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## **Dental caries and periodontitis in macerated skulls demonstrated in badgers (*Meles meles* LINNÉ 1758) and stone martens (*Martes foina* ERXLEBEN 1777)**

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**Keywords:** dental caries, periodontitis, macerated skulls, badger, *Meles meles*, stone marten, *Martes foina*

**Schlüsselwörter:** Zahnkaries, Periodontitis, Schädelpräparate, Dachs, *Meles meles*, Steinmarder, *Martes foina*

### **Summary**

A short overview on dental caries and periodontitis particularly in macerated skulls is given, with examples of badgers and stone martens. In particular, definitions, etiology, mechanisms, phenology, diagnosis, and differential diagnosis are given for the diseases.

### **Zusammenfassung**

Ein kurzer Überblick Karies und Periodontitis (Definition, Etiologie, Phenologie und Diagnose) insbesondere an Schädelpräparaten von Dachs und Steinmarder wird gegeben.

### **1. Introduction**

In studies of comparative periodontology and cariology of humans and animals, skulls are a frequent material for analysis. Depending on the degree of knowledge and instruction of the scientists involved (biologist, dentist, physician, veterinarian etc. ) interpretation of data varies. The problem is that the pathologic symptoms fixed in a *status praesens* in the skulls must be connected with diagnosis of a disease. Diseases similar in their effect on the bone are hard to differentiate. During maceration, transportation and conservation changes on the material occur which cannot be clearly evaluated (SCHNEIDER 1994). Diagnostic tools include the knowledge of the live animal's normal physiological condition and of the macerated skull. A profound knowledge of the course of diseases of the oral region and the jaw, as well as experience in their effect on bone and soft tissue is required.

It is a common thesis that in badger or stone marten dental caries is frequent. Most often dark brown coloured teeth have lead to this opinion. In fact no caries until now has been found in wild population. The osteolytic changes in the alveolar regions of macerated skulls cannot necessarily be connected with periodontitis, as often done. In this paper an account of dental caries and periodontitis with special reference to two common mustelid species, the Eurasian badger and the stone marten is given.

## 2. Dental caries

### Definition:

Dental caries is the disease of hard tissue of the tooth, i.e. demineralisation of enamel and dentin and bacterial destruction of their organic compounds.

### Etiology:

Caries is a multifactor disease. Responsible for its occurrence is a combination of **bacterial microflora**, a **substrate**, factors of the **host** and **time** (KÖNIG 1987). Regarded in terms of microbiology, caries is an infectious disease.

Dominating microorganisms are *Streptococcus mutans* and *Lactobacillus* specimens. The potential to host a dental caries lies in every microorganism which is capable of fermentation of low molecular carbohydrates. The structure of the oral microflora depends on infection in juvenile age, nutrition (substrate), oral hygiene, tooth position and shape, suppliance of fluoride and immunocompetence of the saliva (host factor).

Caries only occurs in the presence of bacterial plaque. Plaque is formed by a structured layer of 60% – 80% microorganisms and extracellular polysaccharides (EPS) produced by the bacteria and water. The bacteria composition of the plaque depends on the maturation state of plaque. In the first 9 days it changes from cocuss flora to anaerob rod-shaped and spirill flora.

In the course of bacterial metabolism fermentation underneath the plaque leads to the production of lactic acid and the pH level lowers. If the pH value drops below 5.5–5.0, demineralisation of the apathit structure of enamel and dentin starts.

### Mechanism and Phenology:

In proper condition there is a balance of demineralising and remineralising influences on the enamel surface. The crystalline structure of apathit in the enamel consisting of  $\text{Ca}_3(\text{PO}_4)_2$ ,  $\text{OH}^-$  and  $\text{F}^-$  is being remineralised by saliva. A condition for this process is a plaque-free surface, sufficiently mineralised saliva and enough

time. An imbalance towards demineralisation results in the macroscopic appearance of dull white spots in the enamel (initial caries). This is a reversible state and remineralisation often takes place by including coloured particles in the crystals. The result is a brownish spot. If changes in the macrostructure have taken place by loss of apatite prisms caries becomes irreversible. The disease proceeds into the dentin and demineralisation is followed by the enzymatic destruction of the collagen matrix. The destruction depends on the duration and frequency of the acid impacts (time factor) and on the protective capability of the saliva, i.e. its mineral contents and immunocompetence (host factor). There is no possibility of restitution. The tooth can only be saved by excavating the bacterially contaminated substances and filling the tooth with an alloplast.

If bacteria penetrate up to the pulp the soft tissue of a tooth is infected. Pressure necrosis induced by the inflammation destroys the pulp cells. Microorganisms may penetrate through the apical foramen into the connecting bone and cause an inflammation and resorb the bone, then granulation tissue spreads. This can be an acute abscess-forming as well as a chronic procedure.

#### **Diagnosis in the macerated skull:**

Caries regions have an opaque whitish, yellowish or darkbrown discolouring, a disturbed surface and loss of material. Cariogenically changed tissue may also be found underneath a macroscopically intact surface. On the other hand a hardened remineralised initial caries might be mistaken for a defect because of its brown discolouring. Stains on the surface may lead to false interpretation. Exact diagnosis requires a dental explorer. Following the definition of WHO „... dringt die tadellose Spitze der Sonde bei mäßigem Druck eindeutig in den Defekt ein und bleibt stecken bzw. setzt dem Instrument einen gewissen Widerstand entgegen wenn man es abheben und die Sondierung fortsetzen will“ (SAUERWEIN 1974). i.e.; ...the tip of the dental explorer penetrates the surface of the defect while exerting slight pressure and sticks or can only be withdrawn with a slight effort.

Macerated skulls require a careful probing. Maceration and dry conservation cause a brittleness of the hard tissue. Intensive bleaching may change the initial colouring and excessive use of highly concentrated hydrogen peroxide leads to crackings in the surface of the enamel.



Figure 1: Human tooth, vertical section with several carious lesions. Dentin discoloured black and brown, demineralised enamel opaque-white (RATEITSCHAK & WOLF 1994)

### **Differential diagnosis:**

The first point to be mentioned is the colouring of hard tissue after tooth fractures with opening of the pulpal cavity. An injured pulp gives way to contamination resulting in the destruction of the pulpal tissue. Biodegradation of tissue and blood cells causes a grey or brown colouring of the dentin without any demineralisation or loss of hard tissue material.

### **3. Periodontitis (parodontitis marginalis)**

#### **Definition:**

An infection and destruction of all parts of the parodontium i.e. the gingiva, the desmodont (parodontal ligament), the root cementum and the alveolar bone (KETTERL 1990) caused by bacterial plaque. Loosening and loss of the tooth is caused by the progressive destruction of the supporting tissue. In most cases the whole dentition is affected.

In 20<sup>th</sup> century literature terminology regarding parodontopathy is without uniformity. Until today there has been no commonly accepted nomenclature throughout the world. It has been suggested to use the definitions of „Deutsche Gesellschaft für Parodontologie“ dating from 1987 (DPG 1987). This definition is strongly influenced by the English terminology of the World Health Organisation (WHO 1978 a, b).

### **Etiology:**

Bacterial plaque is the main cause of the inflammation of the parodontium. Intensive research in the last years has led to the identification of some putative pathogenic bacteria as *Actinobacillus actinomycetem comitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*. Most of the oral microorganisms can be found in healthy subjects as well as in periodontitis subjects, only the ratio of organisms varies. This leads to the conclusion that periodontitis is a case of opportunistic infection.

Pathogenicity of plaque depends on its composition. Number and kind of bacteria vary interindividually and with time of maturation pathogenicity grows. In the first 9 days microstructure of the plaque changes from coccus to rod-shaped, filament, spirochete and spirilla flora and gets more pathogenic while getting more and more anaerobe. Disease process changes from acute to subacute phases and extends in the human over years and decades if not treated. It may lead to loss of the whole dentition.

### **Mechanism and phenology:**

If plaque grows in the gingival region and is not removed within four days, gingivitis will be the consequence. The gingiva turns red, swells and tends to bleed. Bacterial toxins and enzymes penetrate the connective tissue and produce an inflammatory reaction of the host. The swelling causes little pockets in the sulcus region which resist to selfdeposition and mechanical cleaning (toothbrushing). Within the depth of these pockets conditions for anaerobe specimen are most favourable. In the case that periodontitis starts the inflammation proceeds into deeper structures and resorbs the *compacta* of the alveolar cavity. Regression of the bone deepens the pocket, the inflammation becomes a selfconserving process. The periodontal space widens and the tooth loosens. An abscess of the pocket may occur. In the stage of gingivitis a complete restitution is possible. If the inflammation has progressed it can only be halted; a restitution of bone, cementum and desmodontium by itself is not possible. Under what conditions and in what time gingivitis might change to periodontitis is not yet clearly known.

If plaque crystallises with minerals from the saliva, a formation of calculus is being built up, consisting of different types of calciumphosphatecrystallites:  $\text{CaH}(\text{PO}_4)$ ,  $\text{Ca}_4\text{H}(\text{PO}_4)_3$ ,  $\text{Ca}_5(\text{PO}_4)_3$  and  $\text{Ca}_3(\text{PO}_4)_2$ . The calculus is inactive in terms of bacterial fermentation. Its rough surface however is a mechanical irritation to the gingiva and a good surface for further adhesion of microorganisms. Usually calculus has a faint yellowish colour and is secondarily tinted by different substances of nutrition. Its consistency is chalk-like or hard as limestone. Subgingival calculus is often referred to as concrement. Since there is a lot of blood pigment in the inflamed pockets which integrates in the crystalline structures, concrements are often green or black and very hard.

Formation of plaque and massive calculus in animals such as badgers are not necessarily followed by periodontitis. Therefore it is supposed that these species have a primarily apathogenic oral microflora (DIERKS et al. 1997).

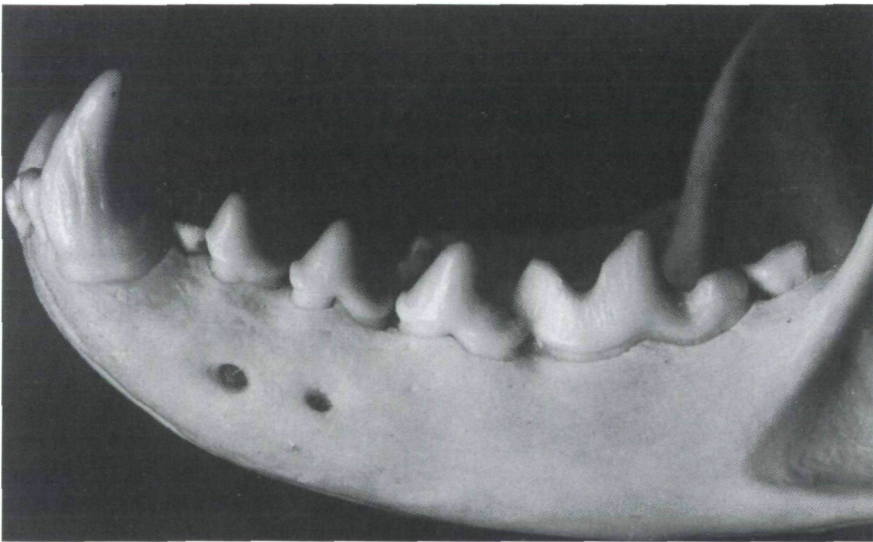


Figure 2: *Martes foina*, lower jaw left side Normal physiological bone margin, enamel at the border to the cementum typically bulged

### Diagnosis in the macerated skull

Diagnosis is done by optic investigation of the alveolar bone crest. A distance of 1.5mm between the border enamel-cementum and the bone is considered physiological in humans (HENKEL 1961). Loss of the bone occurs horizontally and in a progressed stage also vertically. In some cases it is combined with thickening of the crest. Pockets with abscesses *ante mortem* show a cribriform resorption of the bone structure. Vertical regression may lead to infrabony pockets limited with bone on either 1, 2 or 3 sides.

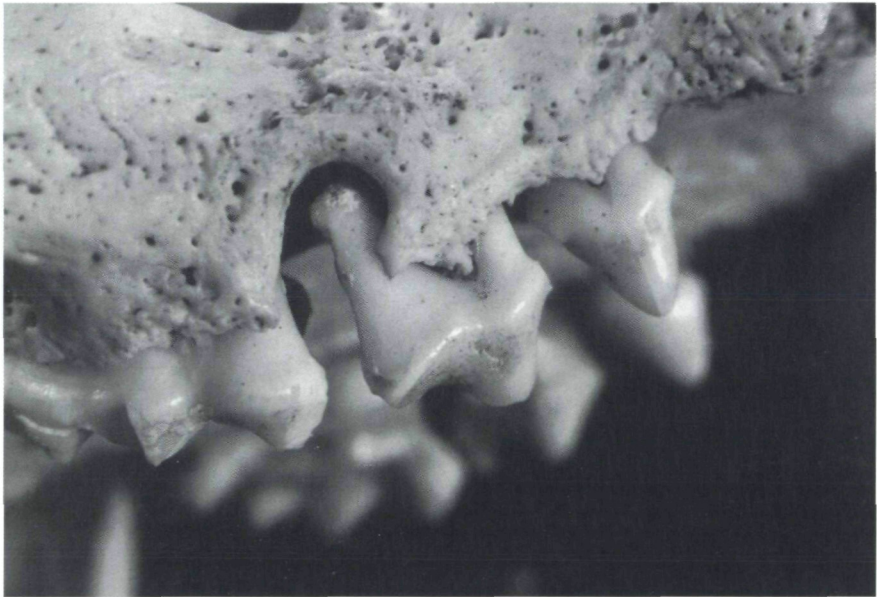


Figure 3: *Meles meles*: P<sup>3</sup>, P<sup>4</sup>, M<sup>1</sup> upper right jaw. Calculus on all teeth, horizontal loss at P<sup>4</sup> and M<sup>1</sup> between 1–1.5mm, progressed, vertical loss at the distal root of P<sup>4</sup>. The root is completely exposed beyond the *apex dentis* (1-sided infrabony pocket, distal bone, vestibular and oral sides missing). The horizontal loss might be a periodontal atrophy due to age. The symptoms at P<sup>4</sup> are rarely found in badgers; only one tooth in the whole dentition is affected. The cause of this lesion is unknown. The hard tissue of the tooth is without injury. The badger was presumably very old.

Relevant for differential diagnosis is a range of other phenomenons

1) Periodontal atrophy

Horizontal shrinking of the alveolar bone throughout the whole dentition without inflammation (involutive parodontopathy). Mostly in the senile stadium. Then it is regarded as a normal symptom of ageing.

2) Secondarily infected parodontium by mechanical lesion

In the ruminant, interdental impacted plant fibres are frequent. In such *loci* mechanical irritation may be followed by a facultative bacterial infection. Trigger organisms are bacteria of the soil and the plants. The parodontal lesion is confined to the injured region. Adjoining teeth without mechanical damage show an alveolar crest on physiological level. Impacted fibres can often be seen remaining stuck between the teeth of the macerated skull.

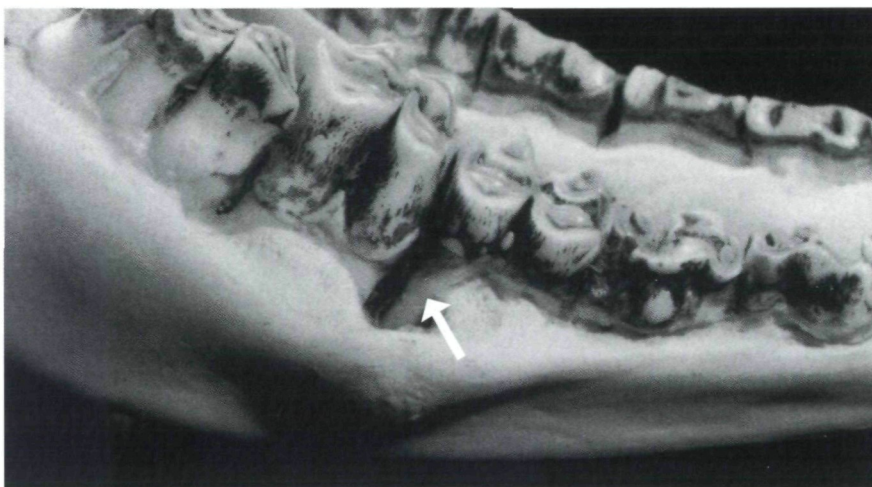


Figure 4: *Cervus elaphus*; two-sided ( → ) infrabony pocket caused by mechanical lesion, plant fibres still sticking between the teeth.

3) Apical osteolysis / parodontitis periapicalis

In the apical region of some teeth osteolysis imposes as single holes or a cribriform lesion. Radiographs and the cutting of bone reveal a cave surrounding the apex of the tooth. In general, there is no connection to the marginal bone crest,



unless the process is quite advanced or is from its beginning a combined periodontal and periapical lesion. The cause is mostly an injured and infected pulp (fracture or deep caries) and a penetration of microorganisms through the *apex dentis* into the bone. Only in rare cases it is the result of a traumatic contusion of the tooth (without fracture of dentin) causing necrosis of the pulp. If the infection proceeds over a long period of time, fistulation and multiple abscesses may lead to confounding the symptoms in the bone with actinomycosis.

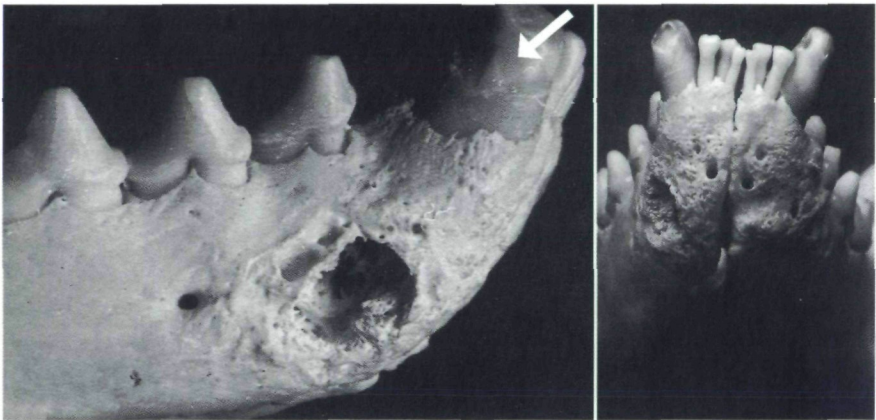


Figure 5: *Martes foina*, lower jaw, big apical osteolysis caused by a fracture of the lower right canine with affection of the pulp. See dark discolouring of the tooth ( → ). The fracture dates back some weeks or month.

#### 4) Recessions

Exposed root surfaces caused by non-inflammatory recession of marginal gingiva and alveolar bone. It is characterised by complete preservation of the interdental *papilla* and the interdental bone. The loss of bone is confined to the vestibular, rarely to the oral part of the alveolar cavity. Reason for the recession is mechanical irritation and/or heavy stress on the tooth. Recessions are found in regions having a physiologically rather thin proportion of alveolar bone. The exposed root surfaces are not covered by enamel and tend to get affected by caries more easily. They are often very sensitive to temperature changes.

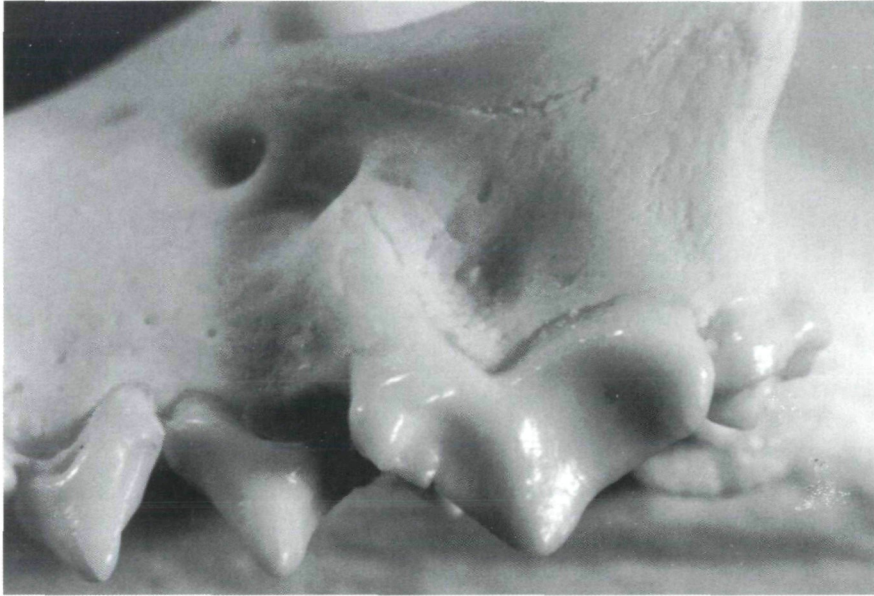


Figure 6: *Martes foina*, P<sup>4</sup>, upper jaw left. Recession of bone at the mesial root, distal root with physiological margin

#### 5) Fenestration

Small, precisely limited exposure of the root without connection to the *limbus alveolaris* (alveolar crest). These surfaces are *intra vitem* covered by gingiva. Fenestrations occur in places with very thin alveolar bone. Their origin is unknown and they have no negative consequences.

#### 6) Bulging of the *Limbus alveolaris*

In some individuals there is a bulging of the alveolar crest without changement of level. This bulging is functionally determined and is found in the elder individual with strong mechanical stress on the dentition.

Figure 8: *Meles meles*, lower jaw : the alveolar margin is clearly bulged ( → ), old individual

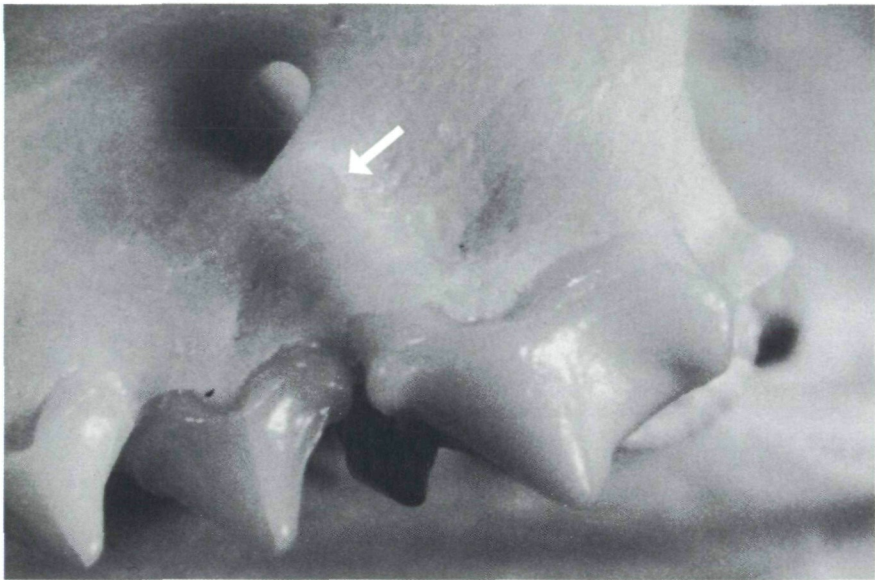
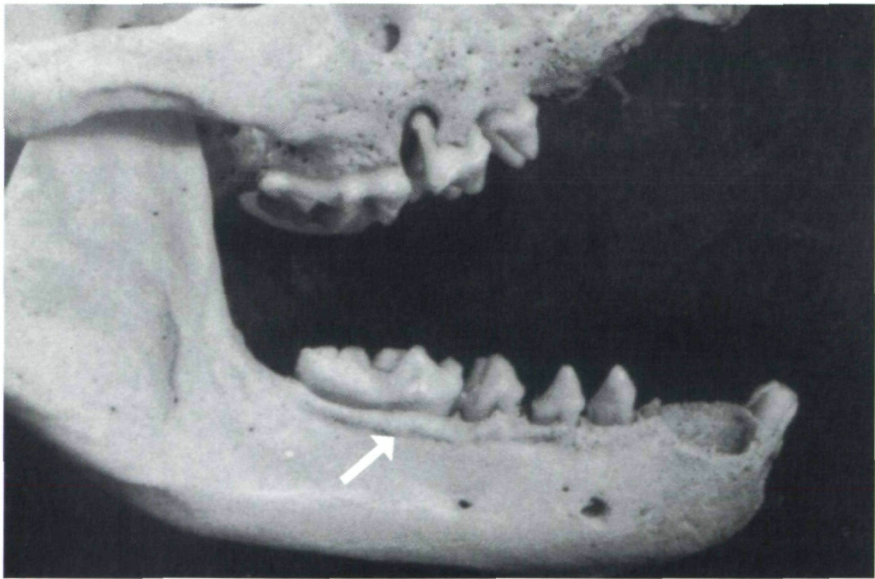


Figure 7: *Martes foina*, P<sup>4</sup> upper jaw left, small round fenestration of the mesial root ( → )



### 7) Actinomycosis („Strahlenpilz“)

Actinomycosis arises from *Actinomyces israelii* in man and *A. bovis* in cattle which are in both, part of the normal saprophyte oral flora. Trauma is considered one of the conditions, that lead the organisms to become invasive (DAVIS et.al 1990). The infection is characterised by chronic, destructive abscesses of the tissue. In ruminantes mostly the lower jaw is affected. The bone in the macerated skull displays a hard, sponge-like perforated and enlarged surface.

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