfort, these mucous membrane ulcers are known to be painful!

Consequences to affected animal

Based on feedback from human patients, uncomplicated periodontitis is not associated with severe pain or discomfort. In contrast, complications such as the development of a lateral periodontal abscess or ulcers in the mucous membranes are very painful. It has been shown that a severe infection in the oral cavity, as with extensive periodontitis, will lead to a transient bacteremia on chewing. In fact, an association has been demonstrated between periodontal disease and histopathologic changes in kidney, myo- cardium, and liver in the dog.

TREATMENT

General considerations

The treatment of periodontal disease is aimed at controlling the cause of the inflammation, i.e. dental plaque. Conservative or cause-related periodontal therapy consists of removal of plaque and calculus, and any other remedial procedures required, under general anesthesia, in combination with daily maintenance of oral hygiene. In other words, the treatment of periodontal disease has two components:

1. Maintenance of oral hygiene
2. Professional periodontal therapy.

Maintenance of oral hygiene is performed by the owner and is often called home care. Its effective- ness depends on the motivation and technical ability of the owner and the cooperation of the animal. Home care is detailed in Chapter 10.

Professional periodontal therapy is performed under general anesthesia and includes:

- Supra- and subgingival scaling
- Root planing
- Tooth crown polishing
- Subgingival lavage
- And sometimes periodontal surgery.
- The term 'dental prophylaxis' or 'prophy' has been used to encompass clinical examination and professional periodontal therapy. This is misleading since the real prophylaxis, i.e. steps taken to prevent disease development and progression, is not the professional periodontal therapy carried out under general anesthesia but the daily home care regime to remove plaque. If no home care is instituted, then plaque will rapidly reform after a professional periodontal therapy procedure and the disease will progress. Before any treatment is instituted, the owner must be made aware that home care is the most essential component in both preventing and treating periodontal disease. Whenever possible it is useful to institute a home care programme before any professional periodontal therapy is performed. The aim of treatment differs whether the patient has gingivitis only or whether the patient also has periodontitis.

Gingivitis

Gingivitis is by definition reversible. Removal or adequate reduction of plaque will restore inflamed gingivae to health. Once clinically healthy gingivae have been achieved, these can be main- tained by daily removal or reduction in the accumulation of plaque. In short, the treatment of gingivitis is to restore the inflamed tissues to clinical health and then to maintain clinically healthy gingivae, thus preventing peri-

odontitis. The purpose of the professional periodontal therapy in the gingivitis patient is removal of dental deposits, mainly calculus (which is not removed by toothbrushing). Once the teeth have been cleaned it remains up to the owner to remove the plaque that reccumulates on a daily basis.

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Summary for treatment of gingivitis

- Educate the owner to understand the disease process
- Train and motivate the owner to perform daily home care
- Institute daily home care regimen by the owner – ideally, toothbrushing with a pet toothpaste in conjunction with a dental hygiene product
- Professional periodontal therapy (supra- and subgingival scaling and polishing) under general anesthesia to remove dental deposits (plaque and calculus)
- Regular check-ups to ensure that the owner is following recommendations and to boost the owner's motivation

Periodontitis

Untreated gingivitis may progress to periodontitis. In most instances in a practice situation, periodontitis is irreversible. It is important to remember that periodontitis is a site-specific disease, i.e. it may affect one or more sites of one or several teeth. The aim of treatment is thus to prevent development of new lesions at other sites and to prevent further tissue destruction at sites which are already affected.

Professional periodontal therapy removes den- tal deposits above and below the gingival margin. It then rests with the owner to ensure that plaque does not re-accumulate. Meticulous supragingival plaque control, by means of daily toothbrushing and adjunctive antiseptics when indicated, will prevent migration of the plaque below the gingival margin. If the subgingival tooth surfaces are kept clean, the sulcular epithelium will reattach.

In patients with suspected periodontitis, I recommend instituting daily toothbrushing three to four weeks prior to the planned professional periodontal therapy if the animal will allow it. This will result in less inflamed tissue at the time of professional therapy and will allow assessment of the ability of the owner to perform home care. If home care is not possible, the professional treat- ment will need to be more radical, e.g. extraction of teeth that could potentially have been retained with good home care.

Periodontal surgery is never first line treat- ment for periodontal disease. Conservative management of periodontal disease, i.e. a thorough supra- and subgingival scale, root planing, polishing and irrigation in combination with daily meticulous home care is the first step. Periodontal surgery should only be performed where the owner has shown the ability to keep the mouth clean. If a client cannot maintain good oral hygiene measures in their pet then in the interest of the well being of the animal there is no indication for surgery.

PROFESSIONAL PERIODONTAL THERAPY

General considerations

Professional periodontal therapy must be per- formed under general anesthesia. Anesthesia and special care of the patient undergoing dentistry and/or oral surgery is covered in Chapter 2. The basic instrument requirements for periodontal therapy are covered in Chapter 1. Antibiotics should not be used to treat periodontal disease in the absence of mechanical debridement.

The degree of discomfort or pain caused by dental and/or oral surgery procedures is usually not considered. Domestic pets have a dental anatomy and nerve paths similar to our own. Their perception of pain may well be different but an analogous view is to assume that procedures that cause discomfort in humans are likely to do the same in dogs and cats.

To master the technical skills required for dentistry and oral surgery, attending practical courses is recommended. In general, dental instruments are held in a modified pen gripand the 4th and 5th fingers are placed on adjacent structures (neighboring teeth, opposite jaw) for stability and support.

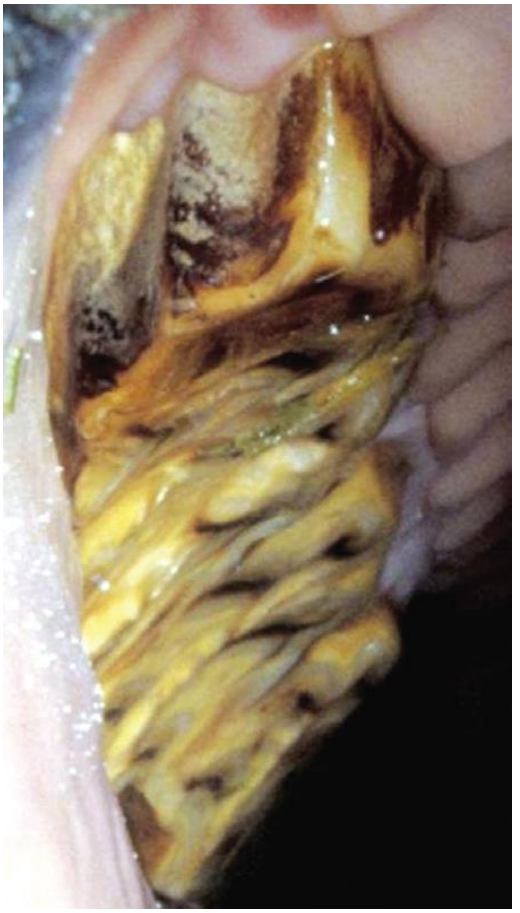
Summary for treatment of periodontitis

- Educate the owner to understand the disease process
- Train and motivate the owner to perform daily home care
- Institute daily toothbrushing regimen by the owner
- Professional periodontal therapy: this includes supra- and subgingival scaling and polishing, root planing and extraction of unsalvageable teeth under general anesthesia
- Regular check-ups to ensure that the owner is following recommendations and to boost the owner's motivation
- Periodontal surgery may be indicated

Equine dental pathology

Shear mouth

This right maxillary cheek teeth (CT) row has markedly increased (circa 45°) occlusal angulation, i.e., shear mouth.



Wave mouth

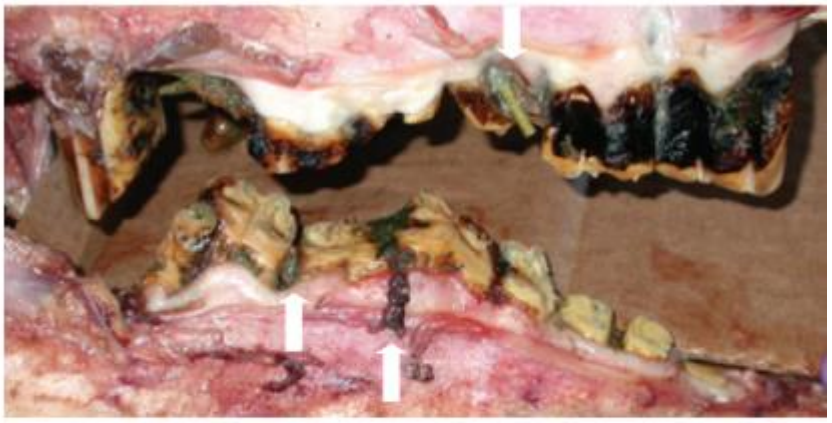


Fig. 10.3 Post-mortem image of a neglected, aged equine mouth that has multiple dental abnormalities, including wave mouth in the centre of the CT rows, smooth mouth of the rostral mandibular CT (406, 407 are worn to their component roots) and step mouth of 111 due to loss of 411. Diastemata and associated periodontal disease are present between some teeth (arrows).

Diastemata

Cheek teeth diastema, which is defined as a detectable interdental (interproximal) space between adjacent teeth, was diagnosed as the primary dental disorder in 4% of horses

Fig. 10.6 This post-mortem image of a caudal right hemimandible has two valve diastemata (narrow occlusally and wider at gingival level) with deep periodontal food pocketing between the two CT on the right.

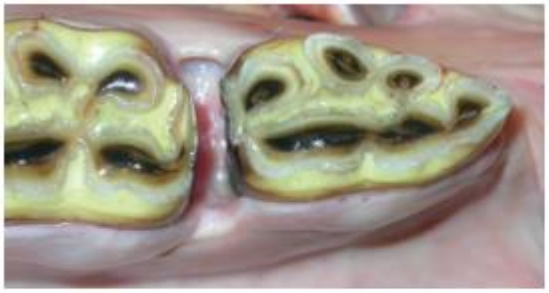


Fig. 10.7 These mandibular CT (306–307) have an open diastema between them, but due to the shape of the diastema, it contains no food pocketing. Consequently, the underlying gingiva, although recessed, has a smooth appearance and is of normal color.

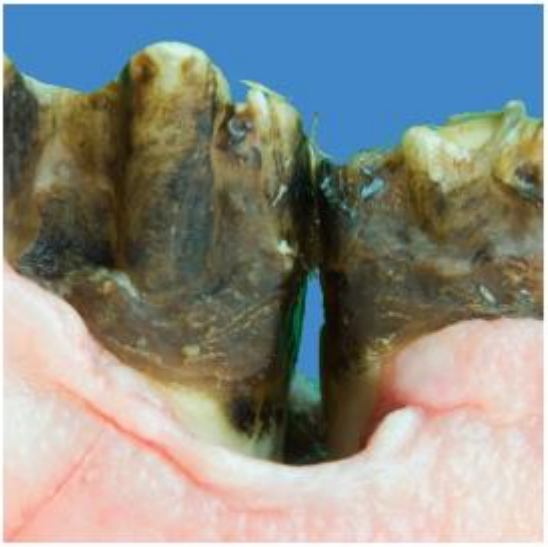


Fig. 10.8 These two mandibular CT have a valve diastema between them that has led to marked periodontal recession at, and adjacent to, the diastema. These two CT also have loss of much of their peripheral cementum likely due to prior peripheral dental caries, with marked staining of all exposed calcified tissues. (Courtesy of Alistair Cox and Sionagh Smith.)