# 1 Alimentary system

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# ORAL CAVITY

Examination of the oral cavity should be standard procedure during any postmortem examination. To obtain a clear view of the mucous membranes of the buccal and oral cavities, teeth, tongue, gums, and tonsils, it is essential to split the mandibular symphysis and separate the mandibles as far as possible. A thorough examination of all structures will reveal not only local lesions, but often those that may be due to systemic disease. Lesions may be associated with congenital anomalies; trauma (physical and chemical); bacterial, mycotic, viral, and parasitic infections; metabolic and toxic diseases; and immune-mediated, dysplastic, or neoplastic disease. The poor physical condition of an animal may be directly related to oral lesions that result in difficulties of prehension, mastication, or swallowing of food.

## Congenital anomalies

The development of normal facies and the oral cavity requires the integration of many embryonic processes. The complexity and duration of this development may lead to a great variety of aberrations. These are usually expressed in the newborn in the form of *clefts resulting from failures of integrated growth and fusion*. A common failure of fusion is that of the maxillary processes to the frontonasal process. This may leave facial fissures, cleft lip (harelip, cheiloschisis) and uni- or bilateral primary cleft palate involving the area rostral to the incisive papilla.

**Facial clefts** may involve the skin only, or the deeper tissues as well. They are variously located, and not all are obviously related to normal lines of fusion. All are rare. The least uncommon is a complete cleft from one angle of the mouth to the ear of that side. This results from failure of fusion of the lateral portions of the maxillary and mandibular processes. A defect extending from a cleft lip to the eye results from failure of fusion of the maxillary and frontonasal processes; its least expression is superficial and a failure of closure of the nasolacrimal duct.

**Primary cleft palate** (harelip, cheiloschisis) includes developmental anomalies of the lips rostral to the nasal septum, columella, and premaxilla. They may be uni- or bilateral and superficial or extend into the nostril. The defect arises from incomplete fusion of the frontonasal process with the maxillary processes.

**Secondary cleft palate** (cleft palate, palatoschisis) (Fig. 1.1) is often associated with primary cleft palate. The normal hard palate is formed, except for a small rostral contribution from the frontonasal process, by the bilateral ingrowth of the lateral palatine shelves from the maxillary processes. At the midline, they fuse with each other and with the nasal septum, and undergo intramembranous ossification, except in their caudal part, which becomes the soft palate. Inadequate growth of the palatine shelves leaves a central defect, in either or both of the hard and soft palates, which communicates between the oral and nasal cavities. Other manifestations of disordered palatogenesis include unilateral defects in the soft palate; bilateral hypoplasia of the soft palate; or dorsal displacement of the soft palate, with excess soft tissue on the caudal portion. Affected animals have difficulty sucking, may have nasal regurgitation, and usually die within the first few days of life from aspiration pneumonia.

*Cleft palates have been reported in most species of domestic animals*. In one extensive survey of Thoroughbred foals, 4% of congenital defects were secondary cleft palates. Most of these foals had a complete cleft of the hard palate; a few had clefts or hypoplasia of the soft palate only. In calves, cleft palate is one of the most common anomalies, but is very uncommon in sheep. Primary cleft palate is less common than secondary cleft palate in swine, although the two anomalies often occur together.

**Figure 1.1 Secondary cleft palate** exposing the nasal cavity in a calf.

The etiology of cleft palate is usually unknown, but examples of *hereditary causes*, maternal ingestion of certain *drugs*, or maternal consumption of *teratogenic plants* during pregnancy have been demonstrated. Secondary cleft palate and arthrogryposis frequently occur together in Charolais calves, and appear to be hereditary (probably simple autosomal recessive), as in Hereford cattle. These two anomalies have also been associated with the ingestion of certain lupines by cows during gestational days 40–70. Cleft palate in lambs may be genetic in origin, but also is associated with the ingestion of *Veratrum californicum*. Secondary cleft palates have been induced experimentally in newborn pigs by feeding gilts seeds or plants of poison hemlock (*Conium maculatum*) during gestational days 30–45. Both tree tobacco (*Nicotiana glauca*) in western USA and tobacco stalks (*N. tabacum*) when fed to gilts early in pregnancy can induce a high incidence of cleft palate and arthrogryposis in newborn pigs. Piperidine alkaloids (coniine, coniceine, and anabasine) in hemlock and

tobacco plants are responsible for the teratogenic effects of these plants. Lupines (*Lupinus formosus, L. arbustus*) produce piperidine alkaloids, including the teratogen ammodendrine, which can cause cleft palate and arthrogryposis (crooked calf disease) in calves born of dams fed the lupine at days 40–50 of gestation. Palatoschisis in piglets has also been associated with consumption of feed contaminated with *Crotalaria retusa* seed by sows during gestation. Primary and secondary cleft palate of German Boxer dogs appear to be hereditary, probably due to a single autosomal recessive gene. Secondary cleft palate occurs in Siamese and Abyssinian cats and is thought to be hereditary, although the mode of inheritance has not been determined. Griseofulvin treatment of the pregnant queen and mare will result in palatoschisis in the offspring. The defect has also been reported in both parts of the doubled face in diprosopus cats.

Anomalies in the growth of jaws are quite common. **Brachygnathia superior**, *shortness of the maxillae*, is an inherited breed characteristic among dogs and swine. It has been reported in the Large White or Yorkshire breed. The condition is progressive with age, resulting in malapposition of the incisor and cheek teeth, which interferes with prehension and mastication. In swine, brachygnathia superior may be confused with atrophic rhinitis. In Angus and Jersey cattle, brachygnathia superior occurs as a hereditary trait. It may, in any species, be associated with chondrodysplasia and is also present with other facial defects.

**Brachygnathia inferior** or micrognathia, *shortness of the mandibles*, may be a mild to lethal defect in cattle and sheep and is a breed characteristic of long-nosed dogs. Micrognathia is a common defect in calves. It is inherited, probably as a simple autosomal recessive trait. There is a higher incidence in males. In Aberdeen Angus cattle, the defect may occur concurrently with cerebellar hypoplasia, and with osteopetrosis in this and other breeds (see Vol. 1, Bones and joints). Mild brachygnathia inferior, termed *parrot mouth*, is a common conformational defect in horses.

**Prognathism** refers to *abnormal prolongation of the mandibles*. It is rather common, especially in sheep. It may develop with recovery from calcium deficiency in this species (see Vol. 1, Bones and joints). The malformation is relative, and it is not always easy to determine whether the jaw is absolutely long or merely apparently so, relative to a mild brachygnathia superior.

**Agnathia** is a mandibulofacial malformation characterized by *absence of the lower jaw*, due to failure of development of the first branchial arch and associated structures. The defect is one of the most common anomalies in lambs but is rare in cattle. Associated malformations in lambs may include ateloprosopia (incomplete development of the face), microglossia or aglossia, and atresia of the oropharynx. Concurrent anomalies affecting other body systems may also be evident.

A lethal glossopharyngeal hereditary defect, termed **bird tongue** and caused by a simple autosomal recessive gene, has been reported in dogs. Affected pups have a narrow tongue, especially the rostral half, where the margins are folded medially on to the dorsal surface. The pups are unable to swallow. The muscle fibers of the affected tongues are normal histologically. **Hypertrophy of the tongue** occurs as a congenital anomaly in pigs.

**Persistent oropharyngeal membrane** in a Hereford calf resulted in separation of the oral cavity from the pharynx, and inability to swallow milk. The cause was unknown, and the lesion is very rare.

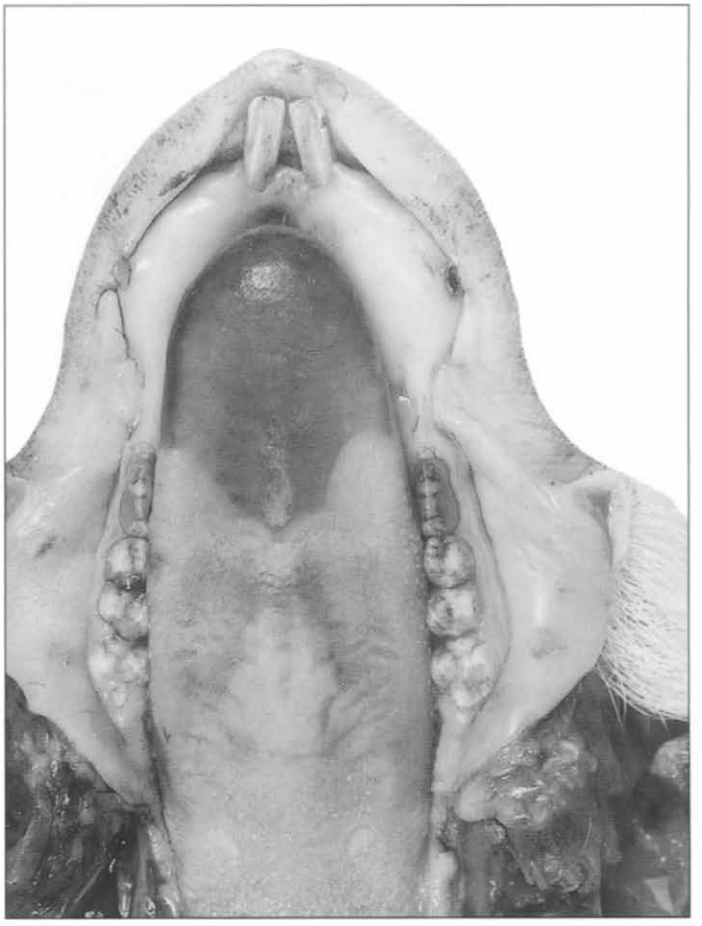

**Figure 1.2 Epitheliogenesis imperfecta** of the tongue of a pig.

**Epitheliogenesis imperfecta** is an anomaly causing *widespread defects in cutaneous epithelium, and also affects the epithelial lining of the oral cavity, especially the tongue* (Fig. 1.2) (see Vol. 1, Skin and appendages). The condition is characterized by irregular, well-demarcated, red areas from which the epithelium of the oral mucosa is absent. Histologically, these consist of abruptly defective areas in the squamous mucosa with inflammation of the submucosal connective tissues. The anomaly occurs in most species and is inherited as a simple autosomal recessive character in cattle, horses, and pigs; the mode of inheritance is unknown in the other species. There are several hereditary skin conditions in animals, such as epidermolysis bullosa simplex in collie dogs, ovine epidermolysis bullosa in Suffolk and South Dorset Down sheep, and familial acantholysis of Aberdeen Angus calves, which have minor involvement of the lips and oral mucosa (see Vol. 1, Skin and appendages).

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## Diseases of teeth and dental tissues

Dental examinations in animals are usually cursory, except to assess age, but dental disease is common and often is the factor that limits useful lifespan, especially of sheep. The comments on dental development and anatomy are intended to assist the understanding of dental disease.

Teeth develop from horseshoe-shaped thickenings in the oral ectoderm called *dental laminae*. Neural crest cells beneath the laminae induce formation of *tooth buds*, which generate the *enamel organs*. These epithelial structures grow into the underlying ectomesenchyme and organize it to form *dental papillae*, which they enclose like a cap. Surrounding both is another mesenchymal condensation, the *dental sac*, which has a collagenous inner layer, the dental follicle, and an outer fibrovascular layer. Developing teeth erupt through a combination of root elongation, alveolar bone remodeling, and periodontal ligament formation. *Periodontal ligament formation* may be more important for species in which the teeth continuously erupt during life. Tooth eruption is induced by the enamel organ that secretes mediators transforming growth factor-α and interleukin-1β to recruit monocytes in the dental follicle for osteoclast resorption and formation of an eruption pathway.

The *inner enamel epithelium* of the enamel organ induces differentiation of odontoblasts from the mesenchyme of the papilla. Odontoblasts produce *dentin*, which in turn induces enamel formation by the inner enamel epithelium. Formation of dentin is essential for the formation of enamel. These inductive interactions of epithelium and mesenchyme are considered important in the histodifferentiation of some tumors of dental tissues.

The free edge of the enamel organ extends beyond the enamel–dentin junction, and this extension is called *Hertwig's epithelial root sheath*. It molds the dental papilla to form the root or apex of the tooth. Subsequently it fragments, allowing mesenchymal cells from the dental sac to contact the root dentin, differentiate into cementoblasts, and deposit cementum on the dentin. Remnants of the root sheath are called *epithelial rests of Malassez*. They persist in the periodontal ligament, and may give rise to tumors or cysts. They may be important in the induction or repair of cementum, and in periodontal reattachment following injury. In pigs and sheep, the rests may be incorporated into the junctional epithelium as it migrates apically in chronic periodontal disease. Cells of the root sheath that adhere to the dentin can produce *enamel pearls*.

Once the dental lamina has produced the buds of the permanent teeth, it degenerates. Epithelial remnants persist as *epithelial pearls or islands* in the gingiva and jaws. These remnants also may give rise to tumors and cysts.

There are important differences between the **brachydont teeth** of humans, carnivores, and swine, in which the enamel is restricted to the tooth crown, and the **hypsodont teeth** of herbivores. In hypsodont teeth, enamel extends far down on the roots, and is invaginated into the dentin to form *infundibula*. Also, the hypsodont teeth of herbivores, except the mandibular premolars of ruminants, are covered by cementum, which more or less fills the infundibula. Exceptions to these rules are provided by the tusks of boars, which are hypsodont, but not covered by cementum, and by ruminant incisors, which are brachydont but do have enamel covering part of the root dentin and cementum covering the root enamel.

The three hard tissues of teeth are dentin, enamel, and cementum. **Dentin** is light yellow and constitutes most of the tooth. It consists of ~35% organic matter and ~65% mineral. Thus its composition is similar to that of bone, and like bone, it contains type I collagen. Dentin is produced by columnar cells with basal nuclei called *odontoblasts*, which differentiate from mesenchyme of the dental papilla. It is formed as unmineralized predentin. The odontoblasts move away from the dentin–enamel junction, gradually encroaching on the pulp cavity as they produce dentin. Each odontoblast has a process extending into the dentin, encased in a dentinal tubule, which arborizes at the dentin–enamel junction. The process also anastomoses with the processes of other odontoblasts. Dentinal tubules are visible in histologic sections, but the anastomoses are not. Except for the processes, and nerve endings in the dentinal tubules near the pulp, dentin is acellular.

Normal dentin contains incremental or *imbrication lines of von Ebner*, which are fine basophilic lines running at right angles to the

dentinal tubules. They represent normal variations in the structure and mineralization of dentin. Sublethal injury caused by certain infections, metabolic stresses, or toxic states may injure the odontoblasts, which then produce accentuated incremental lines known as the *contour lines of Owen*. Sometimes irregular zones of unmineralized or poorly mineralized dentin form between foci of normal mineralization. These are zones of *interglobular dentin*, which may be caused by hypophosphatemia.

Odontoblasts normally are active throughout life, producing layers of secondary dentin, which often contain fewer dentinal tubules than primary dentin. *Reparative dentin* is produced locally in response to injury to dentinal tubules, and contains a limited number of twisted tubules and sometimes a few odontoblasts, which soon die. Reparative dentin may resemble bone and is sometimes called *osteodentin*. *Sclerotic (transparent) dentin* is formed when dentinal tubules are occluded by calcium salts. The junctions between primary, secondary, and reparative dentin are usually demarcated by basophilic lines.

**Enamel** has ~5% organic matter and ~95% mineral. It is produced by the tall columnar *ameloblasts* of the inner enamel epithelium. Enamel is produced in the form of prisms or rods, cemented together by a matrix. Mineralization begins as soon as it is formed and is a two-stage process, somewhat similar to that in bone, but much more rapid. The cells of the inner enamel epithelium also move away from the dentin–enamel junction as the tooth is formed, but, unlike odontoblasts, they do not have processes. Enamel is hard, dense, brittle, and permeable, and is translucent and white. Mature enamel is not present in demineralized sections, but some of the matrix of immature enamel may be visible near ameloblasts of developing teeth.

Ameloblasts are very sensitive to environmental changes. Normal enamel contains *incremental lines of Retzius*, which are analogous to the incremental lines of von Ebner in dentin, and also reflect variations in structure and mineralization. The incremental lines are accentuated during periods of metabolic stress. More severe injury, as in fluorosis, or infections by some viruses can produce focal **hypoplasia or aplasia of enamel**.

Formation of enamel ends before tooth eruption. The inner enamel epithelium then merges with the cells of the underlying stratum intermedium and the outer enamel epithelium to form the *reduced enamel epithelium*. It protects the enamel of the formed tooth prior to eruption. Degeneration of this protective layer permits connective tissue to contact the enamel, and there may be resorption of enamel or deposition of a layer of cementum on it. This normally occurs during odontogenesis in horses.

**Cementum** is an avascular, bonelike substance, produced by *cementoblasts*; it contains ~55% organic and ~45% inorganic matter. In general, the dentin of brachydont teeth is covered by cementum wherever it is not covered by enamel. When dentin formation has begun in the root, degeneration of Hertwig's epithelial root sheath begins and permits mesenchymal cells from the dental sac to contact dentin. They differentiate into cementoblasts, which produce *cementoid*, and later mineralize it. Some layers of cementum do not contain cells (acellular cementum), but in other layers, *cementocytes* are enclosed in lacunae. Sharpey's fibers from alveolar bone are embedded in the cementum. Cementum is more resistant to resorption than is bone, and unlike bone, normally is not resorbed and replaced as it ages; instead a new layer of cementum is deposited on top of the old layer. In some pathologic conditions cementum is resorbed; subsequently, cellular or acellular cementum is deposited, and more or less repairs the defect.

**Hypercementosis** is abnormal thickening of cementum and may involve part or all of one or many teeth. When extra cementum improves the functional properties of teeth, it is called *cementum hypertrophy*; if not, it is called *cementum hyperplasia*. Extensive hyperplasia is often associated with chronic inflammation of the dental root.

The **periodontal ligament** is very cellular, well-vascularized connective tissue that develops from the dental sac. The **periodontium** comprises the periodontal ligament, gingival lamina propria, cementum, and alveolar bone. The ligament supports the tooth and adjusts to its movement during growth. It is well supplied with nerves and lymphatics, which drain into alveolar bone. The periodontal ligament is also a source of the cells that remodel alveolar bone and, in disease, cementum.

**Epithelial rests of Malassez** are present in the periodontal ligament and are particularly numerous in the incisor region of sheep. In all species, they may proliferate and become cystic when there is inflammation of the periodontium. The periodontium is also a site of origin of tumors. The periodontal ligament is normally visible in radiographs as a radiolucent line between tooth and alveolar bone. In prolonged hyperparathyroidism, alveolar bone is resorbed, and the ligament is no longer outlined radiographically, a change referred to as *loss of the lamina dura*.

## Developmental anomalies of teeth

**Anodontia**, *absence of teeth*, is inherited in calves, probably as a sex-linked recessive trait in males, and is associated with skin defects. **Oligodontia**, *fewer teeth than normal*, occurs sporadically in horses, cats, and dogs, and also as an inherited trait in dogs. In brachycephalic breeds, the cheek teeth are deficient; in toy breeds, the incisors are deficient. *Pseudo-oligodontia* and *pseudoanodontia* result from failed eruption. These defects may be associated with bone-modeling defects in gray lethal mice with osteopetrosis. Delayed eruption of permanent teeth occurs in Lhasa Apso and Shih Tzu dogs. **Polyodontia**, *excessive teeth*, occurs in brachycephalic dogs; the incisors are involved, and the defect is probably related to breeding for broad muzzles. A high incidence of canine polyodontia, involving particularly an extra maxillary premolar, is reported from the Netherlands. Polyodontia also occurs in horses and cats, involving either incisors or cheek teeth. **Pseudopolyodontia** *is retention of deciduous teeth after eruption of the permanent dentition*. It occurs in horses, cats, and dogs, especially in the miniature breeds.

**Heterotopic polyodontia** *is an extra tooth, or teeth, outside the dental arcades*. The best-known example is the *ear tooth of horses*, which develops in a branchiogenic cyst. The cysts originate from failure of closure of the first branchial cleft, or from the inclusion of cellular rests in this area. They are lined by a stratified mucous or cutaneous-type epithelium, and may contain one or more teeth, either loosely attached in the cyst wall or deeply embedded in the petrous temporal bone. The tooth is derived from misplaced tooth germ of the first branchial arch, which is displaced toward the ear with the first branchial cleft. The cysts form in the parotid region and may fistulate to the exterior. They are occasionally bilateral.

Rarely the tooth may form a pedunculated mass enclosed by skin, and attached by a pedicle to the skin of the head. Heterotopic polyodontia also occurs in cattle, dogs, pigs, and sheep.

Developmentally **misshapen teeth** are classified as **geminous** (dichotomous) when there is a single root and partially or completely separate crowns; **fused**, when the dentin of two teeth is confluent; and **concrescent** when the dentin is separate but the roots are joined by cementum. Gemination represents the embryologic partial division of a tooth primordium. It occurs in dogs, usually involving the incisors, and the affected tooth usually has a groove dividing the crowns, whose pulp chambers can be seen radiographically to merge in a common root. Misshapen teeth and missing teeth have also been reported in dogs as an X-linked recessive trait of ectodermal dysplasia. Fusion and concrescence represent the joining of two adjacent tooth primordia, one of which may be supernumerary. *Malformation and malpositioning of teeth* accompany abnormalities of the jaw bones. Aberdeen Angus and Hereford calves with congenital osteopetrosis have brachygnathia inferior, malformed mandibles, and impacted cheek teeth. Impacted molars occur as an inherited lethal in Shorthorns; an association with osteopetrosis apparently has not been investigated in this breed.

**Odontogenic cysts** are epithelium-lined cysts derived from cell rests of Malassez, cell rests of dental laminae, reduced enamel epithelium, or malformed enamel organs. **Dentigerous cysts** are, by definition, *cysts that contain part or all of a tooth*, which is often malformed. Of the odontogenic cysts listed, all except those derived from cell rests of Malassez are potentially dentigerous. (The rests of Malassez are the probable source of periodontal cysts.) Dentigerous cysts originating in malformed enamel organs should include malformed teeth, since development of enamel is incomplete until the organ degenerates. Those teeth in cysts of reduced enamel epithelium or rests of dental laminae are not necessarily abnormal. The affected teeth probably erupt into the preformed cysts. Dentigerous cysts enclose at least the crown of the tooth, but may include it all. The most common forms of odontogenic dentigerous cysts in animals are those involving the *vestigial wolf teeth* of horses and the *vestigial canines*, especially of mares. The smaller cysts appear as tumors of the gums, whereas some of the larger ones may cause swelling of the jaw or adjacent maxillary sinus. Dentigerous cysts of animals are not as destructive as those in humans, in which species they are regarded as the most common benign destructive lesion of the skeleton.

The ear tooth of horses is probably the most common nonodontogenic dentigerous cyst. Occasionally true dentigerous cysts form when a tumor prevents normal eruption or when there is maleruption due to odontodystrophy.

**Cystic dental inclusions** about vestigial supernumerary teeth also occur in juxtamolar positions in cattle, but are insignificant. These too may be dentigerous, or they may be primordial cysts developed before the stage of enamel formation, and hence contain no mineralized tooth structures. Either type of cyst may give rise to ameloblastomas.

A high incidence of dentigerous cysts involving incisors occurs in some sheep flocks in Scotland, Australia, and New Zealand. A congenital disease involving the jaws and teeth of calves in Germany (*odontodysplasia cystica congenita*) is characterized by massive fibro-osseous enlargement of the maxillae and horizontal rami of the mandibles. Some teeth are malformed, misshapen, or absent.

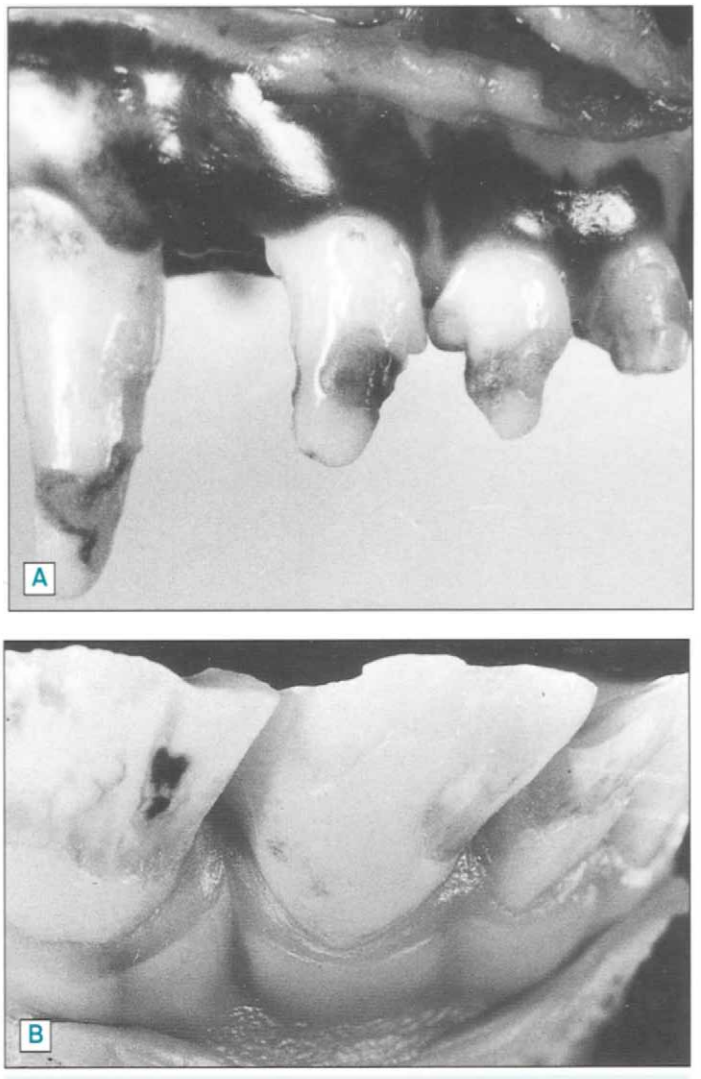



**Figure 1.3 Enamel hypoplasia. A.** A sequel to *Canine distemper virus* infection in a dog. **B.** Subsequent to intrauterine infection by *Bovine viral diarrhea virus* in a calf. (Courtesy of RB Miller.)

Cystic spaces in the jaws are lined by fibrous tissue or epithelium, the latter probably derived from enamel organs. The dental changes are thought to be secondary to those in bone. Most affected calves are aborted or stillborn, and many have ascites and hydrocephalus. The disease may be caused by environmental influences.

The permanent teeth are unique in that they continue to develop for a long time after birth. Thus, inflammatory and metabolic disease of postnatal life, for instance *Canine distemper virus* infection (Fig. 1.3A), can produce hypoplasia of dentin and enamel. Hypoplasia of the enamel of deciduous teeth occurs in some calves with intrauterine *Bovine viral diarrhea virus* infection (Fig. 1.3B). It has also been described in calves and pigs following irradiation of the dam during gestation. Dysplastic proliferation of dentin and enamel involving mandibular premolars and molars has been seen in young uremic dogs. Extreme fragility of deciduous teeth is a feature of bovine osteogenesis imperfecta (see Vol. 1, Bones and joints). Dental dysplasia, characterized by normal dentin, absence of enamel matrix, and excess irregular cementum, is described in a foal with epitheliogenesis imperfecta involving the oral mucosa.

## *Degenerative conditions of teeth and dental tissue*

### Pigmentation of the teeth

Normal enamel is white and shiny, but normal cementum is off-white to light yellow, and normal dentin is slightly darker yellow. Depending on the tooth, or the part of the tooth being examined, the normal color may be any one of these. Normal enamel is never discolored. Hypoplastic enamel of chronic fluorosis is discolored yellow through brown to almost black. Discoloration of brachydont teeth results from pigmentation of dentin, which is then visible through the semitransparent enamel, or pigmentation of the cementum of the root. Dentin may be colored red-brown by pulpal hemorrhages or inflammation, gray-green in putrid pulpitis, and yellow in icterus. Congenital erythropoietic porphyria of calves, cats, and swine discolors the dentin red in young animals (pink tooth) and darker brown in adults, although in swine, the discoloration may disappear with aging. Transient porphyria with pink discoloration of teeth is reported in a dog.

Yellow to brown discoloration of teeth, and bright yellow fluorescence in ultraviolet light, due to deposition of *tetracycline* antibiotics in mineralizing dentin, enamel, and probably cementum, occurs in all species. Treatment of the pregnant dam may cause staining of deciduous teeth in the offspring. Tetracyclines are toxic to ameloblasts in late differentiation and early secretory stages and, at high dose rates, may produce enamel hypoplasia. Black discoloration of ruminant cheek teeth is extremely common, and is caused by impregnation of mineral salts with chlorophyll and porphyrin pigments from herbage.

### Dental attrition

*Dental attrition is loss of tooth structure caused by mastication.* The mature conformation of teeth is largely the outcome of opposed growth and wear, and the degree of wear depends on the type of tooth, the species of animal, and the material chewed. Wear is most evident in herbivores, and irregularities of wear are perhaps the most common dental abnormalities. In general, with normal occlusion and use, the extra-alveolar portion of the tooth does not shorten. Its length is maintained initially by growth, the period of growth depending on the species, then by hypertrophy of the root cementum and/or dentin and by proliferation of alveolar bone, which serves to push the tooth out. Finally, senile atrophy of the alveolar processes and gingival recession may maintain or increase the length of the clinical crown. Cementum hypertrophy and alveolar atrophy may also result in loss of teeth in senility, or, if combined with subnormal wear, produce teeth that in old age are excessively long. Normal wear of the complicated cheek teeth of horses and cattle causes smoothing of the occlusal surfaces. As soon as wear of enamel exposes the dentin, which, being softer, wears more rapidly, secondary or irregular dentin is deposited to protect the pulp. In time, this may fill the pulp cavity and cause death of the tooth. Abnormalities of wear are most common in herbivores (Fig. 1.4). Excessive wear of the deciduous and permanent central incisors occurs in certain sheep flocks in New Zealand. The wear is intermittent and may be severe enough to expose the pulp cavity. The cause is unknown but may be related to delayed eruption of adjacent teeth, leading to increased use of the affected pairs.

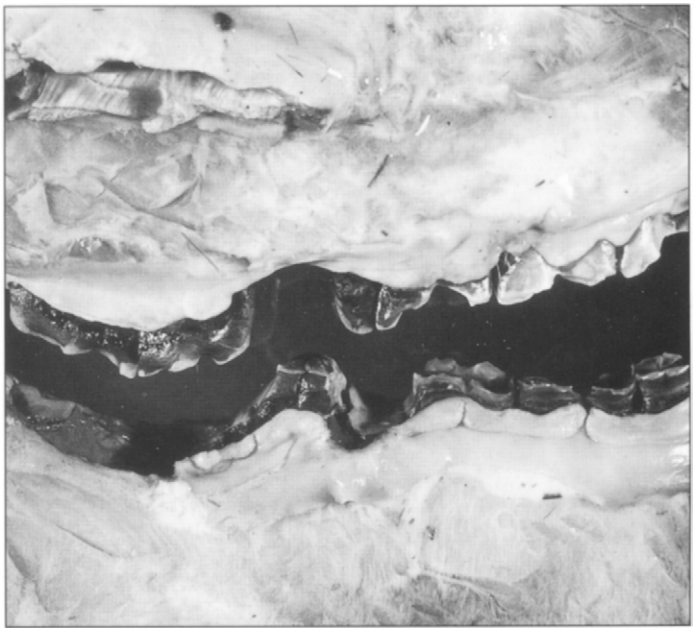


**Figure 1.4 Irregular wear** of the teeth of a horse.

**Subnormal wear**, due to loss of the opposing tooth, occurs in oligodontia, abnormal spacing of adjacent teeth, and acquired loss of teeth; it results in *abnormal lengthening*. Such elongated teeth may grow against the opposing gum or, if deviated, into an adjacent soft structure such as cheek or lip. These teeth usually wear in abnormal places because complete loss of antagonism is unusual, since the upper and lower arcades do not coincide exactly, and the coincidence is further reduced by the displacement of chewing. Incomplete longitudinal coincidence of the molar arcades allows irregular wear and hook formation on the first and last cheek teeth.

**Abnormal wear** due to abnormal chewing is caused by voluntary (as in painful conditions) or mechanical impairment of jaw movement. Lateral movements of the jaws without the normal rotary grinding movements allow the ridges of the teeth of herbivores to become accentuated. Steep angulation of the occlusal surfaces results from inadequate lateral movement of the jaws, and sharp edges form on the buccal aspect of the maxillary teeth and the lingual aspect of the mandibular teeth. This may be unilateral when the animal chews with only one side of its mouth, the other side then being affected. The teeth wear progressively sharper, and pass each other like shear blades; hence the term **shear mouth**. Subnormal resistance to wear on the part of the molar teeth is common, and results in **wave mouth** ("weave mouth") or **step mouth**, in which successive teeth in an arcade wear at different rates. The wave or step form of the antagonistic arcade is reversed, so that the teeth of the two arcades interdigitate. This pattern of attrition is caused by variation in the hardness of opposing teeth, and is usually caused by intermittent odontodystrophy. Opposing teeth of the upper and lower jaws do not develop at the same time; thus, discontinuous nutritional deficiencies often result in unequal wear. Certain vices, such as crib biting, also produce abnormal wear. In severely worn ruminant incisors, a central black core may be visible, which is secondary dentin deposited in the pulp cavity. It is not carious, but stains darker than the surrounding primary dentin.

## Odontodystrophies

*Odontodystrophies are diseases of teeth caused by nutritional, metabolic, and toxic insults.* They are manifest by changes in the hard tissues of the teeth and their supporting structures. Lesions of enamel and dentin are emphasized here. The most prominent effects of odontodystrophies appear in enamel, and lesions of enamel are most significant because they are irreparable.

Formation of enamel occurs in a set pattern. It begins at the occlusal surface and progresses toward the root. Mineral maturation occurs in the same sequence, but for each level, it begins at the dentin–enamel junction and moves toward the ameloblast. Deleterious influences have their most severe effects on those ameloblasts that are forming and mineralizing enamel. Depending on the severity on the insult, ameloblasts may produce no enamel, a little enamel, or poorly mineralized enamel. Removal of the insult permits those ameloblasts that were not yet active to begin making normal enamel. Thus, enamel defects vary in severity from isolated opaque spots or pits on the surface to deep and irregular horizontal indentations. These defects are most clearly seen on the incisor teeth and canine teeth and are usually bilaterally symmetrical. Similar lesions are also produced by infectious agents that injure ameloblasts, such as *Canine distemper virus* and *Bovine viral diarrhea virus* (Fig. 1.3).

Odontoblasts are susceptible to many of the same influences as ameloblasts, but they can be replenished from the undifferentiated cells of the dental pulp. Thus, lesions in actively forming dentin may be repaired, whereas those in enamel are permanent.

Because of their close anatomical association with the jaws, teeth are very susceptible to disruption in the harmony of growth. This harmonious arrangement is often upset in the odontodystrophies and osteodystrophies, and leads to malocclusion and anomalous development of teeth.

Several nutritional and toxic conditions produce odontodystrophy. **Fluorine poisoning** is exemplary (see Vol. 1, Bones and joints). In **vitamin A deficiency**, ameloblasts do not differentiate normally, and their organizing ability is disturbed. As a result, odontoblastic differentiation is abnormal. Several lesions develop, including enamel hypoplasia and hypomineralization, cellular, vascularized dentin (osteodentin), and retarded or obviated eruption.

**Calcium deficiency** retards eruption, and causes enamel hypoplasia and mild dentin hypoplasia. Teeth formed during the period of deficiency are very susceptible to wear. In sheep, recovery from prolonged calcium deficiency results in malocclusion due to inferior prognathia. This reflects inadequate maxillary but normal mandibular repair during the recovery phase.

**Phosphorus deficiency**, combined with vitamin D deficiency, depresses dentin formation slightly, but has virtually no effect on enamel, at least not in sheep. Hypophosphatemia is associated with formation of interglobular dentin in humans. Malocclusion and abnormalities of bite in rachitic sheep are secondary to mandibular deformity.

Severe, experimental **malnutrition** also produces malocclusion. Recovery from malnutrition does not correct the lesion, and in addition, is associated with misshapen, malformed teeth, oligodontia, and polyodontia.

The major **effects of odontodystrophies** in herbivores are *malocclusion*, and/or accelerated *attrition*. Sometimes a high incidence of these abnormalities is attributable to one of the causes previously discussed, but often they are idiopathic. Most of the lesions described in experimental odontodystrophies also occur in natural diseases.

A syndrome of dental abnormalities of sheep in the North Island of New Zealand is characterized by excessive wear of deciduous teeth, maleruption and excessive wear of permanent teeth, periodontal disease involving permanent teeth, and development of dentigerous cysts involving permanent incisors. Mandibular osteopathy is also present. Animals older than 5 years are culled for dental problems. The odontodystrophy (and osteodystrophy) is possibly caused by deficiencies of calcium and copper, and perhaps other nutrients, such as protein, and energy. This syndrome exemplifies the naturally occurring odontodystrophies, in that it probably has a complex pathogenesis, and is associated with an osteodystrophy. The latter association is to be expected, since *bones and teeth are usually susceptible to the same insults.*

While dental lesions are not described, tooth loss due to periosteal dysplasia and osteopenia occurs in Salers cattle afflicted with hereditary hemochromatosis.

## *Infectious and inflammatory diseases of teeth and periodontium*

The role of viruses in enamel hypoplasia is mentioned above. Bacterial plaque, along with other tooth-accumulated materials, is discussed here.

**Bacterial diseases involving tooth surfaces** are caused by the development of supragingival and subgingival **plaque**. *Supragingival plaque* is located on the exposed crown of the tooth and causes dental caries. *Subgingival plaque* is found in the crevicular groove and causes periodontal disease. Tooth enamel is covered by a translucent pellicle, the *acquired enamel pellicle*, which is formed by selective adsorption of complex salivary proteins, and that is essential to the development of supragingival plaque. This is a dense, nonmineralized, bacterial mass, firmly adherent to tooth surfaces, which resists removal by salivary flow and prevents the buffering capacity of saliva from influencing plaque metabolites. Formation of this plaque involves adhesion of bacteria to the pellicle, and adhesion of bacteria to each other, producing a *biofilm*. Only organisms with the ability to adhere to the pellicle can initiate the formation of supragingival plaque; those that cannot are removed by oral secretions and mechanical action. Pathologic reduction of salivary flow increases the prevalence of caries in some species.

The bacteria in supragingival plaque are members of the indigenous oral flora and are usually gram-positive aerobes. Most are streptococci and *Actinomyces* spp., which form an organized array on the tooth surface. Some plaque-forming bacteria synthesize extracellular polymers, which constitute the matrix of the plaque and permit adhesion between organisms of the same species. Some utilize polymers derived from host secretions to adhere to the pellicle, whereas others attach to bacteria of a different species that are already fixed to the tooth. Plaque increases in mass with time, and its composition becomes more complex as anaerobic gram-negative bacteria join the streptococci and actinomycetes that initiated plaque formation.

Supragingival plaque is metabolically active. It utilizes dietary carbohydrates to produce the adhesive polymers and the acids needed to demineralize enamel, and as energy sources for maintenance and for production of various enzymes and stimuli for inflammation. Extensive deposits of supragingival plaque are virtually invisible unless treated with a disclosing solution.

Subgingival plaque is less organized than supragingival plaque, and many of the organisms involved are gram-negative anaerobes that are asaccharolytic, weakly adherent, and motile. They derive their nutrients from the crevicular fluid. The flora of subgingival plaque is less well characterized than that of supragingival plaque. Culture results vary with sample collection technique, site of collection, and selectivity of media, and appear to under-represent the flora detected by molecular means in humans. At least *Porphyromonas* spp. (formerly *Bacteroides*) and spirochetes are likely periodontal pathogens in animals.

**Dental calculus** (*tartar*) *is mineralized plaque.* It is formed by the deposition of mineral, mainly from saliva, in dead bacteria. In horses and dogs, calculus is predominantly calcium carbonate. Calculus is often found in old dogs and cats, occasionally in horses and sheep, and rarely in other species. The distribution is often uneven, but it is *usually most abundant next to the orifices of salivary ducts*. Calculus on horses' teeth is chalky and easily removed. In dogs, it is hard, firmly attached, and often discolored. Red-brown to black calculus with a metallic sheen develops in pastured sheep and goats. It usually involves all the incisors, principally on the neck of the buccal surface. Minor amounts are common along the gum–tooth junction of the molar teeth, but occasionally larger (up to 2 cm) hard, black, rounded concretions may protrude from between opposed surfaces of the premolars. A high prevalence of calculus in sheep on the Scottish island of North Ronaldsay was related to their predominantly seaweed diet. Calculus was most severe around the cranial cheek teeth, increased in severity with age, was associated with periodontal disease, and contained large amounts of calcium, magnesium, and phosphorus.

**Materia alba**, which adheres to teeth, is a mixture of salivary proteins, desquamated epithelial cells, disintegrating leukocytes, and bacteria. The bacteria are not organized, and materia alba is easily removed. It is distinct from dental plaque, and from food debris, which also accumulates between uncleaned teeth.

## Dental caries

Dental caries is a disease of the hard tissues of teeth, characterized by *demineralization of the inorganic part and enzymatic degradation of the organic matrix*. **Erosions** of teeth are characterized by removal of hard tissues layer by layer. These definitions permit the inclusion of equine infundibular necrosis as a form of caries (see following sections). Caries is common in horses and sheep but rare in dogs. Cats are commonly subject to caries-like odontoclastic resorptive lesions of uncertain etiopathogenesis.

There are two types of caries:

1. **Pit or fissure caries** develops in irregularities or indentations, which trap food and bacteria, usually on the occlusal surface of the tooth. Plaque is not essential for initiation of this form of caries, of which *equine infundibular necrosis* is an example.
2. **Smooth-surface caries** usually occurs on proximal (adjacent) surfaces of teeth, typically just below contact points, or around the neck, and requires dental plaque for its initiation.

The organic acids, principally lactic, which initiate demineralization, are produced by bacterial fermentation of dietary carbohydrates. In smooth-surface caries, plaque produces the acid and maintains a low pH on the surface of the tooth. Progression of lesions depends on various factors such as salivary pH, hardness and resistance to demineralization of enamel, and frequency of access to carbohydrate. Demineralization of enamel often occurs in the subsurface enamel but progresses to caries only with prolonged exposure to acid. Infrequent exposure allows remineralization of enamel between meals. The enzymes that lyse the organic matrix are probably produced by plaque, but may be derived from leukocytes, for which plaque is chemotactic. Carious enamel loses its sheen and becomes dull, white, and pocked. When dentin is exposed, it becomes brown or black. Dentin is softer and more readily demineralized than enamel, and a pinpoint lesion in enamel may lead to a large defect when the carious process reaches the dentin. Nerve endings have not been identified at the enamel–dentin junction, and the pain of caries is thought to be caused by chemical or pressure changes in the dentinal tubules. Spread of infection along the tubules to the pulp cavity may result in formation of secondary or reparative dentin, pulpitis, or periapical inflammation and tooth loss.

In **horses, infundibular necrosis** develops most often on the occlusal surface of the maxillary first molar. The enamel invaginations (infundibula) in the cheek teeth of horses are normally filled with cementum before the teeth erupt. Filling proceeds from the occlusal surface toward the apex, but often is not completed before eruption. At this time the blood supply is cut off, and ischemic necrosis of any residual cementogenic tissue in the infundibula occurs. The deficiency of cementum is called *hypoplasia*. Rostral infundibula are affected more frequently than are caudal, and the first molar more often than other teeth (Fig. 1.5A).

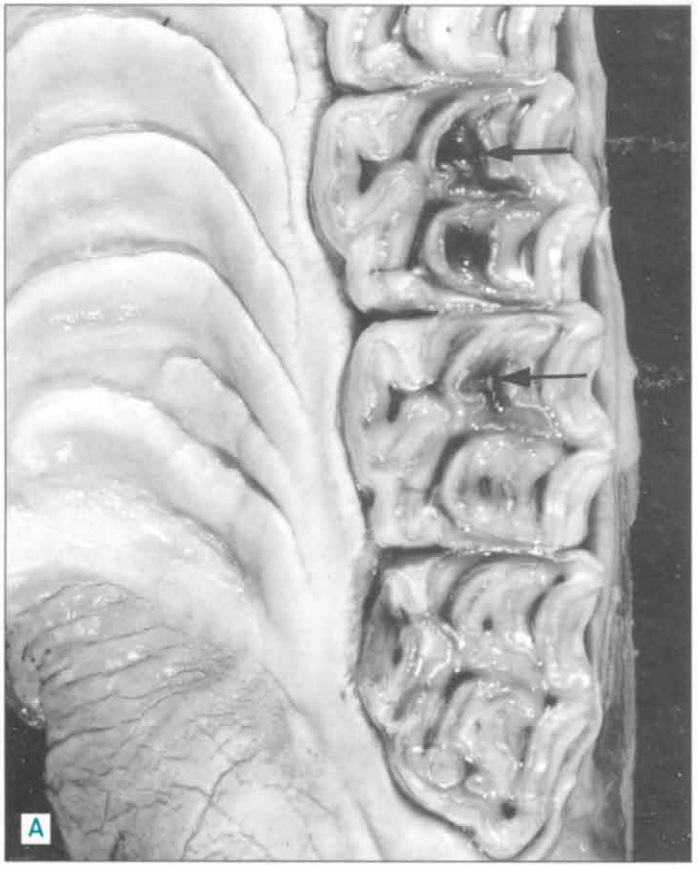
Teeth with incompletely filled infundibula may accumulate food material and bacteria (Fig. 1.5B), and, in some animals, the cavitated area expands to involve all the cementum and the adjacent enamel and dentin. Decay of the mineralized tissues sometimes progresses to coalescence of the cement lakes, fracture of the tooth, root abscess, and empyema of the paranasal sinuses. The incidence of infundibular necrosis increases with age, and 80–100% of horses older than 12 years may have the lesion. Most are without signs, and in most, the lesion does not progress. Inflammation of the dental pulp, in horses and in other species, may result from direct expansion of caries from penetration of bacteria and bacterial degradation products along the dentinal tubules. Production of reparative dentin in the pulp cavity is expected.

In **sheep**, the proximal surfaces of mandibular teeth are usually affected by caries, which is commonly accompanied by periodontitis. Erosions of the neck region of the deciduous teeth occurred in sheep in New Zealand. The lesions were mainly located apical to the enamel–dentin junction on the labial or lingual surface. They did not seem to be related to the usual causes of localized tooth destruction.

**Cattle** develop loss of dentin just below the crown of incisor teeth at increasing frequency with age. This usually follows recession of the gingiva, and is not considered to be a form of caries, but proteolytic digestion of dentin by chyme in an alkaline pH.

In **dogs**, caries most commonly involves the fourth premolar and the first and second molars. Although relatively uncommon, when caries occurs, defects are often multiple and advanced, leading to therapeutic extraction.

**Cats**, whose teeth do not have centers where food can collect, very commonly develop multiple caries-like **odontoclastic resorptive lesions**, initially involving the subgingival neck or upper root,



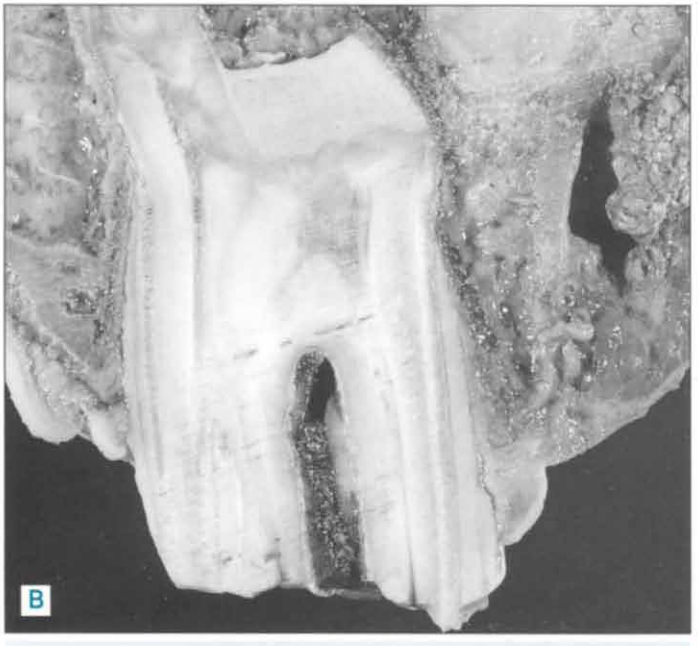




**Figure 1.5 A. Infundibular necrosis** of first and second maxillary molars (arrows) in a horse. Necrosis is confined to cement lakes. **B.** Section through (**A**) showing black discoloration of infundibulum.

most often of cheek teeth, and increasing in prevalence with age. A reddened swollen area of gingiva or granulation tissue often lies over the lesion, which may be on the labial or buccal aspect, and frequently is painful to touch. The resorptive lesions begin as shallow defects in the cementum, lined by odontoclasts, facing a somewhat disorganized periodontal ligament. They progress into the underlying dentin, and, with time, into the root canal. Either or both coronal and apical extension of lesions may occur. Extension of the process coronally more superficially undermines the enamel, which is resorbed or breaks off, causing destruction or loss of the crown. Extension of the dentinal lesion apically leads to resorption of the root. Remnants of the root may persist, often overgrown by gingiva, following loss of the crown. Conversely, destruction primarily of the root may result in obliteration of the periodontal ligament, resorption of adjacent alveolar bone, but with odontoalveolar ankylosis by reparative hard tissues, retaining the crown in the dental arcade. In addition to the odontoclasts that line the resorbing cementum or dentinal surfaces of the lesion, more advanced defects contain a mixed leukocyte population, macrophages, and disordered granulation tissue. Repair is often superimposed, with cementoblastic or osteoblastic cells producing new mineralized tissue of varied osteoid, bone, cementum, or osteodentin morphology. Pulpitis may occur in the affected root canal, and reparative dentin may be deposited there. The prevalence of such lesions has increased markedly in the past 40 years, suggesting an association with changes in form of diet, but the lesion is idiopathic, with no clear relationship to periodontitis, mechanical trauma, viral infections, nutritional or metabolic disturbances. However, it is suggested that lack of masticatory stimulation may lead to loss of the functional integrity of the periodontal ligament, triggering the lesion.

## Pulpitis

The dental pulp is derived from the dental papilla. It is surrounded by odontoblasts and dentin, except at the apical foramen, through which vessels and nerves pass. Pulp is a loose syncytium of stellate fibroblasts, and contains histiocytes and undifferentiated mesenchymal cells. The latter are odontoblastic precursors.

The apical foramen is narrow, and this predisposes to vascular occlusion, ischemic necrosis of the pulp, and death of the tooth. Production of abundant secondary dentin and reparative dentin can cause occlusion, but the usual cause is inflammation. Normally, pulp is the only vascular tissue of the tooth, and, along with the periodontium, the only site of conventional inflammation. *Pulpitis is always related to infection*, the effector bacteria or their products entering through the surface of fractured teeth, carious perforations (especially in teeth with enamel defects), perforations resulting from abnormal wear or trimming, from periodontitis, and possibly hematogenously. In herbivores, in which the pulp is divided by enamel foldings, inflammation is usually limited to one division, and is usually purulent. Very mild pulpitis may heal, but usually it terminates in tooth necrosis, periapical abscessation, perhaps with fistula formation, osteomyelitis, or gangrene as inflammation of the pulp extends to the periodontium and the jaws.

*Periapical abscess and osteomyelitis of the jaws* are complications of pulpitis that may follow clipping the tusks ("needle teeth") of piglets. Trimming of the incisor teeth of sheep to avoid the effects of broken mouth often exposes the pulp cavity, but the pulpitis that ensues is rarely chronic. The exposed pulp canal is healed in 30–50 days by deposition of reparative dentin and secondary dentin. Similar healing presumably occurs in most piglets. *Maxillary (malar) abscess of dogs* involves the periapical tissues usually of the carnassial tooth, and may cause a discharging sinus beneath the eye. The pathogenesis of

the abscess is obscure, but it may be a sequel to crown fractures or to pressure necrosis of periapical tissues. Some chronic inflammations of the pulp are confined to the periodontium and become slowly expansive spherical granulomas about the root apex (*root granulomas*). Occasionally these granulomas are enclosed by an epithelial cyst (*periodontal cyst*) derived from cell rests of Malassez. The epithelium contains plasma cells, and the combination may have a protective role in periapical sepsis.

## Periodontal disease

Periodontal disease is the *most common dental disease of dogs and sheep*, and an important problem in other ruminants, horses, and cats. Although there are minor differences among species, in general, periodontal disease *begins as gingivitis associated with subgingival plaque*, and may progress through gingival recession and loss of alveolar bone to chronic periodontitis and exfoliation of teeth.

The gingival sulcus, or crevice, is an invagination formed by the gingiva as it joins with the tooth surface at the time of eruption. Clinically normal animals have a few lymphocytes, plasma cells, and macrophages under the crevicular epithelium of the gingiva, which forms the outer wall of the crevice, and under the junctional epithelium, which is apposed to the enamel of the tooth.

Clinical **gingivitis** is usually initiated by accumulation of plaque in the crevice, but may be associated with impaction of feed, especially seeds, between teeth. Gingivitis is initially characterized by increased numbers of leukocytes and fluid in the gingival crevice, and then by acute exudative inflammation and accumulation of neutrophils, plasma cells, lymphocytes and macrophages in the marginal gingiva. If the disease progresses, marked loss of gingival collagen occurs in a few days, probably related to the activity of prostaglandins and matrix metalloproteinases generated in inflamed tissue, or possibly enzymes from plaque bacteria, such as *Porphyromonas gingivalis*, which also produce enzymes (gingipains) thought to damage junctional epithelium. *Porphyromonas* spp. are implicated as obligate pathogens for canine gingivitis, and as probable participants in feline gingivitis/periodontitis. Grossly the gingiva is red and swollen, due to the hyperemia and edema of inflammation. Acute gingivitis may become quiescent, with lymphocyte aggregations beneath the junctional epithelium. Halitosis is associated with gingivitis in small animals.

Continuation and exacerbations of the inflammation cause apical recession of the tooth–gingiva junction, and resorption of alveolar bone (Fig. 1.6). Alterations in the periodontal flora may be responsible for these exacerbations. A major part of chronic periodontal disease is *resorption of alveolar bone*, which modifies the attachment site of the periodontal ligament. If concomitant bone loss precedes gingival recession, the sulcus is deepened to form a periodontal pocket, which is lined by transformed junctional "pocket" epithelium, and becomes the site of chronic active inflammation. When gingival recession precedes loss of alveolar bone and gingival collagen, pockets do not form, but tooth roots are exposed. In either case, destruction of the periodontium and periodontal ligament, and resorption of alveolar bone, cementum, and root dentin, *lead to exfoliation of teeth*.

Gingivitis is common in **dogs**. Usually it is proliferative, the gingiva being replaced by collagen-poor, highly vascular granulation tissue, which appears as a red, rolled edge next to the tooth. In dogs, gingival pocket formation is quite unpredictable and may be present on one root of a tooth and absent on the other. Bone loss in dogs is often more severe at the bifurcation of two-rooted teeth than in interproximal areas. Resorption of bone is associated with osteitis as the inflammation extends from the periodontium into alveolar bone. In dogs, the premolars and, to a lesser extent, the first molars and central incisors are most severely affected, whereas the second molars and mandibular canines are quite resistant.

**Figure 1.6** Marked gingival recession with exposure of roots of the molar teeth in advanced **periodontal disease** in a dog.

Gingivitis is among the most common veterinary problems in **cats**. In general, it resembles that in dogs.

In **sheep**, periodontal disease may involve all teeth, but the effects are most severe on the incisors, and periodontal disease is a major cause of premature exfoliation. Sheep develop acute gingivitis during tooth eruption, in association with accumulation of subgingival plaque around the tooth. In some sheep, chronic gingivitis involving the lingual aspect of the incisors ensues, and on farms with a high incidence of broken mouth (lengthening of the incisor crown, forward protrusion, and loosening of the teeth), this progresses to chronic active periodontal disease.

**Cara inchada** (swollen face) is an epidemic periodontitis of **cattle**, formerly common in the west-central part of Brazil. Animals of 2–14 months were mostly affected, and herd prevalences of more than 50% were recorded. When progressive, cara inchada causes loss of teeth, leading to malnutrition. It is associated with dental eruption, and ingestion of forage thought to contain low levels of antibiotics derived from soil actinomycetes that permit colonization of the periodontal space by a variety of gram-negative bacteria, including *Prevotella* (*Bacteroides*) *melaninogenica*.

Severe periodontitis and tooth loss are an important part of the syndrome associated with *bovine leukocyte adhesion deficiency*.

The sequelae of suppurative periodontitis are many, being mainly variations on a theme of *osteomyelitis*. The osteomyelitis of actinomycosis is discussed in Vol. 1, Bones and joints. If the mandible is involved, the fistula usually develops on the ventral margin. If the maxillary molars are involved, fistulation may occur into the maxillary sinus. If the premolars are involved, fistulation may develop into the

nasal cavity or externally. In dogs, involvement of the canine teeth may produce internal or external fistulae, and involvement of the maxillary carnassials usually produces a fistula beneath the eye, and orbital inflammation. Fistulation may be prevented for some time, or permanently, by ossifying periostitis over the involved bone. Fistulae in the upper jaw tend to be persistent. In the lower jaw, they may heal, usually with extensive deposition of new bone. Occasionally, especially in horses, chronic mild periodontitis may be confined by the periodontium, which is, however, expanded by granulation tissue to form a root granuloma. Under the same circumstances there may be hyperplastic exostosis of the cementum.

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## Diseases of the buccal cavity and mucosa

### Pigmentation

Melanotic pigmentation is normal and common in most breeds of animals and increases with age. It may be irregular, or the mucosa may be entirely pigmented. Diffuse yellow discoloration may be seen in icterus.

### Circulatory disturbances

*Examination of the mucous membranes* is an essential detail in any clinical or necropsy examination. Pallor may indicate anemia. In cyanosis, the mucosa is dark red-blue. The mucosa are muddy in methemoglobinemia. Acute congestion and cyanosis, associated with ulceration, are common in dogs and sometimes in cats in chronic uremia.

Hemorrhages are indicative of septicemia, and larger ones may accompany local inflammation, trauma, and the hemorrhagic diatheses. Petechiae on the ventral surface of the tongue and frenulum in horses are consistent with equine infectious anemia, or other thrombocytopenic or purpuric conditions. The active hyperemia that gives the diffuse pink coloration to the mucosa in diffuse stomatitis disappears immediately at death, so that at autopsy the inflamed mucosa is disappointingly blanched.

## Foreign bodies in the oral cavity

*The presence of feed in the mouth of a cadaver is abnormal*, except in ruminants, which may eructate and have feed in the caudal pharynx at the time of death. In most cases it is attributable to disease, which results in paralysis of deglutition or semiconsciousness. It is common in horses with encephalitis, leukoencephalomalacia, and hepatic encephalopathy. The food in such cases is usually poorly masticated and readily differentiated from that refluxed postmortem. Bones or other large foreign bodies lodged in the pharynx of cattle suggest pica of phosphorus deficiency. They may cause asphyxiation or pressure necrosis in the wall of the pharynx. Large portions of root crops may also lodge in the pharynx. Dogs often have bones and sticks that tend to be wedged across the palate behind the carnassial teeth.

In dogs, **foreign-body stomatitis** occurs, caused by plant fibers, burrs, or quills (Fig. 1.7). In mild cases, gingivitis surrounds the incisors and canine teeth. Small papules or vesicles and shallow ulcers may be evident on the tongue. Plant fibers may protrude from the lesions. Chronic cases are characterized by *exuberant granulomas* associated with lingual ulceration and gingival hyperplasia with plant fibers deeply embedded in these lesions. Long-haired dogs are especially prone to develop this type of lesion when they attempt to remove plant material that is trapped in their hair coat. The granulomas must be differentiated from neoplasms.

Sharp foreign bodies which cause laceration of the mucosa predispose to necrotic and deep stomatitis. Grass seeds and awns frequently impact between the retracted gingival margin and teeth in periodontitis of ruminants and exacerbate the local initial lesion, perhaps predisposing to the development of osteomyelitis. Horses fed dry triticale hay may develop severe oral ulceration, with masses of awns embedded in the ulcers. The ulcers vary in size from 1 mm to 5 cm in diameter and are mainly located at the junction of the labial and gingival mucosa adjacent to the upper corner incisors, the lingual frenulum, the sublingual folds, the base of the dorsum of the tongue, and the soft palate. Similar lesions in horses have been associated with contamination of hay by foxtail.

Swine have a diverticulum of the pharynx in the caudal wall immediately above the esophagus, and barley awns and other rough plant fibers occasionally lodge here and penetrate the pharynx. This occurs mainly in young pigs, and death follows pharyngeal cellulitis. Similar problems occur in sheep following improper use of drenching guns, and in cattle injured by balling guns.

## Inflammation of the oral cavity

Inflammatory processes of the oral cavity may be diffuse (*stomatitis*) or localized predominantly in certain regions to produce, if (1) the pharynx is involved, *pharyngitis*; (2) the tongue, *glossitis*; (3) the gums, *gingivitis*; (4) the tonsils, *tonsillitis* (Fig. 1.8); and (5) the soft palate, *angina*. Lesions limited to the mucosa of the oral cavity are termed *superficial stomatitides*. Processes seated in connective tissues of the mouth, the *deep stomatitides*, are usually sequelae to transient superficial lesions.

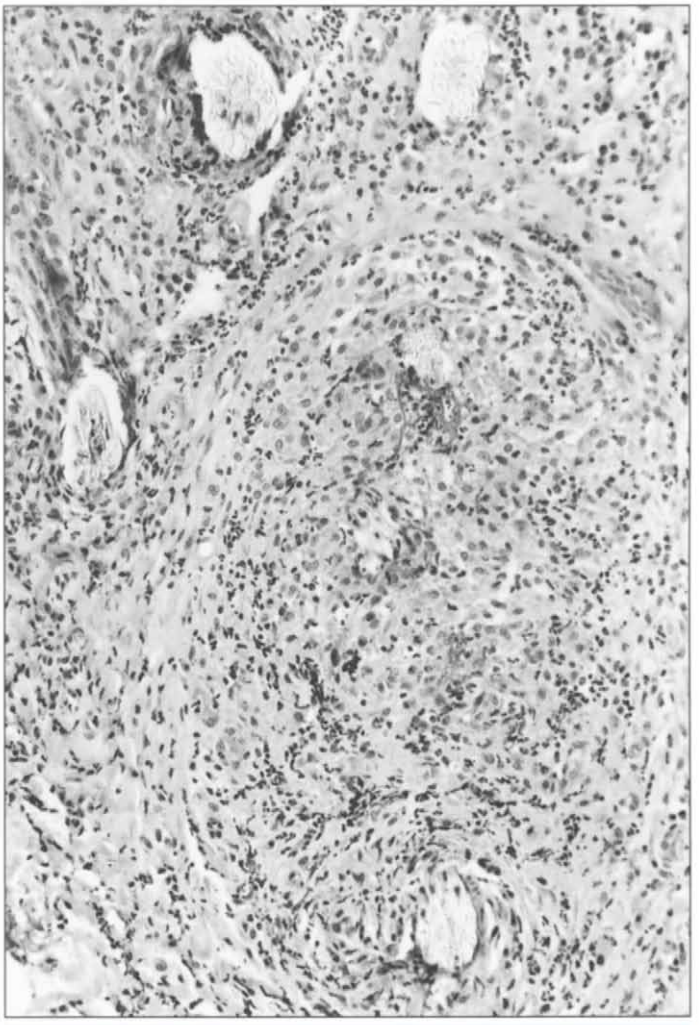

**Figure 1.7** Granulomatous reaction to plant material in the tongue of a dog: **foreign-body glossitis** or "burr tongue."

### Superficial stomatitis

Inflammatory changes may be associated with ingestion of irritating chemicals such as caustic or toxic compounds. An example is paraquat, a herbicide, which may cause severe erosive stomatitis in dogs. Dogs and cats that chew on the plant *Dieffenbachia* may develop oral erosions and ulcers. Electrical burns are occasionally seen in puppies or kittens that chew through electrical wires. It is often not possible to differentiate the cause of diffuse stomatitis, but an attempt to do so is important because it may indicate a systemic disease state. Viral diseases causing stomatitis will be considered in detail in the section on Infectious and parasitic diseases of the alimentary tract, later in this chapter.

Inflammatory disease, localized to the buccal cavity, and not part of systemic viral disease, is also common and important. It is generally due to the indigenous bacterial flora. The oral microbiota

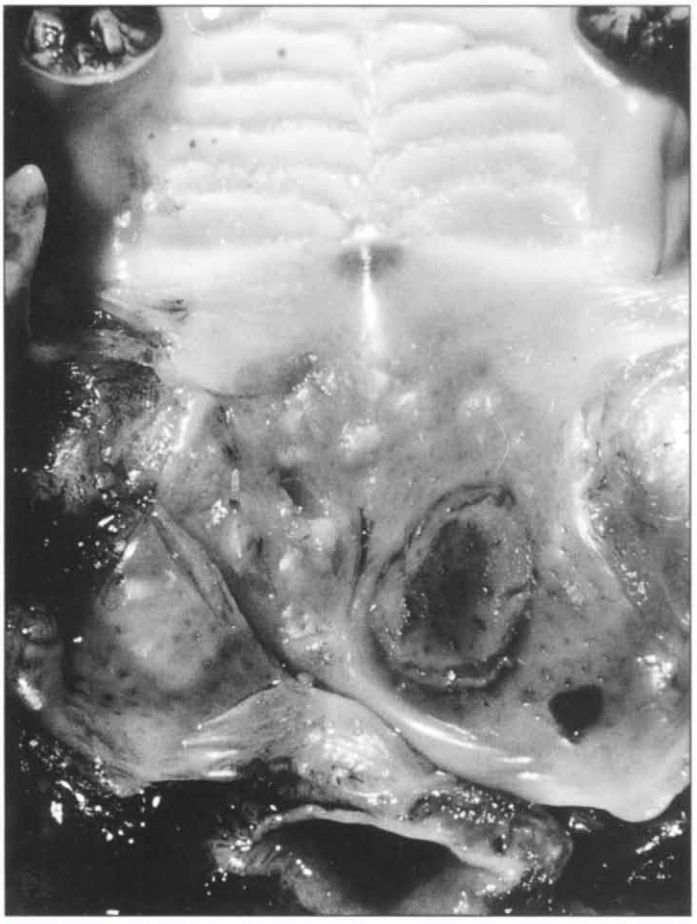

**Figure 1.8** Necrotic palatine **tonsillitis** in a pig.

ordinarily contains many microbial species, mainly anaerobes such as *Actinomyces, Fusobacterium*, and spirochetes, which exist in balance with each other and in harmony with the host. Disruption of this microfloral balance may lead to stomatitis. The oral mucosa is quite resistant to microbial invasion for several reasons. These include the squamous mucosal lining; antibacterial constituents of saliva such as lysozyme; immunoglobulins, especially immunoglobulin A (IgA), in oral secretions; and the presence of a rich submucosal vascular network and inflammatory cells. Factors altering the balance of indigenous organisms are not well delineated. Systemic illness, stress, and nutritional and hormonal imbalances may alter the microbial population by altering the amount, composition, and pH of saliva. The integrity of the oral epithelium depends on a high rate of epithelial regeneration to balance loss due to a high rate of abrasion and desquamation. Rapid epithelial replication promotes quick healing of superficial lesions.

The lamina propria of the oral epithelium is well vascularized, but generally dense and relatively inelastic. For this reason, there is little distension of lymphatics and tissue spaces with fluid exudate, and therefore, swelling due to edema is not a significant part of stomatitis involving hard palate and gums, with the exception of the gingival margins.

## Catarrhal stomatitis

*Catarrhal stomatitis is superficial inflammation of the oral mucosa*, which usually involves the caudal fauces and may be associated with mild gingivitis. It is a common nonspecific lesion, which often develops in the course of debilitating diseases. The mucosae are hyperemic, and the loose texture of the submucosa in the fauces permits development of edema. The swelling is aggravated by edema and hyperplasia of the abundant lymphoid tissues of the soft palate, tonsil, and pharyngeal mucosa. The epithelium accumulates, producing a dull gray mucosal surface. Palatine glands produce excessive mucus. Catarrhal stomatitis resolves with the return of normal oral function.

**Thrush**, or **oral candidiasis**, occurs most commonly in foals, pigs, and dogs. It involves the proliferation of yeasts and hyphae in the parakeratotic superficial layers of the oral epithelium. It appears grossly as patchy pale-gray pseudomembranous material on the oral mucosa and back of the tongue, and probably reflects alterations in epithelial turnover and oral flora (see section on Infectious and parasitic diseases of the alimentary tract, later in this chapter). Mold products of *Stachybotrys alternans* cause catarrhal and necrotizing stomatitis, as well as colitis, if feed is contaminated. Gingivitis and ulceration of the oral mucosa may rarely be associated with infection due to *Nocardia* spp. in dogs.

## Vesicular stomatitides

Stomatitis characterized by the formation of *vesicles* occurs in most species of domestic animals. The vesicles develop as accumulations of serous fluid within the epithelium or between the epithelium and the lamina propria. These may coalesce to form *bullae*, and the elevated epithelium is easily rubbed off during chewing to leave raw eroded patches with bits of epithelium adherent. *The transition from vesicle to erosion occurs rapidly*, so that, in individual animals, vesicles may not be seen. This is especially so in dogs and cats because the oral mucosa is very thin. Because the basal epithelium or basement membrane remains intact, regeneration and healing are complete in a few days unless the local lesions are complicated by bacterial or mycotic infections. However, foci of previous erosion may be identifiable for some months by their slight depression and lack of pigmentation.

Traditionally, vesicular stomatitides in animals were associated with *viral infections*, and these are still important causes. Vesicular stomatitis and foot-and-mouth disease are associated initially with vesicle formation; however, rinderpest, bovine viral diarrhea and malignant catarrhal fever produce sharply demarcated erosive/ulcerative lesions without initial vesicle formation. *Oral erosions and ulcers in horses, ruminants, and swine should be regarded as indicating one of the vesicular diseases to which the species is susceptible, until proven otherwise* (see section on Infectious and parasitic diseases of the alimentary tract, later in this chapter). Sunburn, photoirritation associated with grazing on celery and related crops, and lesions associated with parvovirus infection in swine may cause lesions of the snout resembling vesicular diseases. Animals exposed to irritant chemicals in feed or bedding may develop vesicles and erosions of the face and oral cavity, for example, toxicity in horses and dogs associated with irritant quassinoids found in wood shavings derived from Simaroubaceae species.

*Bullous immune skin diseases* are recognized with increased frequency, especially in dogs, and some of these have severe oral lesions, which will be described here (see also Vol. 1, Skin and appendages).

**Pemphigus vulgaris** is a severe, acute or chronic, vesiculobullous autoimmune disease mediated by autoantibodies to the desmosome

protein desmoglein 3. It is characterized by acantholysis of the epidermis, which results in formation of flaccid bullae and erosions involving mainly mucocutaneous junctions, oral mucosa and, to a lesser extent, skin. Clinically affected dogs and cats have erosions/ulcerations of the oral mucosa and may drool. The oral lesions are generally more prominent than, and precede, the skin lesions. They are most obvious on the dorsal surface of the tongue, which is bright red, with a few scattered pink raised areas representing islands of normal mucosa. The lesions vary greatly in severity and distribution, although the hard palate is often severely ulcerated. Bullae are rarely seen in the oral cavity, because they ulcerate rapidly.

Microscopically, the earliest lesion consists of *suprabasilar acantholysis*, which is followed by the formation of clefts. These lead to ulceration of the mucosa. The basal cells of the epidermis remain attached to the basement membrane and form a so-called "row of tombstones." A few neutrophils and eosinophils may infiltrate the epithelium. There is a variable lymphocytic and plasmacytic lichenoid reaction in the propria. The presence of suprabasilar clefts and bullae due to acantholysis is considered to be diagnostic of pemphigus vulgaris. However, extensive erosion and ulceration of the mucosa and secondary bacterial infections frequently obscure these clefts and bullae. Several biopsies from different areas of the oral mucosa may be required to demonstrate the characteristic lesions. A presumptive histologic diagnosis should be supported by direct immunofluorescence tests that show autoantibodies (usually IgG) and complement in the intercellular spaces of stratified squamous epithelium.

**Bullous pemphigoid** is a term that has been applied generically to superficial autoimmune vesiculobullous or ulcerative disease of mucous membranes (including the oral mucosa) and skin, characterized by subepithelial clefting; acantholysis is not a feature. It has been reported in humans, horses, dogs, and cats. It is now recognized that there is a *complex of autoimmune subepidermal blistering diseases*, varying in their target antigen, clinical manifestations, and prognosis. Those involving the oral cavity of cats and dogs include bullous pemphigoid, mucous membrane (cicatricial) pemphigoid, and canine epidermolysis bullosa acquisita. All are characterized by circulation of IgG and IgE autoantibodies against specific basement membrane antigens. The characteristic microscopic lesions of all are transient subepidermal blisters, which may contain fibrinocellular exudates with variable numbers of neutrophils and eosinophils. Diagnosis of each is confirmed by the detection of circulating antibody directed at appropriate antigens, using enzyme-linked immunosorbent assay (ELISA) or immunofluorescent tests, or by identification of immunoglobulin fixed to basement membrane. Paraneoplastic bullous stomatitis characterized by subepithelial clefting has been reported in a horse with a hemangiosarcoma.

Bullous pemphigoid is retained as the name for the second most common autoimmune subepidermal blistering disease in dogs and cats. The lesions mainly occur on haired skin, and a minority of cases involve the mucocutaneous junctions or mucosae, including the mouth, which is affected about a third of the time. Microscopically, there is a rich neutrophilic and eosinophilic dermal infiltrate adjacent to and sometimes spilling into the subepidermal bullae. The targets for the autoimmune response are epitopes on canine collagen XVII, a component of the basement membrane.

**Mucous membrane pemphigoid** is the most common autoimmune subepidermal blistering disease of small animals, causing about half of all cases. Adults are predominantly affected, and among dogs, German Shepherds are overrepresented. Typically, subepidermal vesicles in mucous membrane pemphigoid are associated with a relatively sparse inflammatory infiltrate. The antigen targeted by autoantibodies is collagen XVII or laminin-5, and basement membrane-fixed immunoglobulin is detected by direct immunofluorescent or immunoperoxidase staining of formalin-fixed paraffin-embedded tissue.

**Epidermolysis bullosa acquisita** is a rare disease of dogs, representing about 25% of cases of autoimmune basement membrane diseases, with a poor prognosis. The associated autoantibodies are directed against collagen VII. The lesions are most common on skin, and advance rapidly to erosions at points of friction, but the oral epithelium often sloughs extensively. Intact subepidermal vesicles may contain no inflammatory cells, or neutrophils may accumulate at the basement membrane, sometimes forming subepidermal microabscesses. The results of serum or cutaneous immunofluorescent tests resemble those in bullous pemphigoid or mucous membrane pemphigoid, but the autoantibodies may be recognized binding to the lower part of the basement membrane.

The oral lesions of pemphigus vulgaris and of the subepidermal blistering diseases must be differentiated from lesions due to trauma, toxic epidermal necrolysis, drug eruptions, chronic uremia, mucocutaneous candidiasis, and lymphoreticular malignancies.

***Feline calicivirus*** causes mainly a respiratory infection in cats. The disease is complicated by *lingual and oropharyngeal ulcers*, which start out as vesicles. They are 5–10 mm in diameter, smooth, and well demarcated from the surrounding normal mucosa. They occur mainly on the rostrodorsal and lateral surfaces of the tongue and each side of the midline of the hard palate. The palatine lesions are apparently more severe in cats fed dry food. Microscopically, the earliest lesions consist of foci of pyknotic cells in the stratum corneum and superficial stratum spinosum. They progress to foci of necrosis with vesicle formation and subsequent erosion and ulceration of the mucosa. Regeneration of the oral mucosa in the ulcerated areas generally occurs within 10–12 days. A single layer of squamous epithelial cells extends from the margins of the ulcer beneath a layer of exudate. Active viral replication also takes place in the tonsillar crypt epithelial cells, and virus may be recovered from these areas for weeks postinfection. Viral inclusions have not been observed in oral epithelial cells. The virus is isolated from a high percentage of cats with chronic stomatitis. Concurrent infection with *Felid herpesvirus 1* may also occur.

## Erosive and ulcerative stomatitides

This form of stomatitis is characterized by local epithelial defects of the oral mucosa and nasolabium and is usually associated with acute diffuse stomatitis and pharyngitis. *Erosions* are circumscribed areas of loss of epithelium which leave the stratum germinativum and basement membrane more or less intact. They are usually associated with acute inflammation in the underlying propria. The erosions vary in size and shape. Although they are often a nonspecific development in a wide variety of conditions, they are also an essential part of a number of important diseases. They heal cleanly and quickly, but if secondarily infected or complicated, may develop into ulcers.

*Ulcers*, in contrast to erosions, are deeper deficiencies that extend into the substantia propria. They too vary greatly in size and shape;

the edges tend to be elevated and ragged, and when they heal, it is with scar formation.

The causes of ulcerative stomatitis are in general those of erosive stomatitis. There are, however, a number of recognized syndromes and specific diseases in which the predominant change is ulceration. *Phenylbutazone* intoxication in horses may cause oral ulcers in concert with ulcers of the stomach, intestine, and colon; the syndrome is discussed with ischemic diseases of the gut (p. 99).

**Feline ulcerative stomatitis and glossitis**, or **lymphocytic-plasmacytic stomatitis**, is an ulcerative and chronic inflammation of the mucosa of the fauces, the angle of the jaws and, less commonly, the hard palate, gingiva, and tongue. Microscopically, there is chronic active inflammation of the oral mucosa and submucosal connective tissues, dominated by lymphocytes and plasma cells. The syndrome is more common in older cats and may accompany periodontitis. The cause is unknown, but is probably multifactorial, involving imbalance in the oral microbial flora, with predominance of gram-negative anaerobes and spirochetes. Some have reported the isolation of *Feline calicivirus* and *Felid herpesvirus 1* from more cats with lesions of chronic stomatitis compared to those without, but the role of these viruses in the etiology is unresolved. *Feline calicivirus* can persist as a sequel to previous disease episodes and in the face of prior vaccination. *Feline leukemia virus* and *Feline immunodeficiency virus* may predispose some cats to chronic stomatitis because of their immunosuppressive effects, but evidence of infection is not consistently found.

**Feline plasma cell gingivitis-pharyngitis** or **feline chronic gingivostomatitis** is characterized by raised erythematous, proliferative lesions, mainly in the glossopalatine arches, extending caudally to the palatopharyngeal arch and rostrally to the gingiva. The lesions may involve Eustachian tubes and also can affect the conjunctiva. Histologically, the mucosa is hyperplastic and frequently ulcerated, with a marked submucosal inflammatory cell reaction, mainly plasmacytes, including binucleate cells and cells containing Russell bodies. Neutrophils, lymphocytes, and histiocytes are scattered among the plasma cells. Affected cats have elevated polyclonal serum gamma-globulin levels. The polyclonal gammopathy and the plasmacytic, lymphocytic reaction are suggestive of an immune-mediated lesion, and differentiation from mucosally associated lymphoid neoplasia may be challenging. The etiology and the relationship of this syndrome to feline ulcerative stomatitis and glossitis (see above) are unclear; the two syndromes probably form a continuum, although the plasma cell predominance and the hypergammaglobulinemia attributed to plasma cell gingivitis-stomatitis are distinguishing. Similar stomatitis occurs in dogs.

**Eosinophilic ulcer** (eosinophilic granuloma, lick granuloma, labial ulcer, rodent ulcer) is a chronic, superficial ulcerative lesion of the mucocutaneous junctions of the lips, and, to a lesser extent, the oral mucosa and skin, in cats of all ages. The cause is unknown but the lesions may respond to corticosteroid, oral progestagens, cryosurgery, or radiation therapy, although recurrences are common. Typically, well-demarcated, red-brown, shallow ulcers, often with elevated margins, occur on the *upper lip on either side of the midline*. They are usually a few millimeters wide and several centimeters long. Occasionally, ulcers are present elsewhere in the mouth, such as on the gums, palate, pharynx, and tongue. Skin lesions are located in those areas that are frequently licked, such as the neck, lumbar area, and abdomen. Microscopically, the squamous mucosa is ulcerated, with large areas of necrosis of the underlying connective tissues and accompanied by a marked inflammatory cell reaction. The cellular reaction consists predominantly of neutrophils at the periphery of the ulcers, with a mainly mononuclear cell reaction (plasma cells and mast cells) in the propria, eosinophils and histiocytes being only occasionally seen. In some cases, the eosinophils and mast cells may predominate, but this difference may be a reflection only of the evolution of the lesion.

Eosinophilic ulcer is one of the three different types of lesions that have been associated with the so-called *eosinophilic granuloma complex*. The other two conditions, eosinophilic plaque and linear granuloma, cause mainly skin lesions, which are different clinically and morphologically from eosinophilic ulcer (see Vol. 1, Skin and appendages).

**Oral eosinophilic granuloma** (linear granuloma) in dogs occurs as a familial disease in young Siberian Huskies. Sporadic cases have been reported in other breeds, especially Cavalier King Charles Spaniels. Affected dogs have single or multiple firm, often ulcerated, raised plaques, which are covered by yellow-brown exudate, on the *lateral or ventral surfaces of the tongue*. Lesions on the soft palate are less common, and here they tend to be oval to circular ulcers with slightly elevated borders. Cytologic preparations made from scrapings of the oral lesions show many eosinophils, a few neutrophils, occasional macrophages, and epithelial cells.

Microscopically, *foci of collagenolysis* (necrobiosis) in the mid and deep zones of the lingual submucosa are surrounded by a mainly histiocytic granulomatous inflammatory reaction, with giant cells, lymphocytes, plasma cells, and mast cells. *Eosinophils* are a constant feature, but their numbers vary from few to many. The lesions are identical to those seen in linear granuloma of cats.

The cause is unknown, although the morphology of the lesion and the response to corticosteroid therapy suggest hypersensitivity. The familial tendency in Siberian huskies indicates that hereditary factors are involved. *Eosinophilic granuloma must be differentiated from oral mast cell tumors*, which also affect the tongue in dogs. Necrobiosis of collagen fibers is often a feature of mast cell tumors; however, in mastocytoma, the characteristic mixture of mast cells and eosinophils infiltrates the tongue and connective tissues more diffusely. The mast cells may be in various stages of degranulation, and inflammation is minimal or absent in mast cell tumors.

**Horses** with **eosinophilic epitheliotropic disease** (see Vol. 1, Skin and appendages and section on Eosinophilic enteritis in cats and horses, below) may also have eosinophilic stomatitis and lingual ulceration.

**Feline viral rhinotracheitis** is a common upper respiratory tract infection of cats caused by *Felid herpesvirus 1* (see Vol. 2, Respiratory system). This virus may cause ulcerative lesions in the mouth, especially on the tongue. Rarely, oral and skin ulcers may occur, without evidence of concurrent respiratory tract infection. Microscopically, foci of cytoplasmic vacuolation in squamous epithelium evolve into areas of necrosis and ulceration. The ulcers are often covered by a layer of fibrinocellular exudate. Herpetic inclusions may be present in epithelial cells at the periphery of the ulcers.

**Uremia** associated with chronic renal disease often causes *fetid ulcerative stomatitis* in dogs, and less commonly in cats. Dirty gray-brown ulcers occur on the gums, lateral surface, and margin of the tongue, and on the inner surface of the lips and cheeks, often adjacent to the openings of salivary ducts. The margins of the ulcers are swollen and hyperemic.

The pathogenesis of the oral lesions in uremia is still poorly understood. Elevations in blood and salivary urea in combination with urease-producing bacteria, normally present in the oral microflora, may generate ammonia from salivary urea. Ammonia has a caustic effect on the oral mucous membranes. Experimental antibody production against urease renders some intestinal bacteria nonpathogenic and prevents uremic colitis, providing evidence of the importance of urease. However, there is poor correlation between the levels of blood urea and the development of uremic stomatitis, suggesting other important factors. Uremic vasculitis and impaired microvascular perfusion may contribute to the pathogenesis of uremic stomatitis.

Salivary glucose levels may be elevated in dogs and cats with *diabetes mellitus*, resulting in an imbalance of the oral microflora and predisposing to chronic gingivitis in diabetic animals.

Ulcerative glossitis and stomatitis in **swine** is commonly part of **exudative epidermitis** (greasy pig disease) of preweaning pigs (see Vol. 1, Skin and appendages). In addition to the characteristic skin lesions, about a third of the piglets may develop ulcers on the dorsum of the tongue. Erosions and ulcers of the hard palate occur in a small number of piglets. Microscopically, there is ulceration of the squamous mucosa with coagulative necrosis, and vesicle and pustule formation in the superficial epithelium over the rete pegs. A pleocellular inflammatory reaction is evident in the connective tissue below the ulcers.

Oral erosions and ulcers have been reported in pigs with *congenital swinepox*. Microscopically, there is swelling and degeneration of squamous epithelial cells with numerous eosinophilic intracytoplasmic viral inclusions. The central areas of the lesions are necrotic. There is a mixture of lymphocytes, neutrophils, eosinophils, and histiocytes in the submucosa. Poxvirus may be demonstrated by electron microscopic examination of the lesions.

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## Deep stomatitides

Lesions of the oral mucosa may permit the entry of pyogenic bacteria, often normal oral flora, into the connective tissues of the submucosa and muscle. Purulent inflammation or cellulitis may develop in the lips, tongue, cheek, soft palate, and pharynx. Abscesses may form and may fistulate through the mucosa or skin. Abscesses in the wall of the pharynx may result from necrosis of retropharyngeal lymph nodes. Necrotic stomatitis with simple necrosis of the epithelium and lamina propria may be produced by thermal or chemical agencies, but in animals, it is usually caused by *Fusobacterium necrophorum* and other anaerobes.

**Oral necrobacillosis** *Fusobacterium necrophorum* is the principal cause of **oral necrobacillosis** or **necrotic stomatitis** in animals. It is also associated with necrotizing lesions elsewhere in the upper and lower alimentary tract, and liver. Wherever it occurs, it is usually a *secondary invader* following previous mucosal damage. The organism produces a variety of exo- and endotoxins; among the latter are leukocidins, hemolysins, and a cytoplasmic toxin, all of which probably enhance the necrotizing ability of the organism. Once established in a suitable focus, *F. necrophorum* proliferates, causing extensive coagulative necrosis.

The best-known form of necrobacillary stomatitis is **calf diphtheria**, an acute necrotizing ulcerative inflammation of the buccal and pharyngeal mucosa, and also of the laryngeal mucosa (**necrotic laryngitis**). The predisposing lesions may include trauma, infectious bovine rhinotracheitis, and papular stomatitis. Necrosis of palatine

and pharyngeal tonsils may be seen. The incidence of diphtheria in slaughtered beef cattle may be as high as 1.4%. The same syndrome is rather common in housed lambs as a complication of contagious ecthyma. The infection may also be initiated in the gums about erupting teeth in any species, and by the trauma produced in baby pigs by removing the needle teeth (Fig. 1.9A). It is frequently fatal in young animals, in which extension often occurs to other organs. In adults, oral necrobacillosis tends to remain localized to the oral cavity, where it may complicate vesicular and ulcerative stomatitides. It is not unusual, however, for the infection to spread down the alimentary tract.

The early lesions are large, well-demarcated, yellow-gray, dry areas of necrosis, surrounded by a zone of hyperemia (Fig. 1.9B). They are found on the sides or dorsal groove of the tongue, on the cheeks, gums, palate, and pharynx, especially the recesses beside the larynx. Primary foci may occur in the laryngeal ventricles. Death may be associated with asphyxia. The necrotic tissue projects slightly above the normal surface and is friable but adherent and is not easily detached. In time it may slough and leave deep ulcers, which may heal by granulation. The necrotic tissues are histologically structureless and are surrounded at first by a zone of vascular reaction, later by a dense but narrow rim of leukocytes, and finally by thick encapsulating granulation tissue. The bacteria are arranged in long filaments, particularly at the advancing edge of the lesions. The submucosal extension of the lesions may take them deeply into the underlying soft tissues and bone.

Spread from the oral foci occurs down the trachea (causing aspiration pneumonia), down the esophagus, and via blood vessels. Death may occur acutely in septicemia with only multiple small serosal hemorrhages as evidence, or metastases may occur in other tissue. Venous drainage from the face to the vascular sinuses of the meninges may lead to pituitary and cerebral abscessation.

*Fusobacterium necrophorum* has also been associated with a *syndrome of necrotic stomatitis, enteritis, and granulocytopenia in calves.* Affected calves have nonregenerative anemia, leukopenia, neutropenia, hypoproteinemia, and increased fibrinogen levels. In addition to the characteristic oral lesions, there is marked depletion of lymphoid tissues and necrotic enteritis. *Fusobacterium* organisms are present in large numbers in a variety of organs, including the bone marrow. Possibly very virulent strains of *F. necrophorum* produce enough leukotoxins, especially in immunodeficient calves, to suppress bone marrow activity. However, granulocytopenia associated with exposure to bracken or treatment with nitrofurans may also facilitate the development of calf diphtheria.

A gross diagnosis of oral necrobacillosis is ordinarily possible, but may be confirmed by a smear from the margin of the lesion. The organism is difficult to cultivate because it is a *strict anaerobe*.

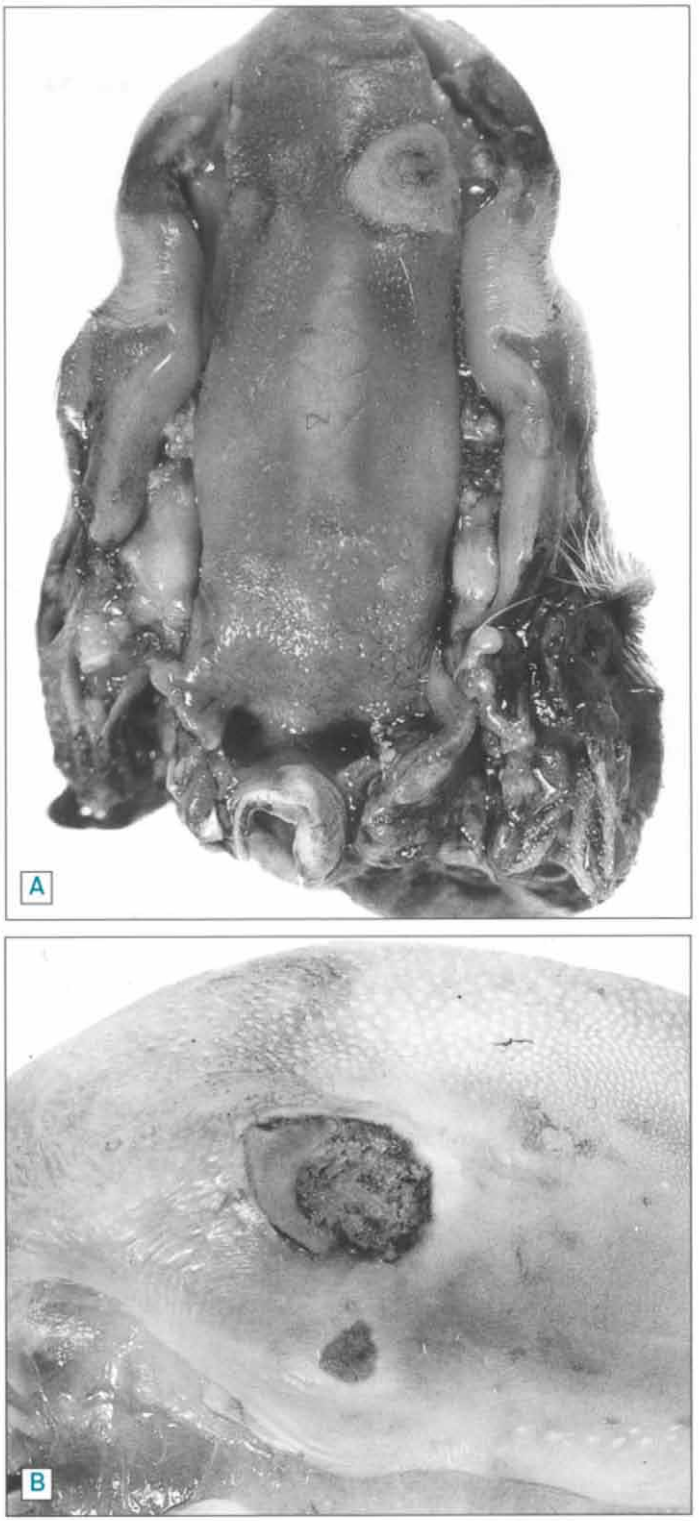


**Figure 1.9 A.** Necrotic **glossitis and stomatitis** in a pig, subsequent to trauma by needle teeth and *Fusobacterium necrophorum* infection. **B. Oral necrobacillosis** in a calf.

**Noma** Noma (cancrum oris) is a rapidly spreading *pseudomembranous or gangrenous stomatitis*; it is not caused by a specific pathogen but is associated with tissue invasion by the normal oral flora, particularly fusobacteria and spirochetes. The predisposing factors are unknown, but they are probably nonspecific and associated with mucosal trauma and debility. The disease, which is occasionally observed in horses, dogs, and monkeys, is in many respects similar to oral necrobacillosis. In the lesions, the spirochetes can be found in large numbers at the advancing margins as well as in peripheral viable tissue. In the deep layers of necrosis, fusiforms predominate, and toward the surface, there is a variety of other organisms, chiefly cocci. The initial lesion is a small tattered ulcer of the cheek or gum, which spreads rapidly and may involve much of the buccal surface of the gums and the mucosa of the cheek. It is intensely fetid and consists of a dirty necrotic pseudomembrane surrounded by a zone