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A STUDY OF DENTAL DISEASE
IN THE HORSE

-oOo-

A THESIS submitted for
THE DEGREE OF DOCTOR OF PHILOSOPHY
in
THE FACULTY OF VETERINARY MEDICINE
of
THE UNIVERSITY OF GLASGOW
by
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SUMMARY

The literature on the subject of equine dental disease has been reviewed under three headings, namely, diseases of tooth substance, diseases of the periodontium and clinical aspects of dental disease. A description of the gross and microscopic anatomy of the teeth has been given, in particular the structure and function of the cheek teeth. It is emphasised that under normal conditions the six teeth of each arcade function as a single unit. With its reserve crown capacity and crown cementum the horse has an efficient masticatory apparatus well suited to its grazing habitat.

A survey of the incidence of equine dental disease was made, using 446 skulls collected from abattoirs and knacker houses. The most common pathological conditions were periodontal disease and caries of cementum. The most severe forms of periodontal disease were associated with increasing age and the development of irregularities of wear of the dental arcades. Caries of cementum was found only in the maxillary cheek teeth and a study of cementogenesis in Thoroughbred fetuses suggested that primary hypoplasia was a major aetiological factor in the development of this condition, while lysis of cementum by the acid products of fermented foodstuffs was of secondary importance.

Periapical infections were the most important clinical dental disease, treated in an equine referral clinic. It was found that pulpitis may arise from caries of cementum in the maxillary teeth. However, in both upper and lower jaws it was concluded that problems of maleruption and the subsequent formation of periapical cysts or granulomata were equally important in the pathogenesis of periapical infections.

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GENERAL INTRODUCTION

Horses may be aged, with considerable accuracy, from a detailed examination of the incisor teeth (Goubaux and Barrier, 1892; Huidekoper, 1903; Galvayne, op. cit.) In the past, sharp trading sometimes involved altering the apparent age of a horse. In one such practice, called "bishoping", the incisor tables of an aged horse were burned to give it a false infundibulum and thereby "reduce" its age. It was important, therefore, that ageing was carried out by truly experienced and expert veterinarians. In this modern era of consumer protection an equivalent expertise is needed to offer sound opinions over the sale and purchase of horses and ponies. Indeed it could be argued that the saying "never look a gift horse in the mouth" is no longer good advice.

In a survey of the incidence of equine diseases the British Equine Veterinary Association (1965) reported that nearly ten per cent of the requests for veterinary attention were for dental reasons. These were mainly requests for routine rasping of the teeth and dental prophylaxis. Other workers (Little, 1913; Harvey, 1920; Joest, 1926) have described significant primary dental diseases of the horse but the literature is, for the most part, concerned with aspects of anatomy, dentistry and surgical techniques. Few investigations

of the aetiology and pathogenesis of dental disease in the horse have been recorded.

It was the aim of this study to review the literature with respect to equine dental disease and to present data from a dental disease survey.

From this survey the aetiology and histopathology of some specific diseases have been studied and the results correlated with, and confirmed by, cases of clinical dental disease examined and treated over a 12 year period.

REVIEW OF THE LITERATURE

INTRODUCTION

Becker (1962), in the revised edition of Joest's Handbook of Special Pathology of Animals, has written extensively on the subject of dental anatomy, physiology and pathology in domestic animals. Much of this material is based on the studies of other German workers and is particularly concerned with the horse (Joest, 1915, 1922, 1926; Herzog, 1937; Kupfer, 1937; Leue, 1941; Becker, 1945, 1954; Bodingbauer, 1948; Eisenmenger, 1959; Gerés, 1962). Merillat's Animal Dentistry and Diseases of the Mouth (1921) gives a brief description of some aspects of dental pathology and details techniques of dental surgery. In other standard surgical texts (Dollar, 1912; Wooldridge, 1934; Silbersiepe and Berge, 1954; Frank 1959; Skewes, 1962; Berge and Westhues, 1965), emphasis is again placed upon aspects of gross anatomy and descriptions of those operations that form the backbone of the work of the equine dentist - namely the rasping and extraction of teeth and the correction of abnormalities of wear. Baker (1972) revised the work of Skewes and discussed the aetiology and pathogenesis of both periodontal disease and dental decay in the horse.

It will be convenient to review the literature under the subheadings Diseases of the Tooth Substance, Diseases of the Periodontium and Clinical Aspects of Dental Disease.

A. DISEASES OF THE TOOTH SUBSTANCE

Dental caries is a disease of the calcified tissues of the teeth, characterised by a demineralisation of the inorganic portion and a destruction of the organic substance of the tooth. It is the most prevalent chronic disease affecting the modern human race (Shafer, Hine and Levy, 1963).

In ancient times dental caries was associated with the idea of worms in or around the teeth. Jacques Houllier (1498-1563) first cast doubt on the existence of worms but Gottfried Schultz declared that the gastric juice from a pig would expel the worms, as large as an earthworm, from a decayed tooth. On the other hand Gallen believed that dental caries was produced by an abnormal condition of the blood which affected the internal structure of the tooth (Guerini, 1909, A History of Dentistry).

Leber and Rottenstein (1867) were among the first workers to suggest that micro-organisms were associated with the production of dental caries in man. Miller, working in Koch's laboratory in 1889, found that organisms from carious dentine produced lactic acid when cultured with starch or with sugar, and from these observations he formulated the chemico-parasitic theory of dental caries. Miller's hypothesis may be stated briefly as follows: "Dental decay is a chemico-parasitic process consisting of two stages, the decalcification of enamel, which results in its total destruction, and the decalcification of dentine as a preliminary stage, followed by dissolution of the softened residue. The acid which affects this primary decalcification is derived from the fermentation of starches and sugar lodged in the retaining centres of the teeth". This theory has been accepted by the majority of

investigators in a form that is essentially unchanged since its original formulation. The bulk of scientific evidence does implicate carbohydrate, oral micro-organisms and acids.

In 1928 Bunting stated that there was a proven correlation between dental caries and the acid producer Lactobacillus acidophilus. Keyes (1960) demonstrated that, under laboratory conditions, dental caries in hamsters and rats could be considered to be an infectious and transmissible disease. Pure cultures of streptococci isolated from hamster caries would induce the typical picture of active dental caries in other hamsters. Confirmatory studies have been reported by Fitzgerald, Jordan and Stanley (1960) who maintained gnotobiotic rats fed on a coarse particle, high sugar diet, which in normal animals would produce dental caries, without caries development until they were inoculated with a single strain of oral streptococcus isolated from a control rat on the same diet.

Cariogenic acid is produced beneath the mucobacterial layer which forms on the surface of the tooth and is known as dental plaque. By the use of antimony micro-electrodes to measure the pH of dental plaque in situ, Stephan (1940) showed that the pH of plaques varied from an average of about 7.1 in caries free persons to 5.5 in persons with extreme caries activity. He also showed that after a rinse of 10% glucose or sucrose solution the pH in dental plaque fell to between 4.5 and 5 within two to five minutes, then gradually returned to the initial level within one to two hours.

More recently work has been carried out on the proteolysis of the organic constituents of the tooth substance which is thought

in some circumstances to initiate dental caries. Both Baumgartner (1911) and Fleischmann (1914, 1921) demonstrated that micro-organisms could invade the enamel lamellae and believed that acids produced by these bacteria were capable of destroying the inorganic portion of the enamel. Gottlieb (1944) and Gottlieb, Diamond and Applebaum (1946) postulated that caries was essentially a proteolytic process, in which the micro-organisms invaded and destroyed the organic pathways, but they did admit that acid formation accompanied the proteolysis. Pincus (1948) had a somewhat different approach to the caries problem. He proposed that Nasmyth's membrane and other enamel proteins are mucoproteins.. yielding sulphuric acid upon hydrolysis and he subsequently isolated gram negative bacilli capable of producing the enzyme sulphatase from the oral cavity. In an attempt to reconcile the two chief theories concerning the aetiology of dental caries Manley and Hardwick (1951) pointed out that whilst the acidogenic and proteolytic mechanisms may be separate and distinct they may also be synergistic. Many bacteria produce acid from an appropriate carbohydrate and may even degrade protein in the absence of carbohydrate. On this basis it has been proposed that there may be two types of caries lesion. In one type micro-organisms invade the enamel lamellae, attack the enamel and involve the dentine before there is clinical evidence of caries. In the other, bacteria in the dental plaque overlying the enamel form acids, which decalcify the enamel prior to invasion by micro-organisms.

Schatz, Karson, Martin and Schatz (1957) proposed the proteolysis-chelation theory of dental caries production. This theory stated that the bacterial attack on enamel, initiated by

keratinolytic micro-organisms, caused a breakdown of the protein and other organic components of the enamel, chiefly keratin. In this way substances are produced which may form soluble chelates with the mineralised component of the tooth and thereby decalcify the enamel at a neutral or even alkaline pH. On the other hand enamel contains other organic components besides keratin such as mucopolysaccharides, lipid and citrate which may be susceptible to bacterial attack and act as chelators. Such a theory resolved the argument as to whether the initial attack of dental caries was on the organic or inorganic portion of the enamel by stating that both may be attacked simultaneously.

Jenkins (1961) produced an excellent review of the proteolysis-chelation theory of dental caries with a careful review of supporting and contradicting evidence.

The use of a coarse particle, high sugar content diet will produce dental caries in all laboratory animals and consequently rabbits, rats, hamsters and mice have been widely used in the investigation of the aetiology and pathogenesis of dental caries. Colyer (1931) published his observations on the examination of specimens of teeth of animals from the wild state. Under such conditions the incidence of caries was very low e.g. 1.40% in Primates, 0.20% in Carnivora, 0.16% in Rodentia, 0.30% in Ungulata, 0.13% in Marsupialia.

The incidence of dental caries in the domestic dog has been reported to be 5% by Bodingbauer (1955) and 6% by Bell (1965). Lewis (1965) showed that, under experimental conditions of xerostomia, high carbohydrate diet and dental trauma, the inoculation of lactobacillus cultures did not produce caries in

dogs' teeth. Gardner, Darke and Keary (1962) based their explanation of the low incidence of naturally occurring dental caries in the dog on three main factors. Firstly, with the exception of the molar teeth, the anatomical form of the tooth in the domestic dog is such that no fissures are present to trap food, bacteria and debris. Secondly the salivary pH in dogs is within the range 7.1 to 7.9 and thirdly the urea content of canine saliva is much higher than in man. The latter two factors, that is the higher pH and greater urea content, give canine saliva a greater buffering power than human saliva and consequently the effect of mouth acids is reduced.

In those dogs in which the disease has been reported the tooth most frequently affected is the first maxillary molar. It should be noted that this tooth is triangular in shape with a central valley in the crown, which encourages the retention of food material. To a lesser degree the second maxillary molar tooth and the second and third mandibular molar teeth are also involved.

In Merillat's text on animal dentistry (1921) a description was given of the condition termed dental necrosis of herbivores. Merillat used the synonyms alveolar periostitis, peridentitis, pericementitis and pulpitis and noted that the term caries was erroneous when applied to solipeds. He suggested that the entrance of food into an imperfectly closed infundibulum was the cause of more than 95% of the decayed molars of horses. He described a sequence of events, beginning with pulpitis, induced by the entry of food into the infundibulum and ending with self-extraction of the affected tooth. Colyer (1931) and Silbesiepe and Berge (1954)

refer to this condition as caries, as it was a progressive destruction of the tooth tissue brought about by micro-organisms. In the opinion of Colyer the disease was initiated by the accumulation and fermentation of carbohydrate food material in the infundibulum. This author found that 13% of 184 skulls were affected and that it was more common in the maxillary arcade, an occlusal lesion of the first molar (i.e. fourth cheek tooth) being seen most frequently. Honma, Yamakawa, Yamauchi and Hosoya (1962) found an incidence of 100% in horses over 12 years of age. Again the first maxillary molar tooth was most frequently affected, but lesions were also found in the mandibular cheek teeth.

Hofmeyr (1960) suggested that the reason for the common occurrence of caries in the cement lacuna of the first molar tooth was that this tooth erupted long before the other teeth in the upper buccal arcade and was therefore subjected to fermentation of carbohydrate food material over a longer period of time. According to Hofmeyr dental caries was almost never seen in the incisors, but Lee (1968) described the presence of labial caries in the central upper incisors in a 7 year old Apaloosa mare. It was suggested that the lesion developed as a result of high levels of molasses in the diet.

Jubb and Kennedy (1970) stated that dental caries was common in the horse and it may begin on the occlusal surface where there is cement hypoplasia. However, they further commented that it was more likely to develop on the approximated surfaces of adjacent teeth and was always associated with periodontitis. According to Skewes (1962) there was considerable doubt that dental caries existed in the horse and, in his opinion, if it did occur it was

secondary to alveolar periostitis. He preferred to use the term decayed teeth to describe destructive diseases of the tooth.

Joest and Becker (1962) described four forms of dental caries in the horse. Firstly caries of cement from the occlusal surface, secondly caries of the peripheral cement, thirdly caries of root cement originating from purulent periodontitis and fourthly caries from an open pulp cavity.

Baker (1970) noted that Goodall (1895) felt that erosion of the cement lake was quite normal in horses, while caries did not occur except in extreme old age. Consequently Baker suggested that until further studies had been made of the histology and pathogenesis of the lesion known as dental caries in the horse that the term infundibular necrosis might be more appropriate.

B. DISEASES OF THE PERIODONTIUM

Diseases of the periodontal structures have been known since antiquity. Skulls of some ancient cave dwellers show evidence of chronic periodontal disease. An acute form, now known in man as acute necrotizing gingivitis or Vincent's infection was reported at least as early as 400 B.C. in soldiers of the Greek army of Xenophon (Shafer, Hine and Levy, 1962).

Classification of various periodontal diseases is difficult as in nearly every case the condition begins as a minor localised disturbance which, unless adequately treated, gradually progresses until the alveolar bone is resorbed and the tooth is exfoliated. This means that a variety of aetiological stimuli may produce identical pathological results. In other words the reaction that occurs in the tissues supporting the teeth is usually a non-specific

one and histological studies of the periodontium seldom indicate the type of irritant causing the disease.

Colyer (1931) quoted the description of John Hunter on periodontal pathology and recorded that the condition had been variously designated Rigg's disease, pyorrhoea alveolaris, alveolar osteitis and periodontal disease. Colyer felt that none of these terms was really descriptive of the pathological process and he proposed the use of the term "paradontal disease". This term was introduced into the veterinary literature by Wright (1939) and its use was continued by Shuttleworth (1948) and Hofmeyr (1960). Baker (1970) reviewed the more recent literature and referring to the classification of periodontal diseases as used in man concluded that the generic term periodontal disease was most suitable.

The report of the committee on classification and nomenclature of the American Academy of Periodontology in 1957 suggested that the pathological involvement of periodontium may be grouped in the following four categories:-

1. Inflammation (gingivitis, periodontitis).
2. Dystrophy (gingivosis, periodontosis).
3. Neoplasia.
4. Anomalies.

All four categories are known to occur in domestic animals including the horse, but the literature is mostly occupied with the first category i.e. inflammatory changes. However there has been considerable confusion of terminology in the veterinary literature and opinions have varied as to the aetiology and pathogenesis of the condition. Periodontal inflammation has been recognised for years as being extremely important in the horse.

Little (1913) reported that 'quidding' was a pathognomonic sign of periodontal disease or alveolar periostitis. He observed that the lesion started primarily in the spaces between the cheek teeth, usually of the lower jaw, and that the molars were more often affected than the premolars. Colyer (1931) examined 500 equine skulls and in approximately one third of these he found evidence of periodontal disease. He concluded that the lesion was initiated by gingival trauma caused by coarse chaff feeding. Pillers (1933) was of the opinion that it formed one of four corner stones on which other diseases of the cart-horse were built. Voss (1937) felt that infection of the periodontal structures resulted from interference with salivation. He was of the opinion that saliva washed and disinfected the teeth and that irregular feeding resulted in a reduced salivary flow and consequent devitalisation of the gingival epithelium.

Harvey (1920) thought that the deeper tissues of the alveolar margin may be bruised by abnormal pressure during mastication. He felt that the shedding of the first and second temporary cheek teeth was a critical period in the pathogenesis of the disease. He observed that the lesion often became quiescent after this time only to develop in later years. Baker (1970) reported a high incidence of inflammatory gum disease during the period of eruption of the teeth (40% in horses 3-5 years of age) followed by a reduction during subsequent years (14% in horses 5-10 years). After 15 years of age Baker found up to 60% of horses suffered from some degree of periodontal disease.

Baker referred to work in other species which concluded that dental work was necessary for the maintenance of the health of the

gingival mucosa. It has been shown conclusively in ferrets (King 1947) and dogs (Brown and Park, 1968) that the frictional force associated with chewing hard substances was sufficient to keep the teeth free of dental scale and the gums in a healthy state. Baker therefore suggested that the peak incidences of periodontal disease in the horse were associated with other factors which interfered with normal mastication. In young horses it was the shedding of deciduous caps, while in older horses more severe lesions and a greater incidence were associated with irregularities of crown wear and other diseases of tooth substance. Leue (1941) used a pen recording device attached to the muzzle of horses to investigate the pattern of jaw movements during mastication (molographs) in normal horses and in horses with dental disease. It was found that the amplitude of the side to side movement of the mandible within the maxilla was reduced with soft food-stuffs and in cases of dental disease.

Mellanby (1952) found frank periodontal lesions in dogs on diets deficient in Vitamin A, and Shuttleworth (1948) felt that under some conditions of winter feeding avitaminosis A may be instrumental in initiating degeneration of the oral mucosa in horses.

Tumours originating from dental tissue elements are rare in animals. Ameloblastoma, a slow-growing epithelial tumour resembling the developing enamel organ, is seen most frequently in cattle (Joest, 1926). Complex dental tumours composed of more than one tissue type have been described in the horse. Peter, Myers and Ramsey (1968), Lingard and Crawford (1970) and Roberts, Groenendyk and Kelly (1978) have all described ameloblastic odontomata or calcified mixed odontogenic tumour consisting of odontogenic

epithelium and connective tissue in foals and yearlings. The characteristic clinical signs are mandibular or maxillary swelling with radiographic evidence of abnormal tooth development and a calcified mass distorting the bones of the head. The diagnosis can be confirmed by histological examination and affords the differential diagnosis from maxillary follicular cysts as described by Espersen (1962) and by Rubarth and Krook (1968).

C. CLINICAL ASPECTS OF DENTAL DISEASE

Much has been written of the importance of dental disorders in causing unthriftiness and wasting in horses and comments have been made upon the techniques of equine dentistry in correcting these disorders. (Little, 1913; Merillat, 1921; Joest, 1926; Pillers, 1933 and Shuttleworth, 1948). It has also been recognised that in many training yards routine dental prophylaxis is carried out by non-veterinarians and consequently the modern equine practitioner may not be as experienced in the art and science of equine dentistry as was his predecessor (Cosgrove, 1965). Baker (1970) recommended the routine rasping of the dental arcades as the only effective method available to maintain normal occlusal contact and thereby preclude irregularities of wear and their associated periodontal disease.

When discussing dental surgery in the horse Cook (1965) classified three types of periapical abscess.

1. Mandibular root sepsis with mandibular sinus formation.
2. Sepsis around the roots of the first two maxillary cheek teeth accompanied by maxillary osteitis and sinus formation.
3. Infection of either of maxillary cheek teeth 3, 4, 5 or 6 with concomitant maxillary sinus empyema.

Both Eisenmenger (1959) and Hofmeyr (1960) had stated that sepsis around the first two maxillary teeth produced chronic ossifying alveolar periostitis. Baker (1971) reviewed a series of cases of dental root infection and described periodontal alveolar and paranasal sinus changes associated with both acute and chronic pulpitis in the horse. He suggested that subclassifications as proposed by Eisenmenger, Hofmeyr and Cook may be useful under some circumstances, but that the pathogenesis of periapical abscess formation was a non-specific reaction and the end result e.g. mandibular sinus or maxillary sinus empyema was related to the anatomical position of the tooth involved. The importance of radiology in the clinical diagnosis of equine dental diseases has been illustrated by Baker (1971) and Scott, Gallagher, Boles, Beasley and Reed (1977). In particular it was shown that tooth fragments could be left within the alveolus after dental extractions. These fragments, under some circumstances, act as sequestra and promote further sinus formations. Three such cases were described by Scott et al. (1977) and Baker (1971) reported that 20 per cent of horses undergoing tooth extractions required further surgery to facilitate alveolar curettage.

The clinical signs of dental disease in the horse were listed by Scott et al. (1977) as including -

1. Difficulty in mastication.
2. Quidding i.e. dropping partially chewed food material from the mouth.
3. Unthriftiness accompanied by abnormal mastication.
4. Persistent sinus formation.
5. Maxillary sinus empyema with malodorous nasal discharge.

6. Maxillary or mandibular enlargement.

7. Problems in biting and riding the horse.

Burns (1969) described the characteristic lesions of dental fluorosis as stain, abnormal wear and various forms of dental hypoplasia. In most cases such changes were irreversible.

CHAPTER I

DENTAL ANATOMY OF THE HORSE

INTRODUCTION

Four tooth types can usually be distinguished in adult mammals. These are, in cranio-caudal order the incisors, a single canine followed by a series of premolars and finally a molar series. In early fossil mammals the number of teeth in these various categories has varied widely with, quite often, a greater total number than is seen in any living forms. The primitive placentals, however, settled to a count in which there were three incisors, one canine, four premolars and three molars in each quadrant. Few mammals retain precisely that number today (Romer 1962).

Incisor teeth, useful in nearly every mode of life are retained in most mammalian groups. Various herbivores have evolved specialised cropping methods in which the incisors are modified or lost - an incisor mucosal pad replaces the upper incisors in ruminants. The elephant's tusks are greatly elongated upper incisors and rodents have developed a pair of incisor chisels that grow continually throughout their life.

Canine teeth are prominent biting and piercing weapons and as such are unique features of carnivores.

The cheek teeth of carnivores are reduced in number but in herbivores they have developed into an efficient grinding system separated from the cropping incisors by a gap - the diastema.

The study of the history of the cheek teeth is the best known feature of horse evolution and has been well documented (Clarke 1880; Simpson 1951). The teeth of Equus caballus have evolved into the structure and form they now have as a result of changes in the environment of its early Eocene ancestor and in particular in the change from a browsing animal to a grazing animal.

The Eocene ancestor of the family Equidae (Hyracotherium or Eohippus) was a small (26") three toed animal with low crowned (brachydont) cheek teeth made up of four premolars and three molars. All the teeth had simple crown patterns and chewing depended mainly on the three molar teeth. Subsequent changes in the environment during the later Miocene period brought about a rapid evolution of the teeth as a result of the survival of favourable mutations. These changes were a modification of crown pattern, an increase in height of the crown and the development of cementum. What had been two open valleys on the teeth now became deep, closed pits and numerous wrinkles and spurs appeared on the sides of the main crests. These changes were similar in both upper and lower jaws, but the changes were less extreme in the lower teeth.

The process whereby the premolar teeth became anatomically similar to the molar teeth is described as molarization. The formation of high-crowned cheek teeth of Merychippus in the late Miocene period was accompanied by the development of cementum. This appeared on the outside of the enamel crown and developed to fill all its valleys and in so doing protected the brittle enamel from

cracking. In this way a permanently erupting cheek tooth was formed with a crown height at least twice its width. Such a tooth erupts at a rate equal to that of the rate of wear of the crown by attrition.

DENTAL FORMULAE OF EQUUS CABALLUS

$$\begin{array}{l} \text{Deciduous} \quad I \frac{3}{3} \quad C \frac{0}{0} \quad P \frac{3}{3} \quad \times \quad 2 \quad = \quad 24 \\ \text{Permanent} \quad I \frac{3}{3} \quad C \frac{1}{1} \quad P \frac{3 \text{ or } 4}{3} \quad M \frac{3}{3} \quad \times \quad 2 \quad = \quad 40 \text{ or } 42 \end{array}$$

Eruption Times (After Sisson 1910)

A Deciduous	1st Incisor	Birth or first week
	2nd Incisor	4-6 weeks
	3rd Incisor	6-9 months
	1st Premolar	Birth or first two weeks
	2nd Premolar	
	3rd Premolar	
B Permanent	1st Incisor	2½ years
	2nd Incisor	3½ years
	3rd Incisor	4½ years
	Canine	4-5 years
	1st Premolar (Wolf tooth)	5-6 months
	2nd Premolar	2½ years
	3rd Premolar	3 years
	4th Premolar	4 years
	1st Molar	10-12 months
	2nd Molar	2 years
3rd Molar	3½-4 years	

A number of standard variations in the dental formula are recognised. The canine teeth are absent or rudimentary in the mare, thus reducing the total number of teeth found in mares by four. It is usual to include the vestigial first premolar in the upper jaw in the dental formula but it can also be found in the lower jaw in some cases thus increasing the total number of teeth by two.

MATERIALS AND METHODS

The gross morphology and anatomical relations of the teeth of the horse were studied from both fresh and preserved specimens. Reserve crown, roots and alveolar attachment were recorded from sculpted and radiographed specimens.

Histological Methods

Individual teeth were isolated and transverse and vertical sections made using a diamond-coated disc cutter (Cut and Grind).^{*} These sections were prepared by hand under a water spray from fresh material.

Two mm. sections were stored in 10% buffered formalin. Decalcification was carried out in Cal-Ex and ethyl alcohol and xylene used for dehydration. The tissues were embedded in wax and 12 μ sections were prepared and stained with haematoxylin and eosin.

A limited number of ground transverse sections were prepared using carborundum grinding pastes and a microtome knife sharpener.

^{*} Agate and General Stonecutters Ltd., London.

RESULTS AND DISCUSSION

(1) Gross Morphology

Incisor Teeth There are six teeth in each jaw and they are placed close together so that their labial edges almost form a semi-circle. The occlusal surface has a deep enamel invagination, the infundibulum, which is only partly filled with cementum. As the teeth are worn a characteristic pattern forms in which the infundibulum is surrounded by rings of enamel, dentine and external enamel in a concentric pattern. Each incisor tooth tapers regularly from a broad crown to a narrow root so that with wear, as the mid-portion of the incisor is exposed, the two diameters of cross-section are about equal. Observation of state of eruption, table pattern and shape and the angle of incidence are used as guides to the ageing of horses. With age the infundibulum becomes smaller, it approaches the lingual border and finally disappears. It persists for a longer period in the upper incisors than in the lower ones because of its greater depth. Its rate of disappearance should not be relied upon too closely when ageing horses. As the infundibulum disappears a mark appears on the labial aspect of the table of the incisor tooth, this mark represents the arc of dentine formed to protect the pulp cavity from exposure by attrition of the crown. The average length of incisor teeth in a 6 year old horse is 7 cm.*

Deciduous incisors are much smaller than permanent incisors and have a distinct neck at the junction of the root and crown.

* All sizes stated are taken from the measurements of the teeth of adult Thoroughbred horses.

When viewed from the front the crown is shell shaped rather than rectangular. The infundibulum is shallow.

Canine Teeth The stallion's canine teeth are simple (i.e. without complex crown and cement) and are curved. The crown is compressed and is smooth on the labial aspect but carries two ridges on its lingual aspect. The upper canine is situated at the junction of the premaxilla and maxilla whilst the lower canine is nearer the corner incisor. No occlusal contact is made between the upper and lower canines. These teeth do not exhibit continual eruption.

Cheek Teeth It is common in veterinary works to call all the cheek teeth of the horse molar teeth since the premolars (with the exception of the wolf tooth) do not differ materially from the true molars (i.e. posterior cheek teeth - Brit. Med. Dict.) in size or form. In this account the term cheek teeth will be used to include both premolars and molars and they will be numbered 1-6 (i.e. cheek tooth 1 \equiv PM2 and cheek tooth 4 \equiv M1).

The constant number of cheek teeth is 24 making four dental arcades of 6 teeth in each. In addition the persistent wolf tooth is commonly found in the upper jaw. At its maximum development the crown of this tooth is small and irregularly conical presenting some of the indication of enamel folding that is so characteristic of the other cheek teeth of the horse. When less developed the crown is generally a small and simple cone. It is this latter pattern which is seen with the greatest frequency.

Maxillary Cheek Teeth With the exception of the first, each tooth is in the shape of a slightly bent four-sided prism. The first tooth is, however, three-sided because its rostral border

does not contact another tooth. The first tooth also differs in the character of its buccal (or labial) surface: in the second to the sixth tooth this surface carries a longitudinal rounded ridge (cingulum) that separates two grooves, however in the first tooth there are two such ridges, the anterior being somewhat less prominent. The lingual surface of the crown of each tooth is marked by a longitudinal ridge that corresponds in position to the groove and ridge of the chewing surface. The occlusal surface is not positioned at right-angles to the longitudinal or vertical plane of the tooth but is set somewhat obliquely with the labial side of the maxillary cheek tooth taller than the lingual side.

The unworn maxillary tooth presents a surface on which there are two undulating and narrow ridges (styles) one lateral and the other medial. On the anterior and lingual side of the medial style there is an extra hillock. The central portion of the surface is indented by two depressions comparable to, but much deeper than, the infundibula of the incisor teeth. When the teeth have been subjected to wear the enamel clothing the ridges is worn through and the underlying dentine appears on the surface. The result is that after a time the chewing surface displays a complicated pattern that may be likened to the outline of an ornamental or ornate capital letter B, the upright stroke of the B being on the lingual aspect. The brittle enamel is supported by dentine internally, cementum within the enamel lakes and a surround of peripheral cementum that fills in the spaces between the teeth so that all 6 teeth function as a single unit - the dental arcade (Figure 1). Each tooth is crossed by

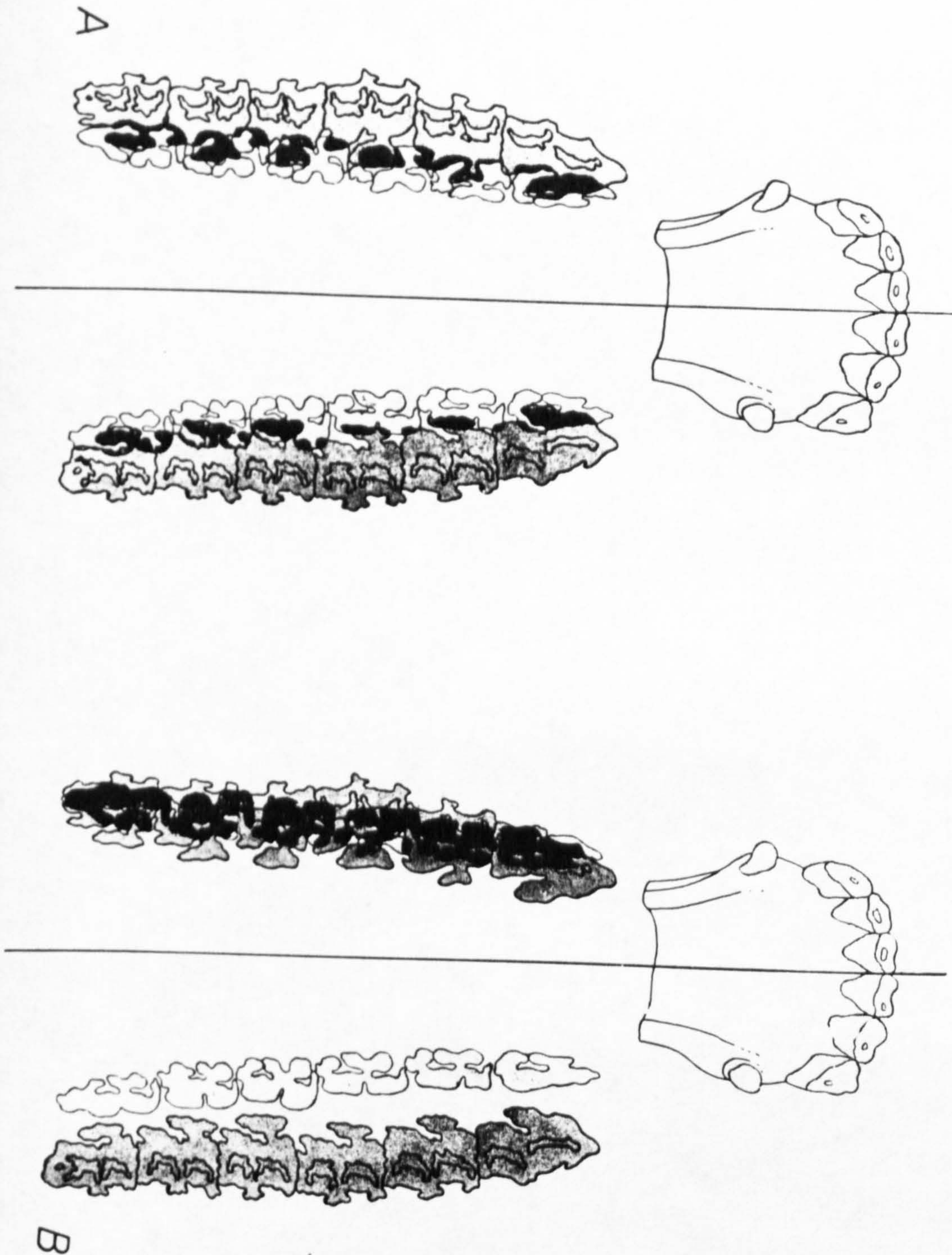


FIGURE 1: Relative positions of upper and lower dental arcades.
 A. In central occlusal contact B. Left side contact.
 (Modified and redrawn after Kupfer 1937).

transverse ridges so that the whole of the maxillary arcade seems to consist of a serrated edge. The serrations are formed in such a way that a valley is present at the interproximal* area of contact.

The true roots of the cheek teeth are short compared with the total length of the teeth and are generally three in number. There are two small lateral roots and one large medial root. If the term "crown" is held to include all that part of the tooth on which enamel is present then the teeth of the horse must be described as having a considerable amount of their crown buried beneath the gum. It is customary to refer to this portion of the crown as the reserve crown and to confine the term root to that area of the tooth that is comparatively short and enamel free. As the tooth wears away the reserve crown is gradually exposed and the roots lengthen.

The exposed crowns are in close contact forming a slightly curved continuous row with the convexity on the buccal aspect. The embedded crowns vary in length and lie at a slight angle to each other so that the emerging crowns fit close together to form the compact arcade mass. In the adult horse the first is directed upward and slightly forward and is 6.8 cm. long, the second is vertical and 8.3 cm. long, the third and subsequent teeth incline backwards to an increasing degree and are 9.0, 7.8, 8.7 and 7.6 cm. long respectively.** The embedded portions of cheek teeth 3-6 occupy part of the paranasal maxillary sinuses. In younger horses the position of the teeth relative to the sinuses changes because the

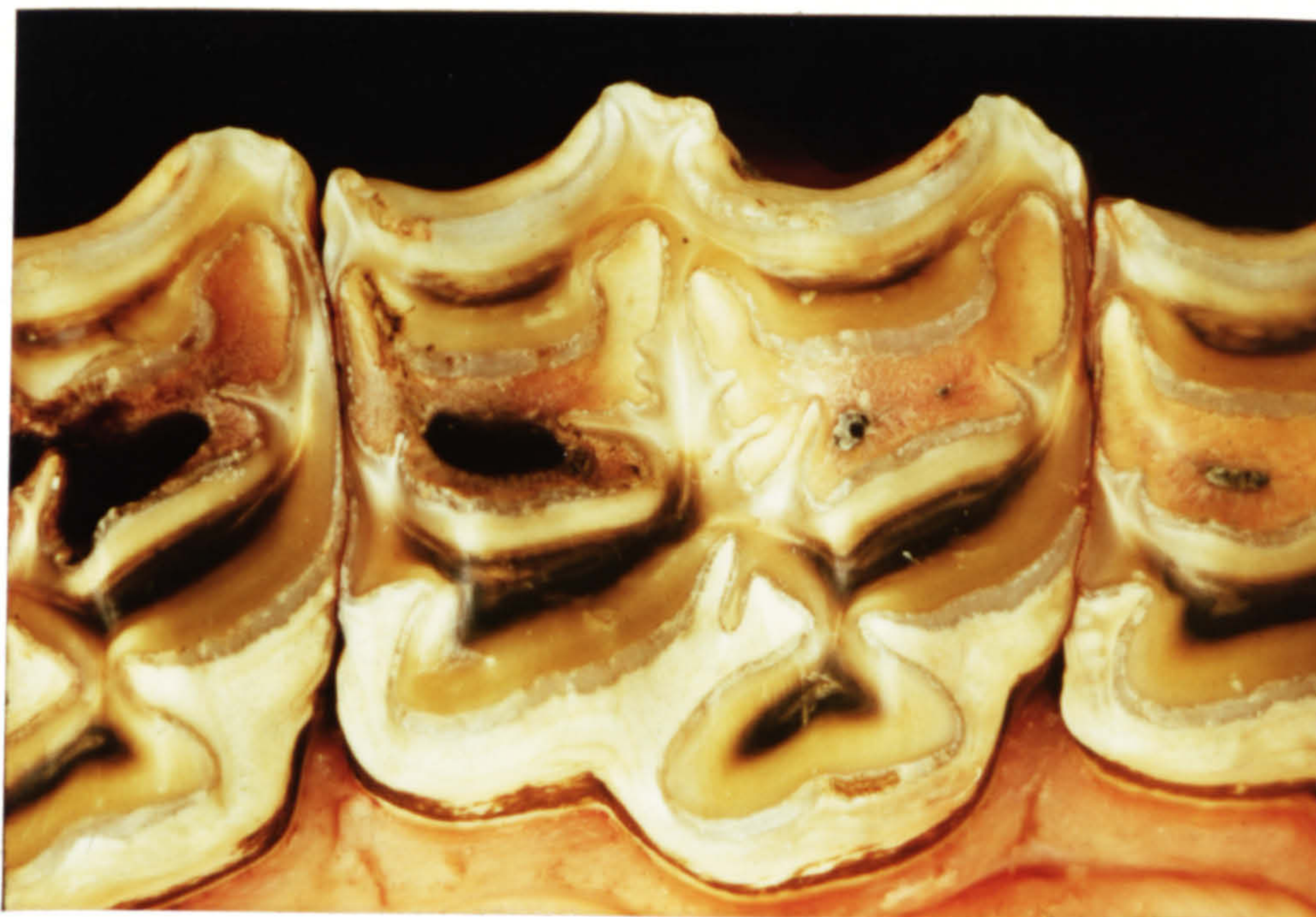
* interproximal \equiv interdental.

** Cheek tooth measurements quoted are the average of the apex of the root to the crown measurements taken from the rostral and caudal roots.

extent of the reserve crown and the caudo-rostral angle of eruption of the teeth. This effectively reduces the size of the rostral maxillary sinus.

Goodall (1895) illustrated these changes by describing the position of a perpendicular line from the front of the orbit. He found that in a 180 day foetus it bissects the molar arcade between the last two deciduous cheek teeth, at birth it is posterior to the 3rd cheek tooth, at 2 years it is posterior to the 5th cheek tooth and subsequently the teeth move forward in the mouth so that at 19 years this line crosses the arcade behind the last cheek tooth.

Mandibular Cheek Teeth These teeth are, on the whole, about as long as the maxillary teeth but their transverse measurement is much less and consequently they have an oblong instead of an approximately squared chewing surface. On the labial aspect of the first five teeth there is a longitudinal groove. The sixth tooth has two longitudinal grooves on the labial aspect. The lingual aspect is irregularly grooved longitudinally. The exposed part of the crown is taller on the inner or lingual side with the result that as in the maxillary teeth the chewing surface is set obliquely to the longitudinal plane of the tooth. The pattern assumed by the worn occlusal surface on the mandibular teeth is more simple than that of the maxillary teeth (Figure 2). While there are two infundibula these are not closed on the lingual side until cementum has been extensively developed. The consequence is that in the worn tooth the enamel fold lingual to each infundibulum is incomplete. The occlusal surface is serrated in a manner that presents a mirror image of the serrations of the



A



B

FIGURE 2: Occlusal surfaces of fourth cheek teeth.
A. Maxillary B. Mandibular.

upper arcade. Each of the mandibular cheek teeth has two relatively short roots with the exception of the last which usually has three.

Each mandibular arcade is straighter than its maxillary partner but they are similar in that the distance across the mouth from the first teeth is narrower than between the sixth teeth (Figure 1). In general the mandibular arcades are 30% closer than the maxillary arcades so that the lingual margin of the mandibular cheek teeth lie medial to the lingual aspect of the maxillary cheek teeth.

The horse is therefore equipped with an efficient dental apparatus for grazing and grinding food in which the hypsodont nature of its cheek teeth ensures that the arcades are maintained throughout its life. The deposition of crown cementum and the close apposition of individual teeth in each arcade gives a grinding system which presents essentially one complex occlusal surface.

(2) Dental Histology

Dentine Dentine is the major component of the tooth, it consists of 72% inorganic material and 28% organic material. It is harder than compact bone and in section it is seen to be formed of a mass of fine canals or tubules - the dentinal tubes. Dentine formation is cyclical and not regular so that in sections of a fully developed tooth growth lines can be seen (Owen's lines). Each tubule may be up to four microns in diameter with the tubules of greater diameter forming peripheral rings conforming to the intricate shape of the tooth within the peripheral enamel.

In the peripheral layers of dentine the tubules may branch or fuse together.

A process of the dentine-forming cell, the odontoblast, occupies each tubule. These processes are termed Tome's dentinal fibres. A thin sheath (Neumann's sheath) surrounds each tubule and because it contains less collagen than the intertubular material produces a more refractive zone around each tubule when examined by light microscopy. The remainder of the intertubular matrix consists of a meshwork of collagen fibres embedded in a calcified ground substance.

There are no nerve fibres or blood vessels within the dentine. Sensations of touch, cold and pH changes are conducted by the Tome's dentinal fibres. Odontoblasts covering the pulp cavity remain viable throughout life and respond to irritation by producing layers of secondary dentine thus protecting the pulp from exposure as a result of attrition.

Enamel The unique gross structure of the enamel pattern of the tooth of the horse has been described previously (page 23). Its microscopic appearance and chemical composition are of a protein nature, the remainder being inorganic salt. Of the inorganic material 90% is calcium phosphate.

Enamel consists of myriads of uniformly wide crystals of hydroxyapatite packed into an organic matrix. A repetitive pattern of change in the orientation of its constituent elements is responsible for its division into so-called prisms and interprismatic areas. The crystals of hydroxyapatite are formed as an extra-cellular secretion from the ameloblast cell layer (Fernhead 1960; Watson 1960). Each prism of enamel is formed

perpendicular to the surface of the dentine. Confirmation of the pattern of crystallite orientation resulted from scanning electron microscopy of developing teeth from mammals (Boyd 1967).

Enamel formation is completed prior to the eruption of the tooth and at this stage the enamel anlage has a series of integuments. Huxley (1856) named these integuments Nasmyth's membrane, in tribute to Nasmyth who described them in 1839 as the persistent dental capsule (Dawes, Jenkins and Tonge 1963).

In the horse as the tooth remnants and the integuments of embryological origin are worn away the enamel acquires secondary cuticles as in other species. In animals with brachydont teeth these cuticles are produced by a mucin plaque, food debris, bacteria and calculus. In animals with hypsodont teeth the major structure surrounding the exposed enamel is peripheral crown cementum. In brachyont species cementum is confined to the roots of the teeth.

Cementum In chemical composition cementum is very similar to bone being two thirds inorganic and one third organic material. Its microscopic structure is also similar to compact bone having an intercellular matrix or ground substance, canaliculi and cell lacunae containing cementocytes structured to resemble an haversian system. The matrix is eosinophilic in nature. The peripheral cementum is formed continuously from modified odontoblasts - cementoblasts, within the alveolar periodontal membrane. This cementum is nourished by blood vessels and lymphatics within the periodontal membrane. The cementum within the enamel invagination of the maxillary cheek teeth is deprived of its blood supply at eruption and cannot receive material from

the pulp or dentine because of the impervious avascular nature of the formed enamel and dentine. Sections of this cementum therefore show empty cell lacunae and canaliculi which can become packed with cellular material from the animals' food (Fig.3).

(3) Tooth Attachment

Each tooth is independently and firmly attached to the alveolar processes of the mandible and maxilla within an alveolus (socket) that is formed from the connective tissue of the dental sac and the surrounding bone of the jaw. The teeth are suspended and attached by bundles of connective tissue fibres referred to as the periodontal membrane or the periodontal ligament.

The arrangement of fibres in the periodontal ligament is complex and dense bundles of collagen run in various directions from the bone of the socket wall to the cementum covering the roots and reserve crowns. The embedded portion of these fibres are called Sharpey's fibres. The periodontal ligament contains blood vessels and nerves and the collagen bundles are arranged to protect these vessels and those of the pulp from ischaemia that might result from occlusal pressure. In this way the tooth is suspended firmly within the alveolus and at the same time is permitted some slight movement within its alveolus (Fig. 4).

Because of the relative sizes of the teeth when compared with the bone width of the mandible or maxilla there is only a thin plate of bone covering the lateral aspects of the roots and reserve crowns. In the boiled out skulls of young horses it is common to see areas of apparent maxillary and mandibular porosis over these areas (Figure 5).

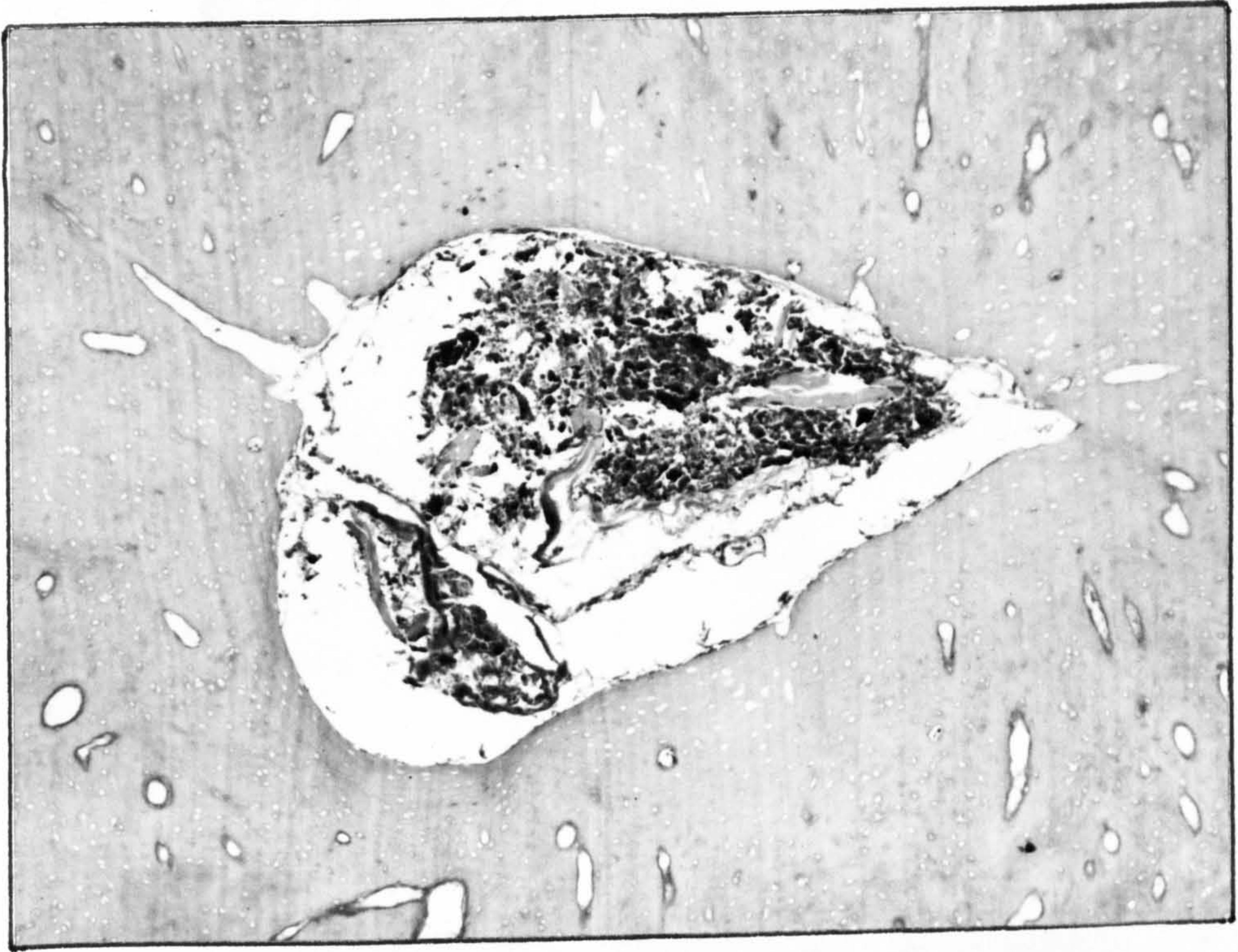


FIGURE 3: Maxillary enamel lake cementum.
Acellular cementum and debris
filled infundibulum.
Decalcified H and E x 55.

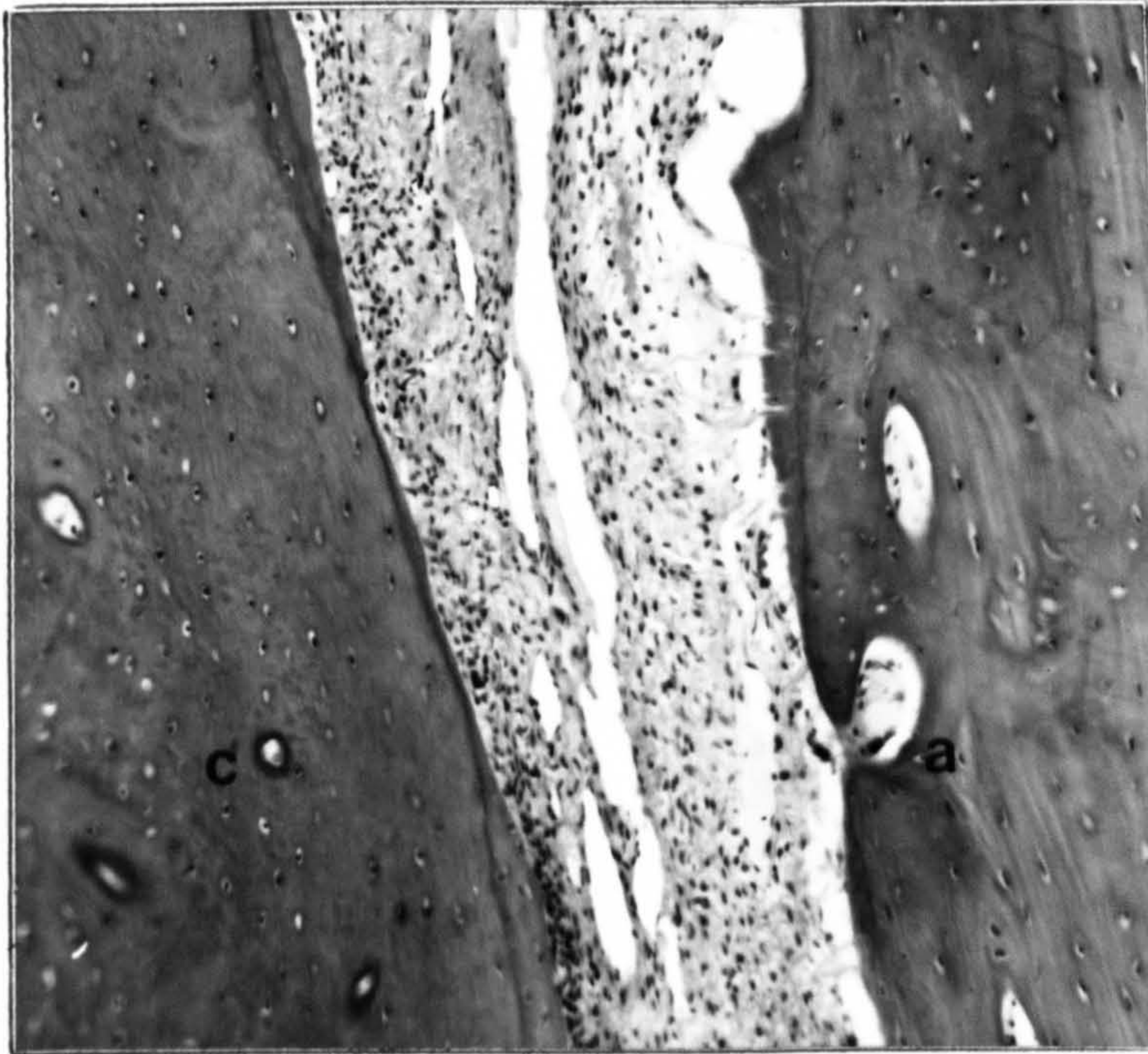


FIGURE 4: Vertical section of periodontal ligament.
C = Cementum. A = Alveolar bone.
Decalcified H and E x 110.

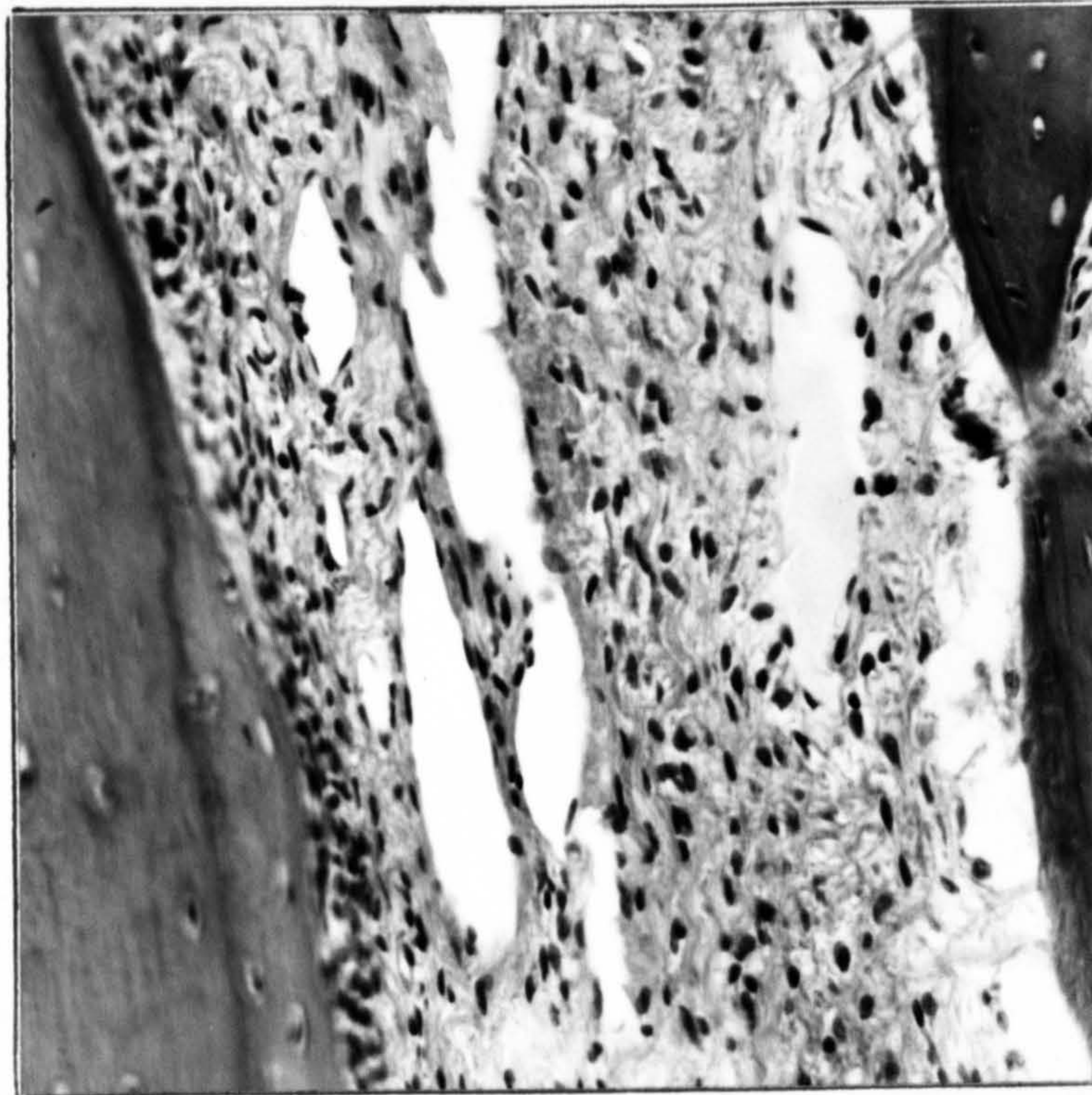


FIGURE 4a: High power of Figure 4.
Periodontal fibres inserting into
bone and cementum. x 250.



FIGURE 5: Maxillary porosis associated with tooth eruption in a two year old pony.

The mucous membrane of the mouth covers the alveolar processes externally. This covering, the gums, is composed of dense fibrous tissue which is intimately connected with the periosteum. The gums are covered by a smooth stratified squamous epithelium and have a few glands.. In addition they are relatively insensitive and do not bleed easily (Baker, 1970). Extending from the epithelial attachment of the gum to the crown of the tooth is a free margin of gum enclosing the gingival crevice. This crevice is continuous between adjacent teeth of the arcade, but is reduced by the intimate contact of the teeth at the interproximal areas.

These arrangements are modified during the eruption of the teeth. It is assumed that in the continually erupting teeth of the horse that movement of the reserve crown is accommodated by re-arrangement of the collagen bundles of the periodontal ligament.

CONCLUSIONS

The horse has evolved an efficient food grinding apparatus and with the remainder of the alimentary canal is suited to a life of almost continuous grazing nature. The lips are prehensile and selective in grazing. The incisor teeth are an efficient cutting apparatus and the modification of the cheek teeth into a functionally single serrated arcade in each jaw. Simpson (1951) showed that during the evolution of Equus caballus, with an increase of size there is a cube factor in the increase of food required, so that a doubling of height requires eight times the food intake. The grinding system of the cheek teeth accommodates such an increase of food intake.

The effects of continuous grazing and mastication result

in dental attrition by the forces of wear and the development of the hypsodont tooth with continuous eruption of reserve crown maintains the functional integrity of each dental arcade.

Tyler (1972) observed that free-ranging New Forest ponies under winter conditions almost all the daylight hours were devoted to grazing and browsing. At the same time the brittle nature of the enamel of the tooth is protected by the surrounding dentine and cementum.

CHAPTER II

DEVELOPMENTAL ANATOMY OF EQUINE CHEEK TEETH

INTRODUCTION

Embryologically a tooth is a greatly modified connective tissue papilla (mesoderm) that has undergone calcification into dentine externally (Owen, 1845), and is capped by a hard enamel crust elaborated from the epidermis (ectoderm). There are no significant differences between the development of the temporary and the permanent teeth (Kupfer, 1937; Arey, 1965). A summary of tooth development relative to its ectodermal and mesodermal origin is shown in Tables 1 and 2.

The following description of the development of the cheek teeth of the horse is based on the dissections and histological sections obtained from the deep-frozen skulls of aborted Thoroughbred foetuses.

MATERIALS AND METHODS

The heads of 65 aborted Thoroughbred foetuses were collected from the Equine Research Station, Balaton Lodge, Newmarket. The specimens were usually deep-frozen and conception dates were known.

The mandibles were disarticulated and separated into individual rami. Similarly the cranium was divided and the

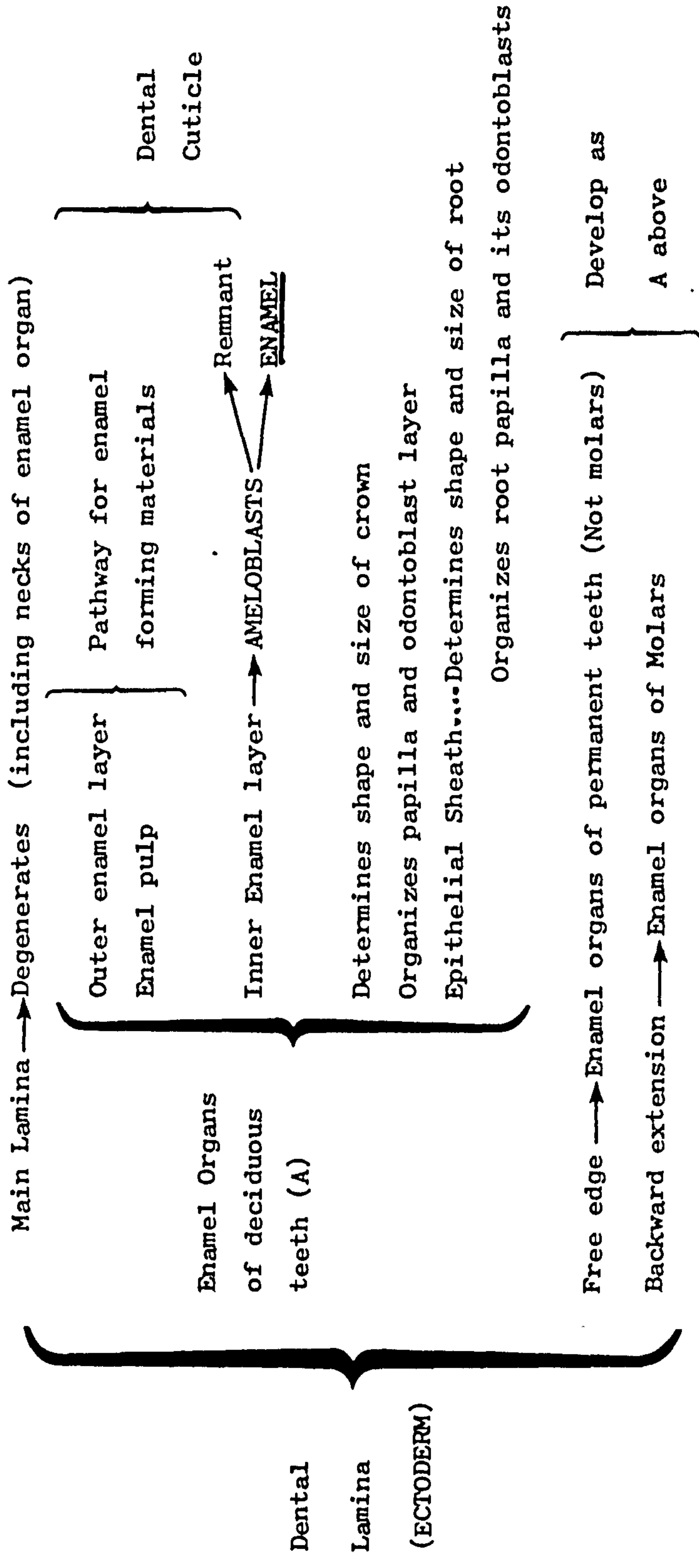


TABLE 1 Dental Embryology: Ectodermal Derivatives (after Arey, 1965)

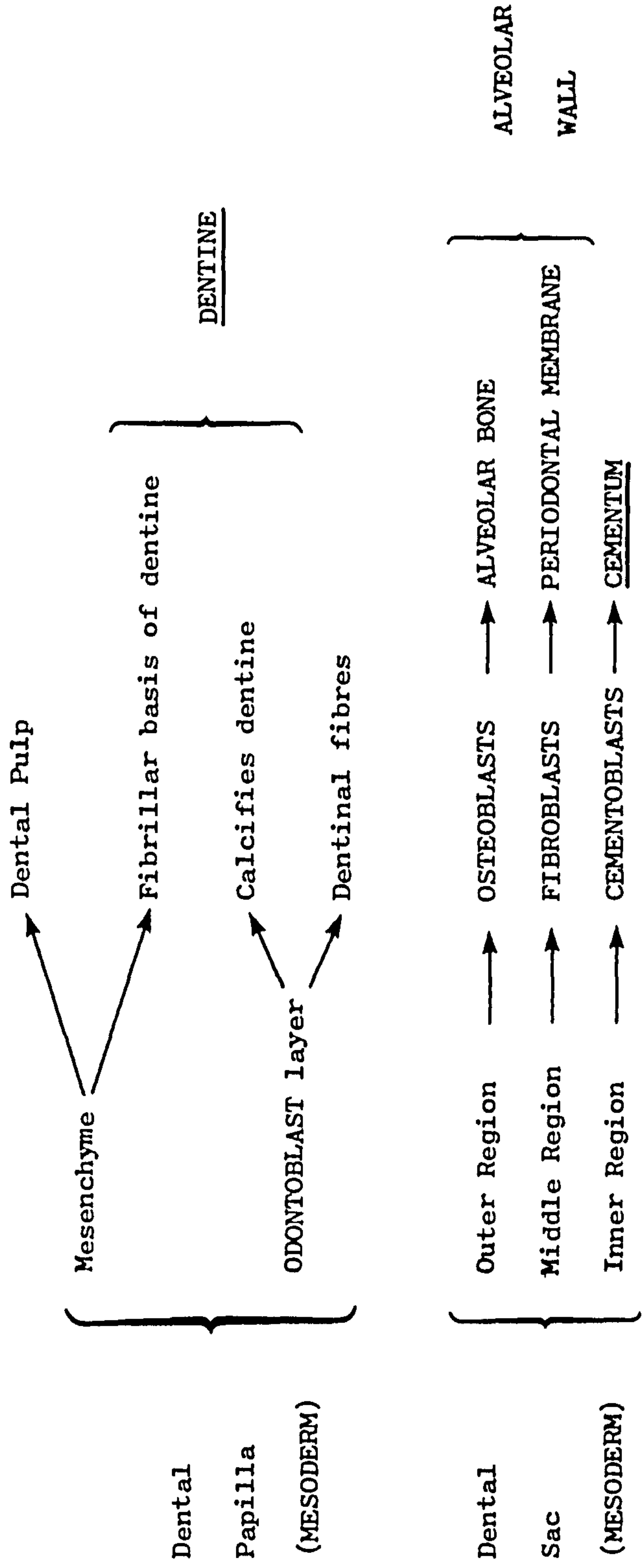


TABLE 2 Dental Embryology: Mesodermal Derivatives (after Arey, 1965)

turbinates removed from the maxillae. Isolated jaws and teeth were dissected, radiographed and sectioned.

Histological Methods

Vertical and transverse 10 μ sections were prepared and stained with haematoxylin and eosin. In specimens younger than 250 days gestation decalcification was not necessary and sections were made using cryostat techniques. Older tooth follicles were found to be difficult to section in this manner and decalcification (as described in Chapter I) was necessary to prevent damaging the microtome blade.

RESULTS AND DISCUSSION

(1) Early Tooth Buds

The earliest specimen examined was a foetus 112 days from conception. At this stage the dental lamina was enveloped by the gum and three distinct cyst-like structures in each mandible and maxilla represented the developing tooth bud (Figure 6). Their relative sizes indicate that it is the bud of the second premolar that leads the development sequence. Within each tooth bud a cupped enamel organ overlays the dental papilla (Figures 7a and 7b).

(2) Enamel Organ

The shape of any tooth at its stage of eruption is basically controlled by the developmental folds that take place in the enamel organ. The details of the folding of maxillary and mandibular enamel organs of the horse were first detailed by Kupfer (1937) in his study of tooth structure in horses and donkeys.

In the series of specimens examined of foetal ages 112 days, 120 days, 134 days, 182 days, 200 days, 210 days, 250 days, 285 days,

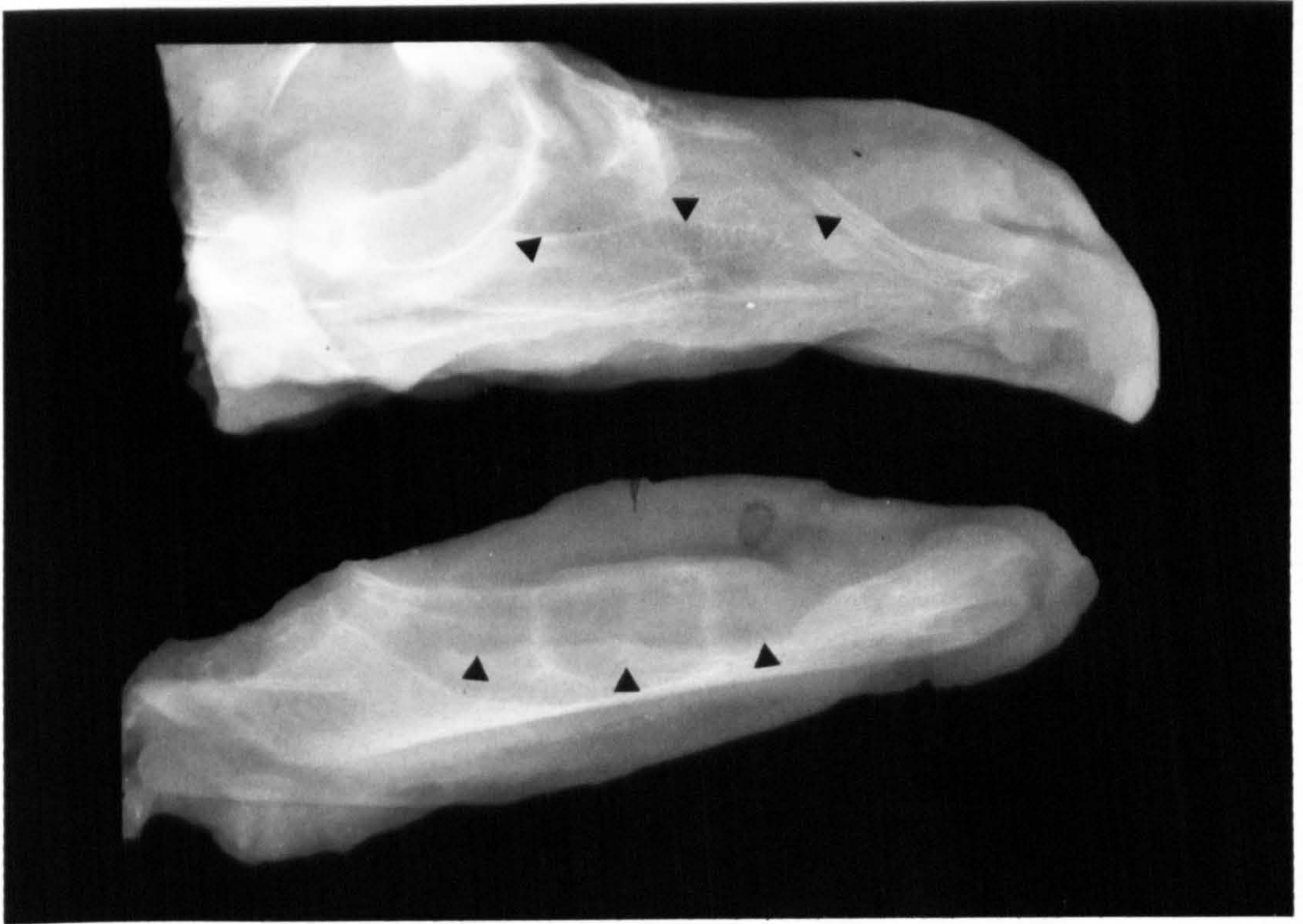


FIGURE 6: Radiograph of 120 day Thoroughbred foetal maxilla and mandible showing developing tooth buds.

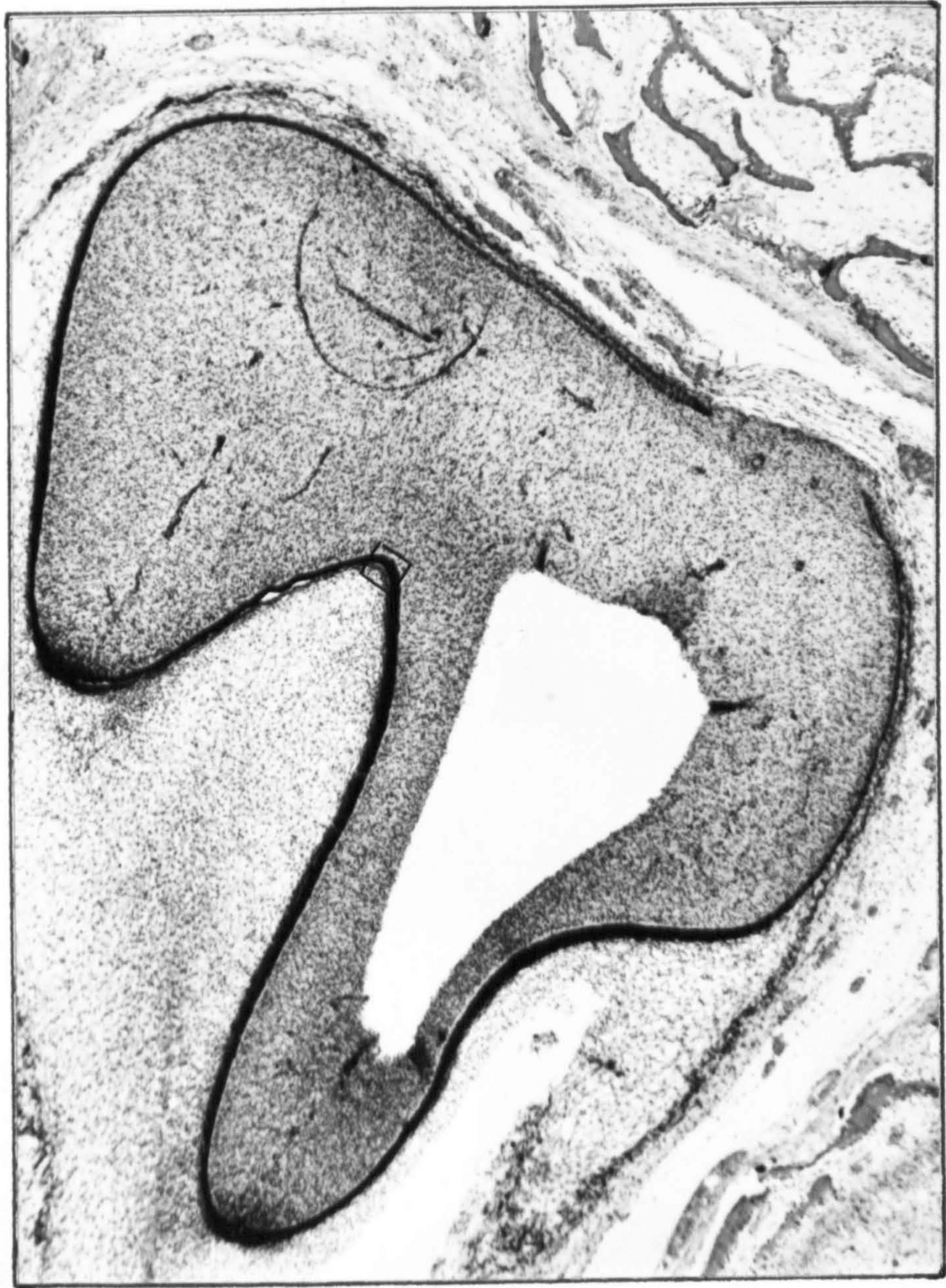


FIGURE 7a: Cryostat section of maxillary tooth bud from 120 day Thoroughbred foetus.
H and E x 25.

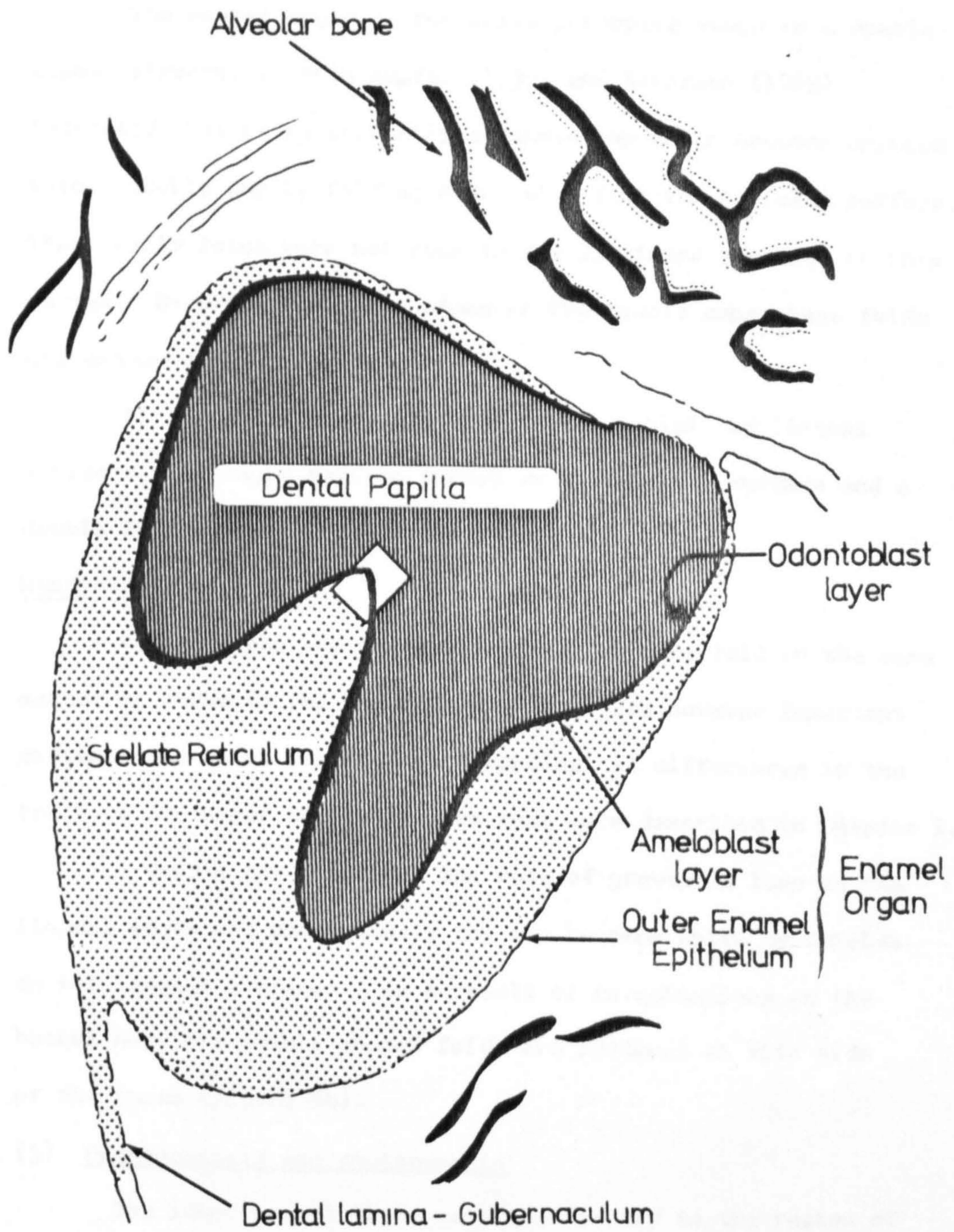


FIGURE 7b: Diagram of developing equine cheek tooth.

300 days, 325 days, new born foals, weanlings and yearlings these folds were found to be as follows.

Maxillary Teeth

The enamel organ at the initial cupping stage is a double cupped structure. Both Kupfer (1937) and Espersen (1962) indicated that it is initially a single cup which becomes changed into a double cup by folding over twice from the occlusal surface. These early folds were not seen in any specimens examined in this series. By growth from the edges of the double cups these folds are deepened.

At the same time folds form in the labial and lingual surfaces. A single fold is formed on the lingual surface and a double fold on the labial surface (Figure 8a).

Mandibular Teeth

In principle the mandibular enamel organs fold in the same manner as those in the upper jaws. There are however important distinctions which lead to the morphological differences in the teeth of the upper and lower jaws that were described in Chapter I.

A double cup forms but the rate of growth is less on the lingual edges and consequently the cup becomes split by infolds on the lingual surfaces. As a result of invaginations on the buccal surfaces double enamel folds are produced on this side of the tooth (Figure 8b).

(3) Odontogenesis and Amelogenesis

The inner enamel layer matures, firstly in the region of the future crown, into tall columnar cells that are designated ameloblasts. Individual ameloblasts are tall columnar cells with an oval nucleus situated close to the basement membrane.



FIGURE 8a: Labial view of 280 day Thoroughbred foetal maxillary (A) and mandibular (B) enamel anlagen.



FIGURE 8b: Occlusal view of 8a.

The cells of the dental papilla, at their interface with the inner enamel layer, develop into the dentine secreting cells, the odontoblasts (Figure 9). Each odontoblast has a long cell body extending through a terminal web into the odontoblast process within the dentine itself. The ultrastructure of both ameloblasts and odontoblasts has been described by Weinstock and Leblond (1974).

Odontoblasts may be separated from each other by clefts containing collagen fibres and later, capillaries. Initially the odontoblasts are separated from the ameloblasts by only the basement membranes. The odontoblasts deposit a collagen rich material comprising fibrils, Korff's fibres (Korff 1904), which become orientated perpendicular to the basement membrane (Figure 9). Dentine that is formed is produced as a matrix (predentine) and is calcified within 24 hours of its secretion. It is produced in layers and the cytoplasmic processes of the odontoblasts become trapped within the calcified matrix in canals known as dentinal tubules. As further layers of dentine are secreted so the odontoblasts are removed from their original position but maintain their contact with the basement membrane by elongation of the odontoblastic processes.

The first thin layer of dentine that is produced appears to be the stimulus for the ameloblasts to produce enamel. It forms a poorly calcified matrix which later becomes almost totally calcified. Enamel forms in rods that retain the shape of the cells so that they are prismatic.

The detailed foldings of the enamel organ have been described and this shape is maintained as dentine and enamel are deposited upon

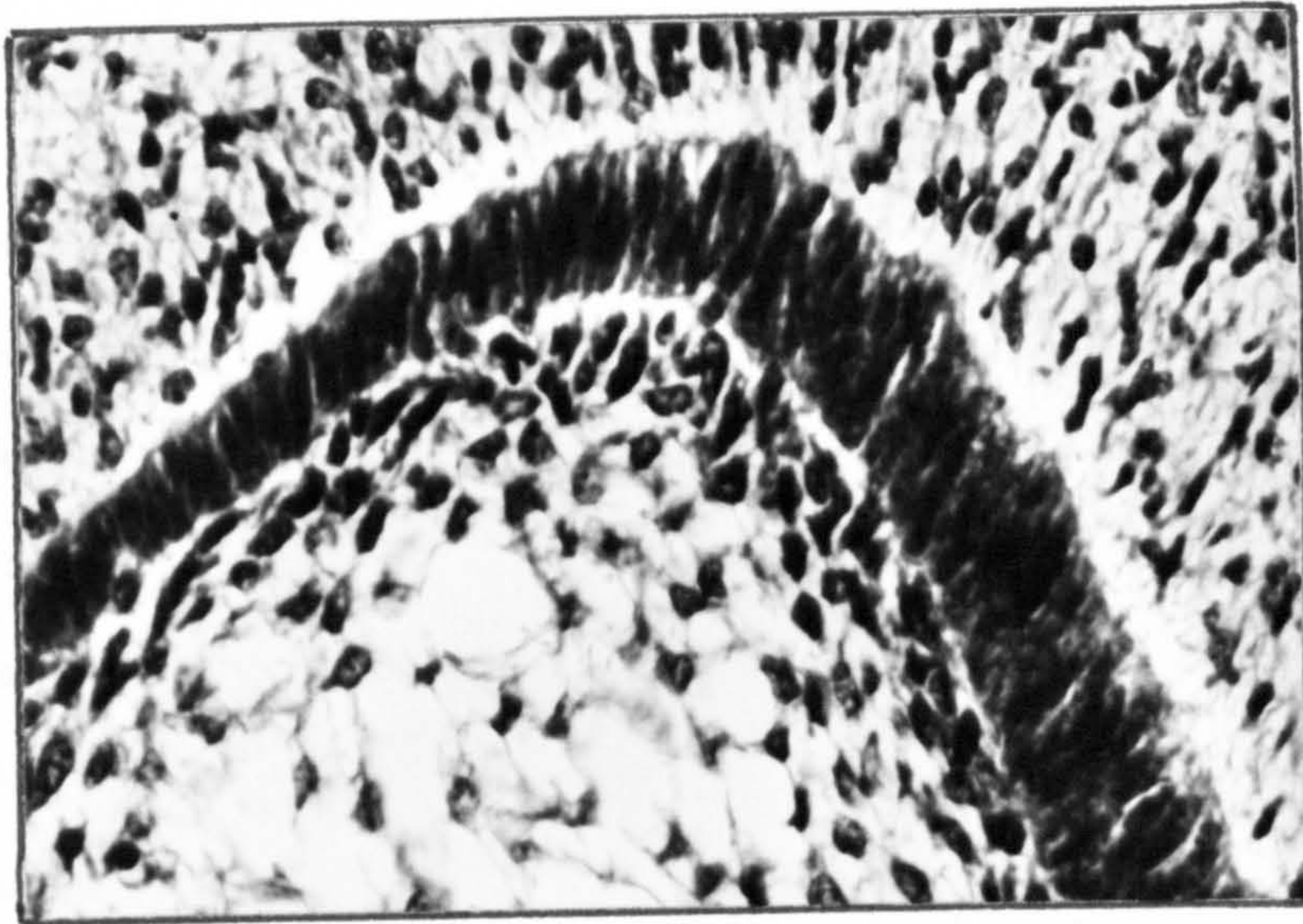


FIGURE 9a: Odontoblast and ameloblast layers.
Enlarged from 7a. x 550.

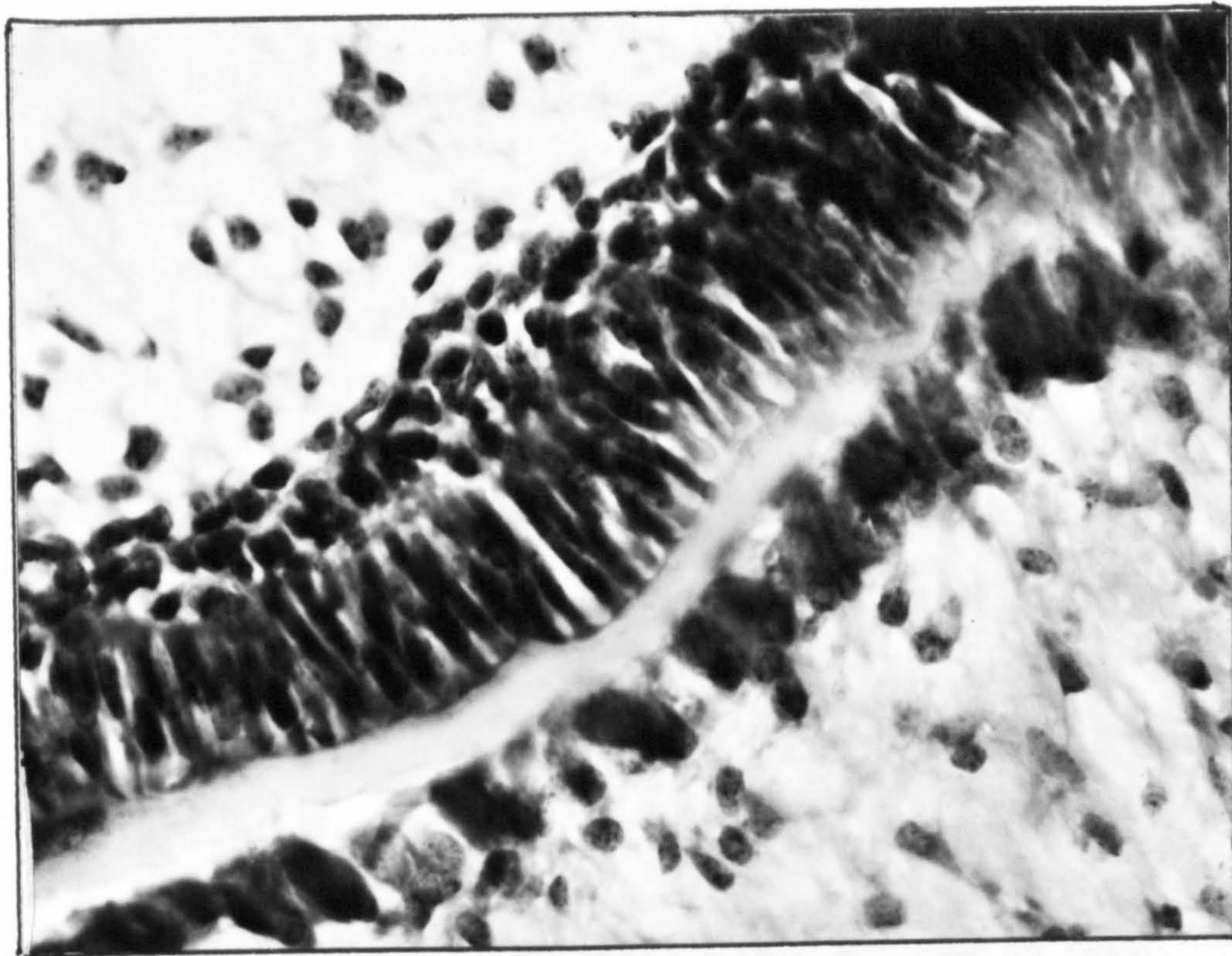


FIGURE 9b: Predentine separating odontoblast and
ameloblast layers. x 950.

the future crown down to the root. The first radiographic signs of calcification within the tooth were seen at 120 days, and it was concluded that it takes a total of 240 days for the completed enamel anlage to form. The wave of formation and maturation of the tooth buds of the permanent teeth is first seen at 9.5 months of foetal life. Calcification within the bud of the fourth cheek tooth (M1) is seen from 10 months foetal age, the maxillary bud precedes the mandibular bud by up to 3 weeks. Subsequent enamel anlages form in sequence, each taking 360 days to develop from bud to eruption (Figures 10a and 10b).

(4) Cementogenesis

The mesenchymal tissue that surrounds the developing tooth is continuous with that of the dental papilla and it differentiates into connective tissue making up the dental sac. In the area of the future root (see Eruption and Maturation) its inner cells differentiate into cementoblasts and as the epithelial sheath (outer enamel cell layer) disintegrates these cells deposit root cementum. These changes can be seen from 280 days of foetal life.

At an earlier stage, from 210 days foetal age, cellular changes can be seen around the occlusal margins of the central invaginations of the enamel anlage of the maxillary cheek teeth. Cementoblasts arising from the mesenchymal cells of the dental papilla break through the epithelial sheath and come to rest beneath the ameloblast layer. In this way a collar of cementoblasts forms within the enamel invagination (Figure 11). These cells are nourished from the dental sac which develops capillaries within the stratum reticulare. The gubernacular cord and later the infundibulum are the remnants of this vascular



FIGURE 10a: Radiograph of maxilla of three week old Thoroughbred foal showing deciduous teeth and M1 tooth bud.



FIGURE 10b: Maxilla of 11 month old Thoroughbred foal. M1 is now in wear and PM2 and M2 developing.

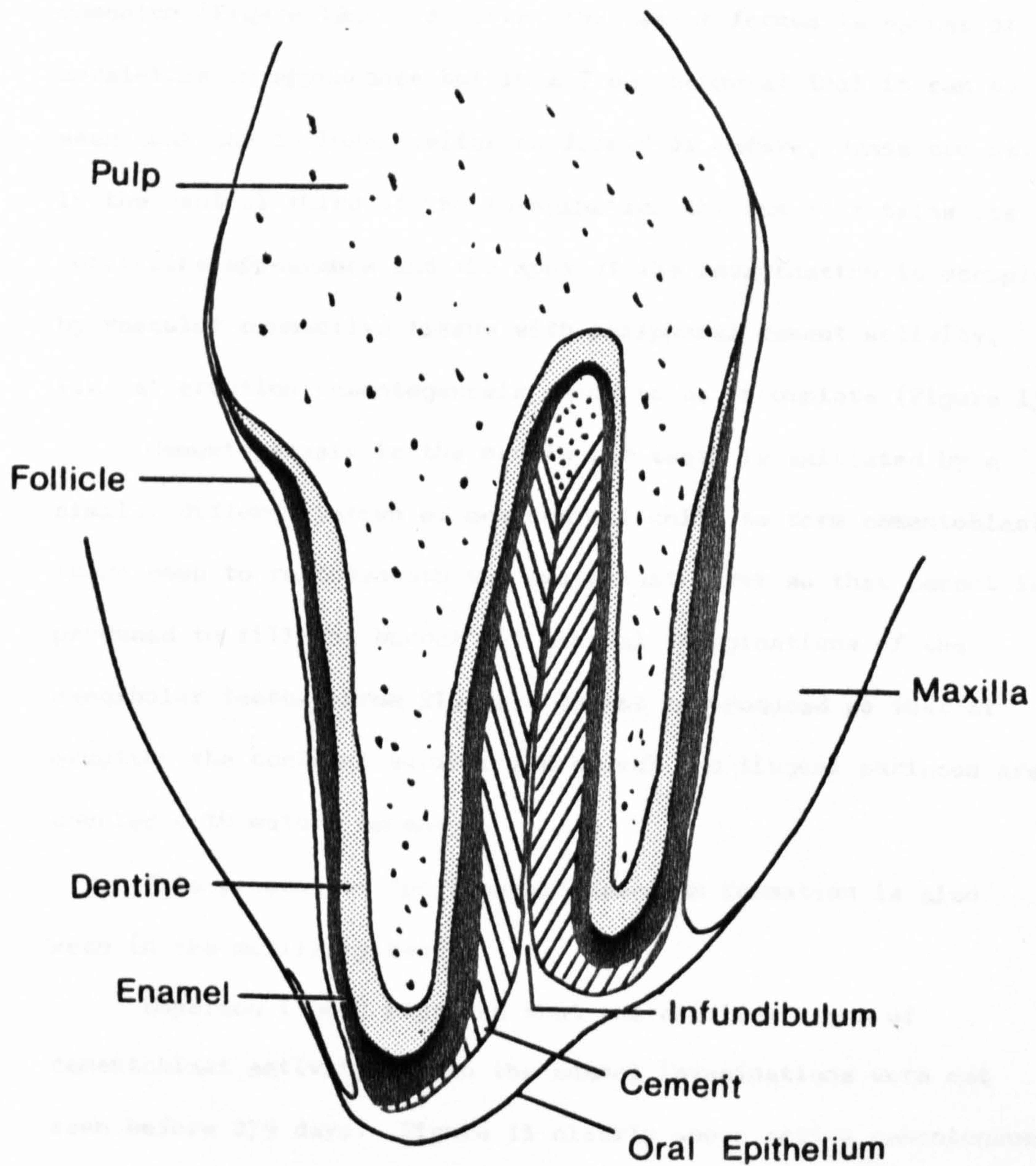


FIGURE 11: Diagram of vertical section of enamel lake of developing maxillary cheek tooth.

pathway into the enamel invagination that is closed at its apex.

By 280 days (9 month foetus) a discreet cement collar can be seen within the anlage (Figure 12). Subsequently these areas expand to fill the depths of each enamel invagination with cementum (Figure 12a). At first the cement formed is sponge or coral-like in appearance but in a 7 day neonatal foal it can be seen that the occlusal collar is formed of mature, dense cement. In the central third of the invagination the cement retains its coral-like appearance and the apex of the invagination is occupied by vascular connective tissue with peripheral cement activity, i.e. at eruption cementogenesis seems to be incomplete (Figure 13).

Cementogenesis in the mandibular teeth is initiated by a similar differentiation of mesenchymal cells to form cementoblasts. These come to rest beneath the ameloblast layer so that cement is produced to fill the buccal and lingual invaginations of the mandibular teeth. From 210 days cement is produced so that at eruption the occlusal surface and buccal and lingual surfaces are covered with mature cementum.

This process of "peripheral" cementum formation is also seen in the maxillary teeth.

Esperson (1962) reported that the earliest signs of cementoblast activity within the enamel invaginations were not seen before 279 days. Figure 11 clearly shows active cementogenesis at 240 days, and the first stage at which cementoblasts could be recognised beneath the ameloblast layer was 210 days. Joest, quoted by Becker (1962), recognised that some molar teeth may show incomplete cementogenesis (cement hypoplasia) at eruption and indicated that this could lead to dental decay or dental fracture

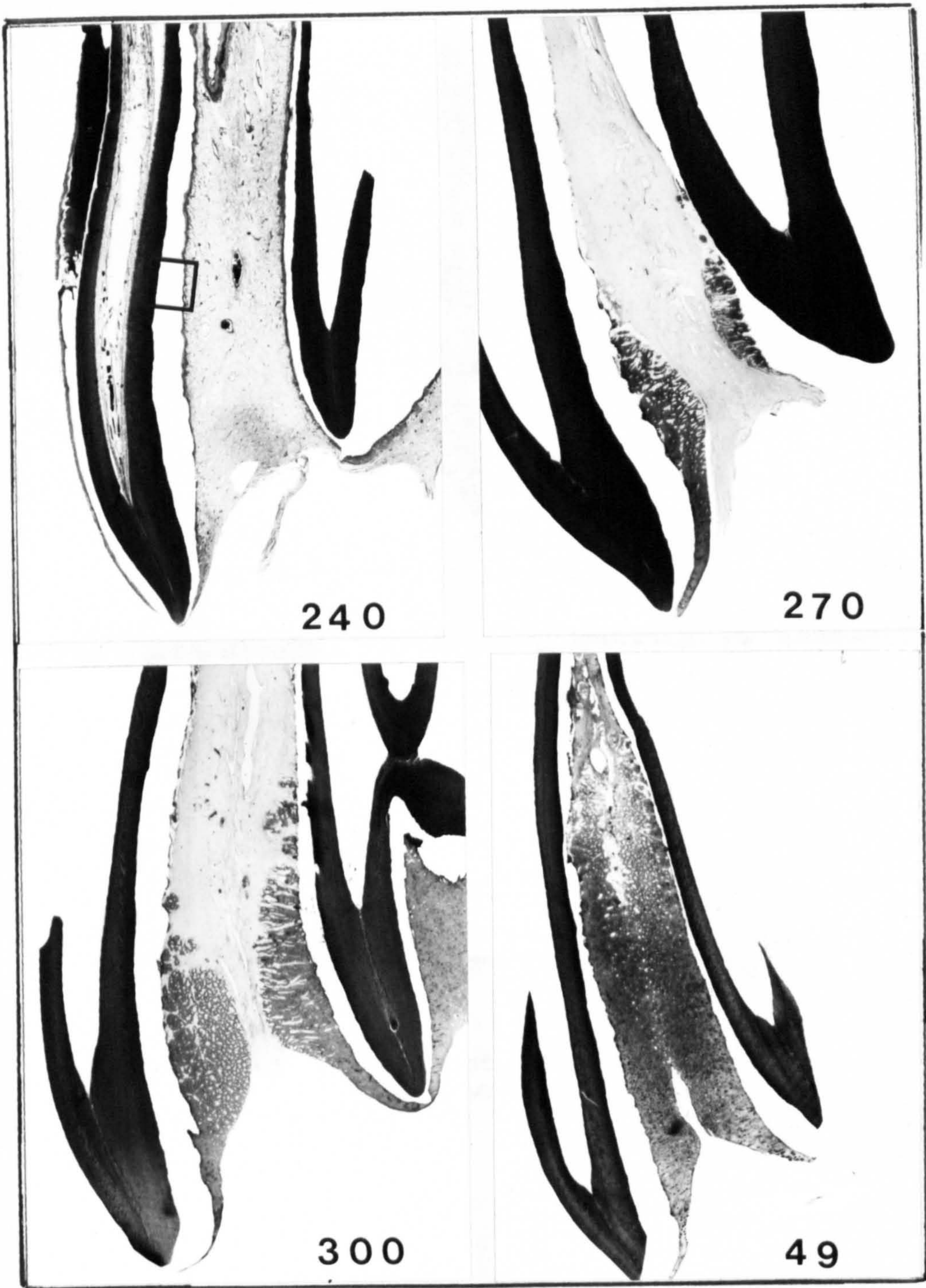


FIGURE 12:

Four stages of enamel lake cementum formation.

240 day foetus x 8.25

270 day foetus x 8.25

300 day foetus x 8.25

49 day neonate x 4.25

Decalcified H and E.

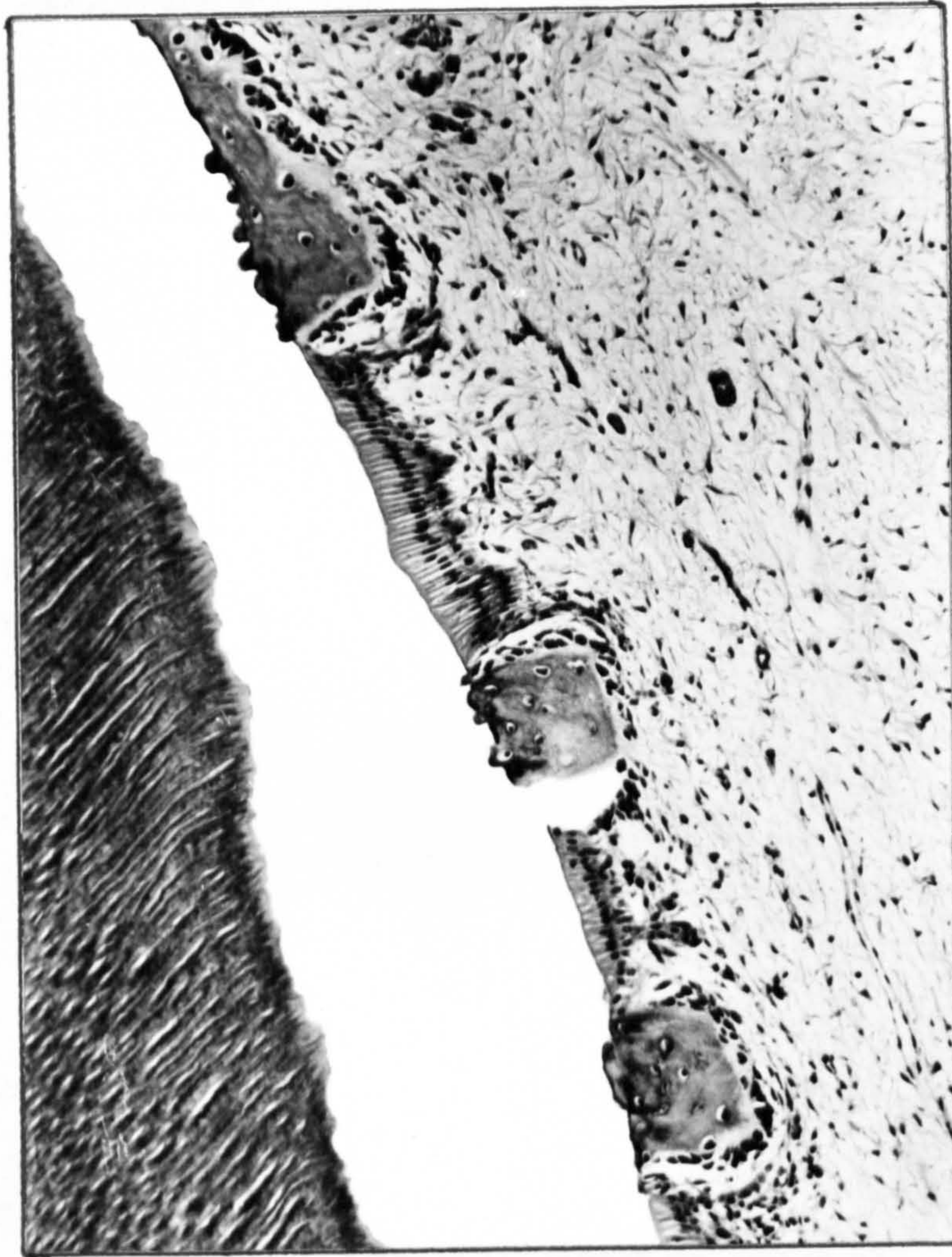


FIGURE 12a: Cementoblast activity in ameloblast layer of 240 day foetus from figure 12. x 150.

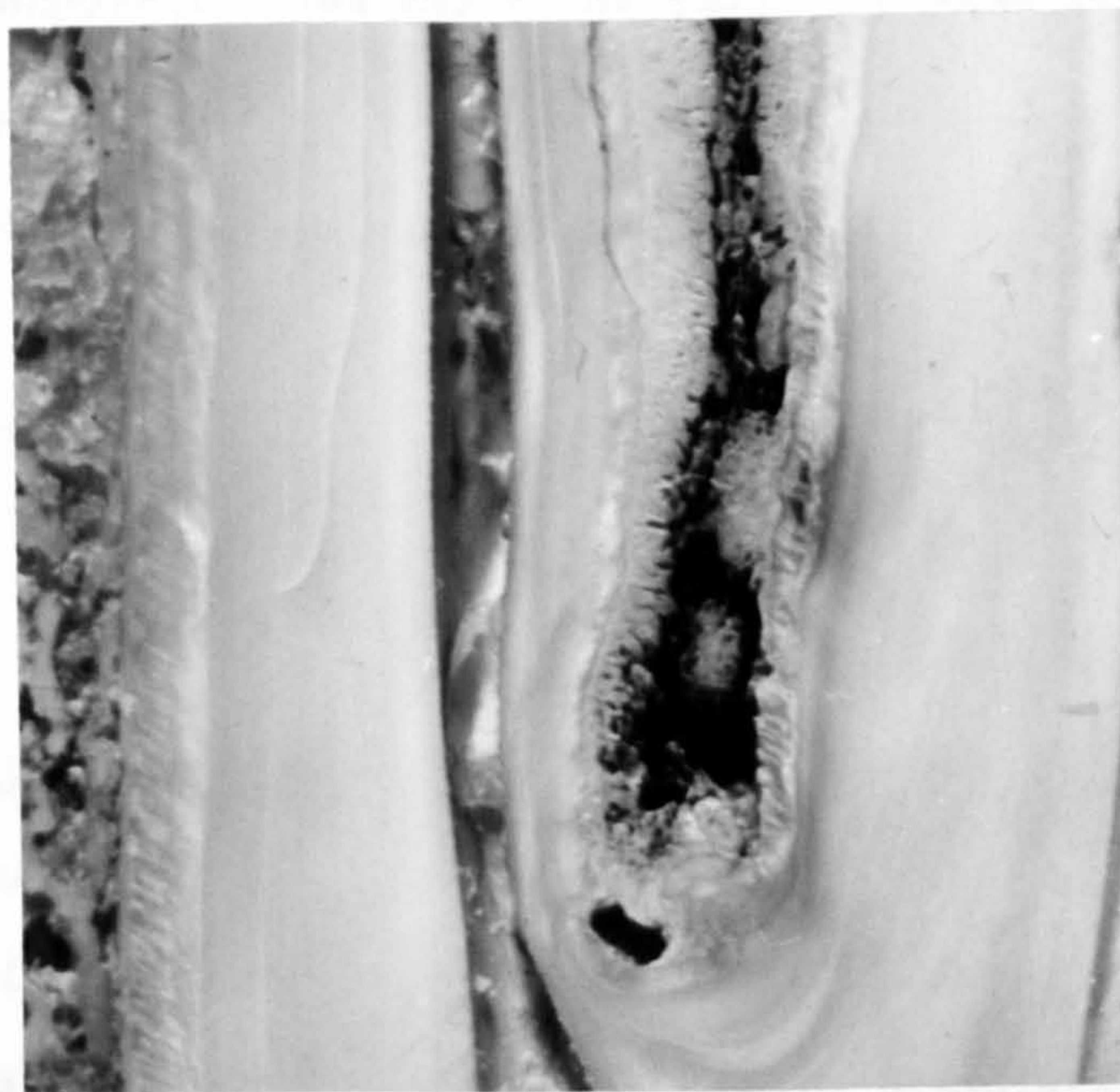


FIGURE 13: Vertical section of third cheek tooth from a five year old showing hypoplasia of cementum.

but that it was comparatively infrequent. Becker (1945) recorded 0.38% incidence in 30,000 military horses and Herzog (1937) reported an incidence of 1.8% in 500 horses aged between 4 and 22 years. Baker (1974), after sectioning a series of erupted teeth, reported that, although the occlusal surfaces appeared normal in all cases of the teeth examined, up to 43% of cement lakes had a residuum of connective tissue at the apex of each invagination. At this stage cementoblast activity was not detected within these areas and in many cases food debris was packed within this space (Figure 14). No evidence was seen of cement hypoplasia in mandibular teeth.

(5) Eruption and Maturation

The eruption times of the teeth of the horse have been listed in Chapter I. The timing of tooth eruption and rate of occlusal wear of the teeth (particularly the incisors) is used as the basis of the ageing of horses (Goubaux and Barrier, 1892; Huidkoper, 1903). The rate of wear of the cheek teeth has been studied radiographically by Westhues (1942) and by dissection and measurement of specimens by Baker (1971) in the horse, and by Klingel and Klingel (1966) and Spinage (1972) in the Plains Zebra (Equus quagga boehmi Matschie).

The general mammalian pattern of the sequence of cheek teeth eruption is as follows:- deciduous premolars 1-4 followed by the molars 1-3 and finally the permanent premolars 1-3, so that three eruption waves form in a rostro-caudal direction. Sisson (1910) lists the horse as varying from this sequence as far as the six permanent cheek teeth are concerned as shown in Table 3. It should be noted that there is a broad range of variation in



FIGURE 14: Vertical section of cement lake showing hypoplasia of cementum and remnants of gubernacular connective tissue. Decalcified H and E x 55.

Cheek Tooth Number	Mammalian Eruption Sequence	<u>Equus</u> <u>caballus</u>
1	4	3
2	5	4
3	6	6
4	1	1
5	2	2
6	3	5

TABLE 3

Eruption Sequence of Permanent
Cheek Teeth of the Horse

the normal eruption times because of inherent biological variation. Eruption times are also affected by familial variations, the effects of endocrine glands, nutrition and intercurrent disease.

The essential forces which result in the penetration of the gum by teeth have been the subject of much theory and experimentation (Massler and Schour, 1941; Cahill, 1970). Noyes, Schour and Noyes (1938) described six anatomical stages in the eruption of the teeth.

Stage I : Preparatory stage (opening of the bony crypt)

Stage II : Migration of the tooth toward the oral epithelium

Stage III : Emergence of crown tip into oral cavity
(beginning of clinical eruption)

Stage IV : First occlusal contact

Stage V : Full occlusal contact

Stage VI : Continuous eruption

From experimental animal studies and clinical observations, evidence has been published to support the theory that eruption is a result of pressure due to the vascularity of the periapical tissues (Massler and Schour, 1941; Bodingbauer, 1960; Ham, 1974). In the young horse these changes can be seen and palpated as symmetrical swellings along the ventral border of each mandible and along the maxilla. Radiographically cystic distension of the lamina dura accompanies eruption (Figure 15). Baker (1971) referred to these structures as pseudo-cysts and noted that with maturation there is a remodelling of both the mandibles and the maxillae by 8 years of age.

With occlusal contact there is attrition of the dental arcade surfaces so that the styles of the teeth are removed and the characteristic tooth surfaces exposed (Figure 2). As the

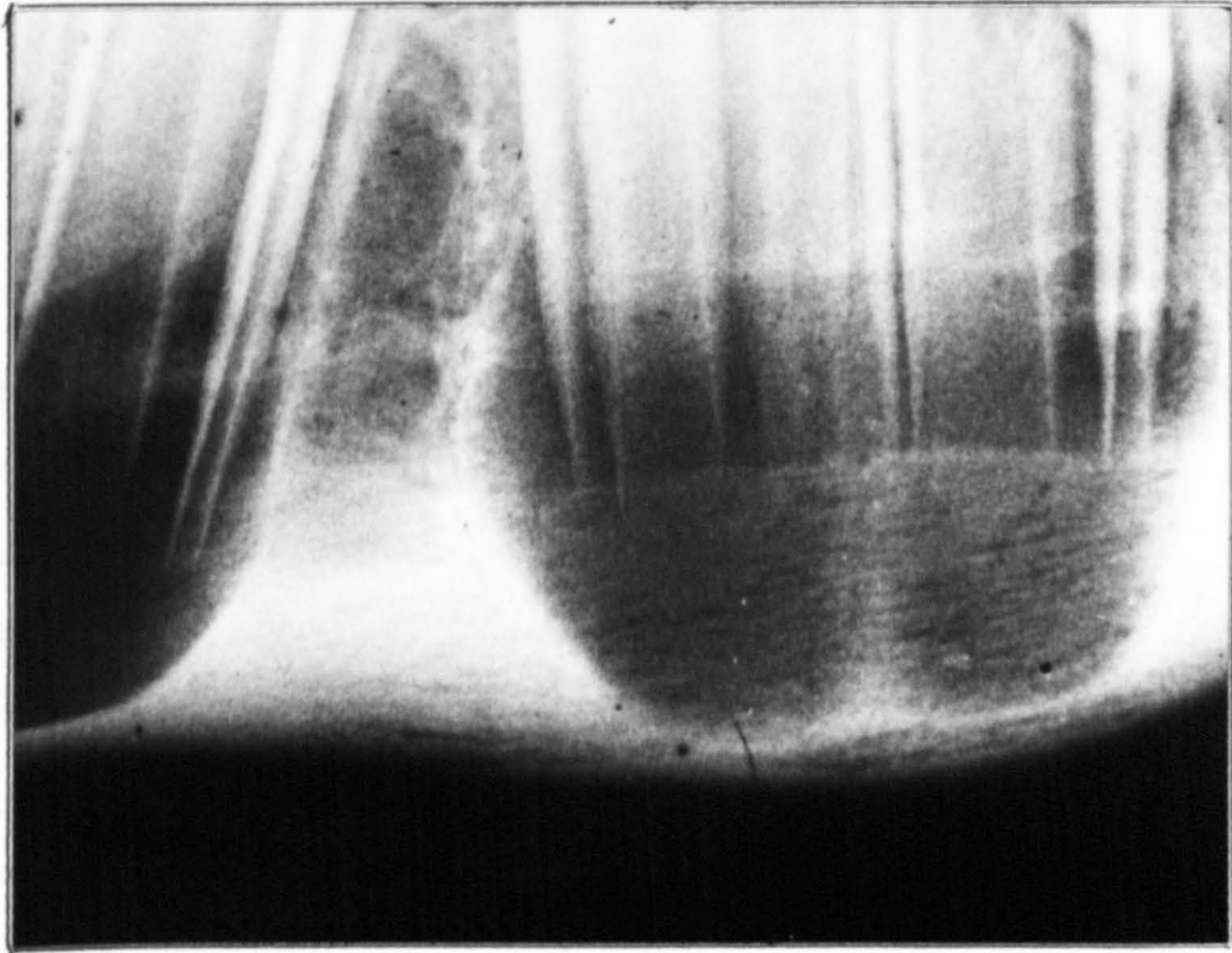


FIGURE 15: Radiograph of erupting third mandibular permanent cheek tooth showing cystic distension of the lamina dura.

deciduous teeth are replaced the shell of the deciduous tooth may be retained as a cap for some months before finally being dropped out of the mouth.

The forces of occlusal contact and the side to side masticatory jaw movements produce the transverse ridges across the occlusal surfaces (see Chapter III).

As the crowns are worn away with age the cheek teeth continually erupt so that the dental arcades are maintained. In this process the reserve crown is reduced and the true roots are lengthened (Figure 16). In extreme old age the teeth are finally lost. Under normal circumstances dental attrition due to wear occurs at a rate of 3 mm per year. Consequently it can be appreciated that with an effective tooth length of 80-90 mm and a deciduous arcade lasting up to 4 years, that after 30-34 years the domestic horse will be without teeth. Records show that some horses may survive beyond this time with special diets. It has been suggested that the rate of attrition in donkeys may be less than in the horse so that ages of 40 years may be obtained (Svendson 1978, personal communication).

CONCLUSIONS

No specimens were available for investigation of the earliest stages of dental lamina, tooth bud and dental follicle formation. By 112 days the enamel organ had already achieved a double folding and the simple enamel cup stage as described by Kupfer (1937) was not seen. Espersen (1962) has supported Kupfer in reporting that cementogenesis within the enamel invaginations of the maxillary teeth does not start before 279 days of gestation.



FIGURE 16: Radiographs of equine mandibles
upper - four year old Thoroughbred
lower - 25 year old Thoroughbred.
Note increase in length of true roots
and alveolar periodontitis.

In the sections examined in this investigation migration of cementoblasts from the connective tissue of the dental papilla was seen as early as 210 days and by 240 days calcification of the matrix of the cementum was seen.

The formation of cementum within the enamel invaginations of the maxillary teeth is generally incomplete at the time of eruption of the teeth. At eruption the vessels supplying the cementum through the gubernacular cord are stressed and finally destroyed by occlusal contact. From this time cementogenesis within these areas stops and as a result no living cementocytes are found within the lacunae of the cementum of the enamel lakes of teeth in wear.

CHAPTER III

SURVEY OF DENTAL DISEASE

INTRODUCTION

The aim of this study was to gather data on the prevalence and pathogenesis of the dental diseases of the horse. The British Equine Veterinary Association (1965) reported that in their survey dental disorders accounted for nearly 10% (1608 out of 17,268) of the requests for veterinary attention. It should be noted that an undefined proportion of these requests would in fact be owner/trainer requests for the rasping of teeth. Consequently the 10% figure will be biased upwards. Silbersiepe (1954) recorded that 6% of horses admitted to the Berlin Clinic were treated for dental disease. It seemed likely therefore that a clinical prevalence of dental disease in the horse would be in the 6-10% range.

It could be anticipated that a post-mortem survey involving detailed dissection and radiology would show a much greater prevalence of dental disease.

MATERIALS AND METHODS

Morbid Anatomy

During the period 1967 to 1969 fresh horse skulls were collected from abattoirs and knacker houses in Croxley Green and Abbots Langley, Hertfordshire. Subsequently more material was gathered from Motherwell and from the University of Glasgow Veterinary Hospital.

The specimens were examined in the fresh state and the following data recorded:-

1. Age, sex and type of horse. These results were inferred from the size of the skull and the state of eruption and type of teeth present (Sisson and Grossman, 1910; Habermehl, 1961; American Association of Equine Practitioners, 1966).
2. Reason for destruction.
3. Details of dental disease
 - (a) Presence or absence of first premolar i.e. Wolf tooth
 - (b) Abnormalities of eruption
 - (c) Abnormalities of wear
 - (d) Diseases of the gum and periodontium
 - (e) Diseases of tooth substances.

The major sub-divisions of the diseases of the gum (d) and diseases of the tooth substances (e) were graded to indicate the severity of the lesion present. Periodontal diseases were graded using 1 (+) to 4 (++++) plus system as follows:-

- + = local gingivitis with hyperaemia and oedema
- ++ = erosion of gingival margin
- +++ = periodontitis with gum retraction
- ++++ = gross periodontal pocketing and destruction of alveolar bone.

For diseases of the tooth substance the system devised by Honma, Yamakawa, Yamauchi and Hosoya (1962) was followed:-

- Grade 1 = decay of the cement alone
- Grade 2 = decay of cement and enamel
- Grade 3 = decay of cement, enamel and dentine
- Grade 4 = splitting of the tooth
- Grade 5 = loss of the tooth.

Data were recorded from the individual teeth of each skull. The cheek teeth were numbered 1 to 6 and referred to as maxillary or mandibular in origin i.e. maxillary 1 equals first upper cheek tooth equals premolar 2. The data were recorded on card indexes and subsequently collated and analysed.

Method of Examination

The position of the incisor teeth was recorded and the length of the diastema was measured. The mouth was then opened by division of cheek skin and muscles and the jaws were forced widely apart. The mouth and tooth surfaces were washed clean of food debris and blood by jets of water. The dental arcades and oral mucosae were then examined in detail. Crevices within the teeth and interproximal pockets were probed with dental picks.

Each specimen was numbered and photographic, radiographic and preserved specimens were collected as indicated by the detailed examination for oral pathology. Many gross specimens were subsequently macerated and prepared as dry specimens. Individual teeth were removed by striking away the lateral alveolar bone using a heavyweight cold chisel and hammer. In this manner details of root anatomy and pathology were recorded.

Radiographic Methods

Isolated jaws i.e. single mandibles or maxillæ were dissected and mounted on plasticine so that exact lateral projections could be taken. Radiographs were made using stationary grids and fast screens for large specimens, and wrapped non screen films for the tooth sections. Satisfactory films were made with exposure factors in the range of 60-65 Kv and 20-30 mAs, using a film focal distance of 100 cm.

RESULTS

A total of 446 horse and pony skulls were examined.

(1) Age, Sex and Type of Horse

An analysis of the ages of skulls examined is shown in Table 4 and Figure 17. It can be seen that similar numbers were examined in each age category with the exception of the youngest and oldest categories. This distribution reflects the throughput of material in the horse abattoirs and knackeries. There is a greater percentage of males seen in all age groups up to 10 years of age. Of the total horses seen over 10 years of age a male/female ratio of 113/95 was seen compared to 97/51 in the ages 3-10 years.

From the skull size three types were categorized, viz:

- a) Pony
- b) Hunter/Cob
- c) Thoroughbred

Table 5 details the types examined and compares the group size with the results of the British Equine Veterinary Association's survey of equine diseases group sizes.

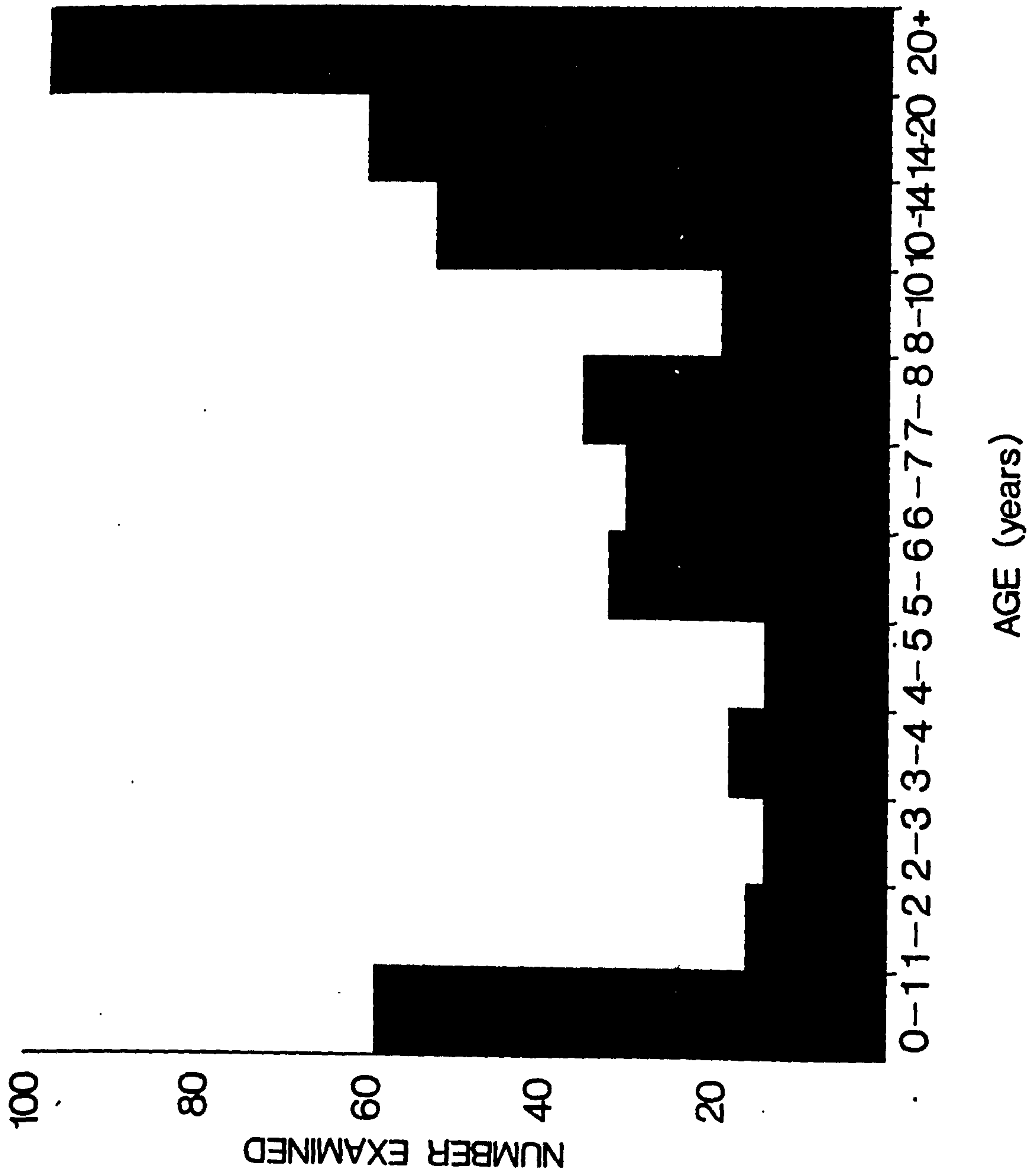
Age in Years	0-1		1-2		2-3		3-4		4-5		5-6		6-7		7-8		8-10		10-14		14-20		20+			
Sex					M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
	59	16	14	14	12	6	10	4	18	14	16	14	26	9	15	4	29	23	33	27	52	45				

Total	59	16	14	14	18	14	14	30	35	19	52	60	97
%	13.2	3.6	3.1	4.0	3.1	7.2	6.7	7.8	4.3	11.7	13.5	21.8	

Total 446

TABLE 4 Dental Disease Survey: Age and Sex of Skulls examined

FIGURE 17: Dental disease survey:
Age and number of skulls examined. Total 446.



	No.	%	BEVA 1962/3 Survey %
Pony	243	54.4	49.2
Hunter/Cob	107	24.0	28.6
Thoroughbred	84	18.8	19.2
Heavy Horse	12	2.8	3.0
Total	446	100.0	100.0

TABLE 5

Dental Disease Survey: Skull Types
examined

(2) Reason for destruction

No significant information was gained although it was inferred that the gross dental pathology observed in some cases (19) would have caused significant dental dysfunction and may well have been the reason for humane destruction.

(3) Dental Disease

a) Wolf Teeth

Of the skulls examined, 58 (13%) were recorded as having a wolf tooth (teeth). All were seen as small, cone-shaped, simple crowns rostral to the first maxillary cheek tooth. In most cases the tooth was unilateral (9 left and 32 right with 17 bilateral). No evidence was seen of erupted mandibular wolf teeth although in three specimens that were subsequently radiographed or macerated a very small tooth could be identified as the vestigial mandibular first permanent premolar.

b) Abnormalities of eruption

In two specimens palatal displacement of the third maxillary cheek teeth was found. Both were estimated as 20 years of age and both had other forms of dental disease (periodontitis and cement necrosis). In each case there was found to be a partial rotation of each third tooth so that in effect there was a shortening of each upper arcade and a corresponding, though reduced, distortion of the contact arcade of each mandible.

Two specimens (6 years and 9 years) were found containing only 5 maxillary cheek teeth i.e. anodontia. Radiographic examination and dissection revealed no evidence of trauma or dentistry. In one (6 years) skull the anodontia was bilateral, whilst in the other the contralateral maxilla contained 6 normal

teeth. When the dental arcades were measured the side with 5 teeth was 12 mm. shorter than the 6 tooth side.

In an aged mare a gross mandibular swelling was radiographed and dissected and shown to be the result of rotation and total impaction of the third cheek tooth (Figure 18). There was gross irregularity of the mandibular arcade and induced wave formation of the maxillary arcade.

Of the 121 skulls examined of horses 5 years and under, 11 were under 6 months of age, as indicated by the non-eruption of the corner deciduous incisors. A detailed analysis of the remaining 110 record cards showed that retention of some deciduous premolar caps was common and that frequently the third was still attached in both jaws at four years of age. Eruption cysts with enlargement of the mandibular rami into symmetrical swellings was universally present. Maxillary swellings associated with eruption were seen more commonly in pony skulls than in bigger horses. Delayed eruption of a wolf tooth occurred in one case (3 years old), and three cases of unilateral delayed eruption of the canine tooth or teeth were found.

Enamel hypoplasia was seen on the labial surface of the deciduous incisors of a yearling (Figure 19).

Examples of dental fusion or of supernumerary teeth (polyodontia, Joest 1926) were not found.

c) Abnormalities of Wear

In the adult horse it is recognised that mastication is essentially a side to side movement of the mandibles accompanied by a degree of caudal pull as food is made into bolus form for swallowing. These movements result in incomplete occlusal contact

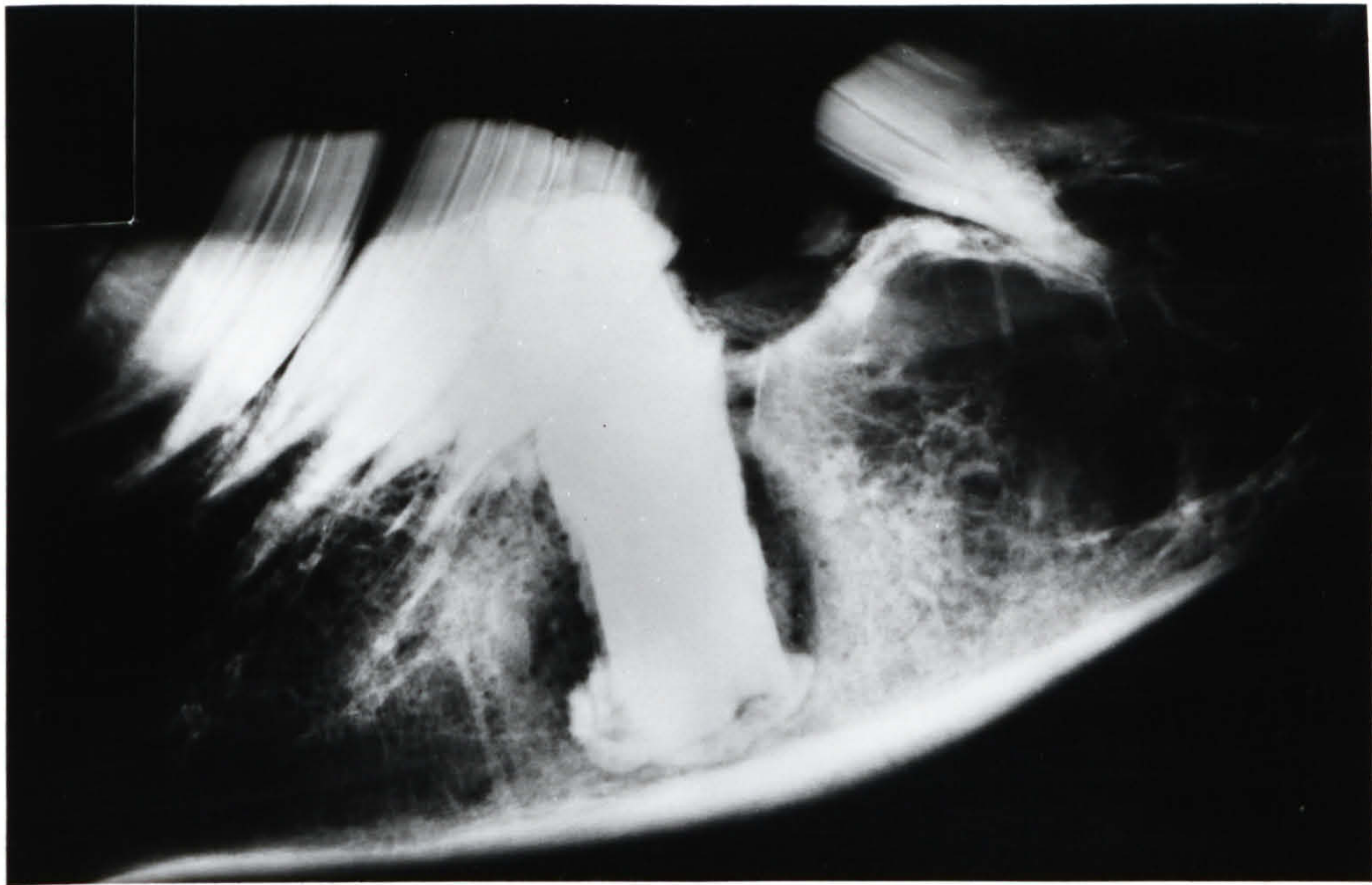


FIGURE 18: Radiograph of mandible with total impaction of third cheek tooth.



FIGURE 19: Enamel hypoplasia of labial surface of deciduous incisors.

and the subsequent development of a raised edge of enamel on the occlusal surfaces - these edges (enamel points) form on the lingual aspect of the mandibular teeth and buccal aspect of the maxillary teeth (Figure 20). The caudal pull results in the formation of small hooks on the rostral points of the first maxillary cheek teeth and caudal margins of the sixth mandibular tooth (Becker 1962). Similarly normal wear of the dental arcade surfaces leads to the formation of ten transverse ridges with corresponding valleys and ridges on the apposing arcade.

Records were made of those specimens with irregular dental attrition outside the norm as described above.

A total of 126 specimens were classified as showing marked abnormalities of wear (28%). A greater incidence was seen in older horses. Of the 97 horses aged more than 20 years, 46 (47%) were found to have grossly irregular arcades (Figure 21). Many were accompanied by periodontal disease and/or dental decay with loss of teeth. In three jaws it was deduced that a tooth had been extracted and the socket had healed. Measurements showed that, as in other animals including man, following dental extractions there had been rostral and caudal movement of adjacent teeth so that the size of the defect in the arcade was reduced.

Twelve adult horses exhibited attrition of the labial surfaces of the incisors indicating chewing or crib biting. In two other horses attrition of the rostral margin of the upper cheek teeth resulted from bit contact. Three examples of mandibular brachygnathia (Parrot mouth) were found with acquired overgrowth of the first cheek tooth as a result of malocclusion (Figure 22).



FIGURE 20: Labial enamel points and rostral hook on maxillary arcade. Six year old Hunter.

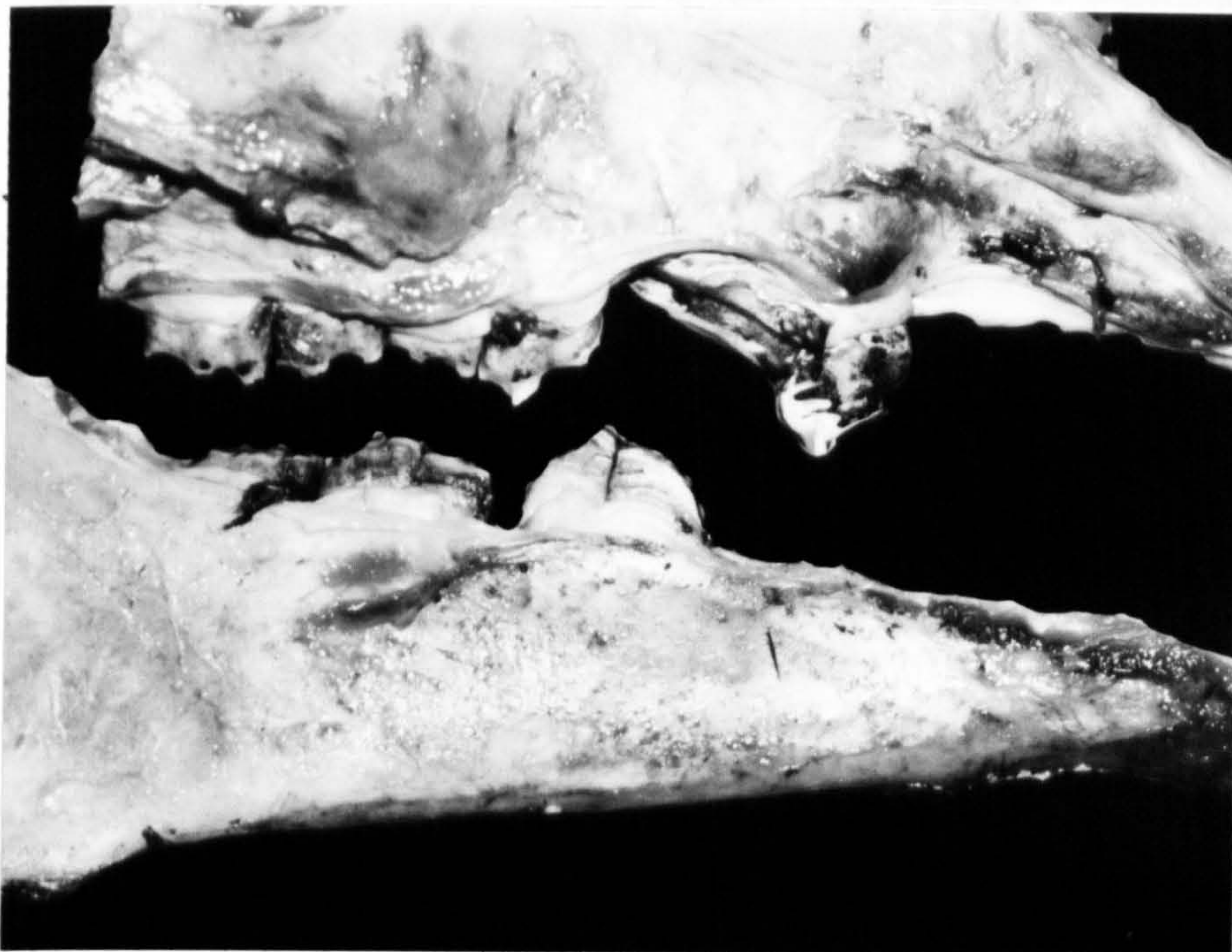


FIGURE 21: Gross irregular wear of dental arcades.



FIGURE 22: Gross mandibular brachygnathism (Parrot Mouth) in a three year old Thoroughbred.

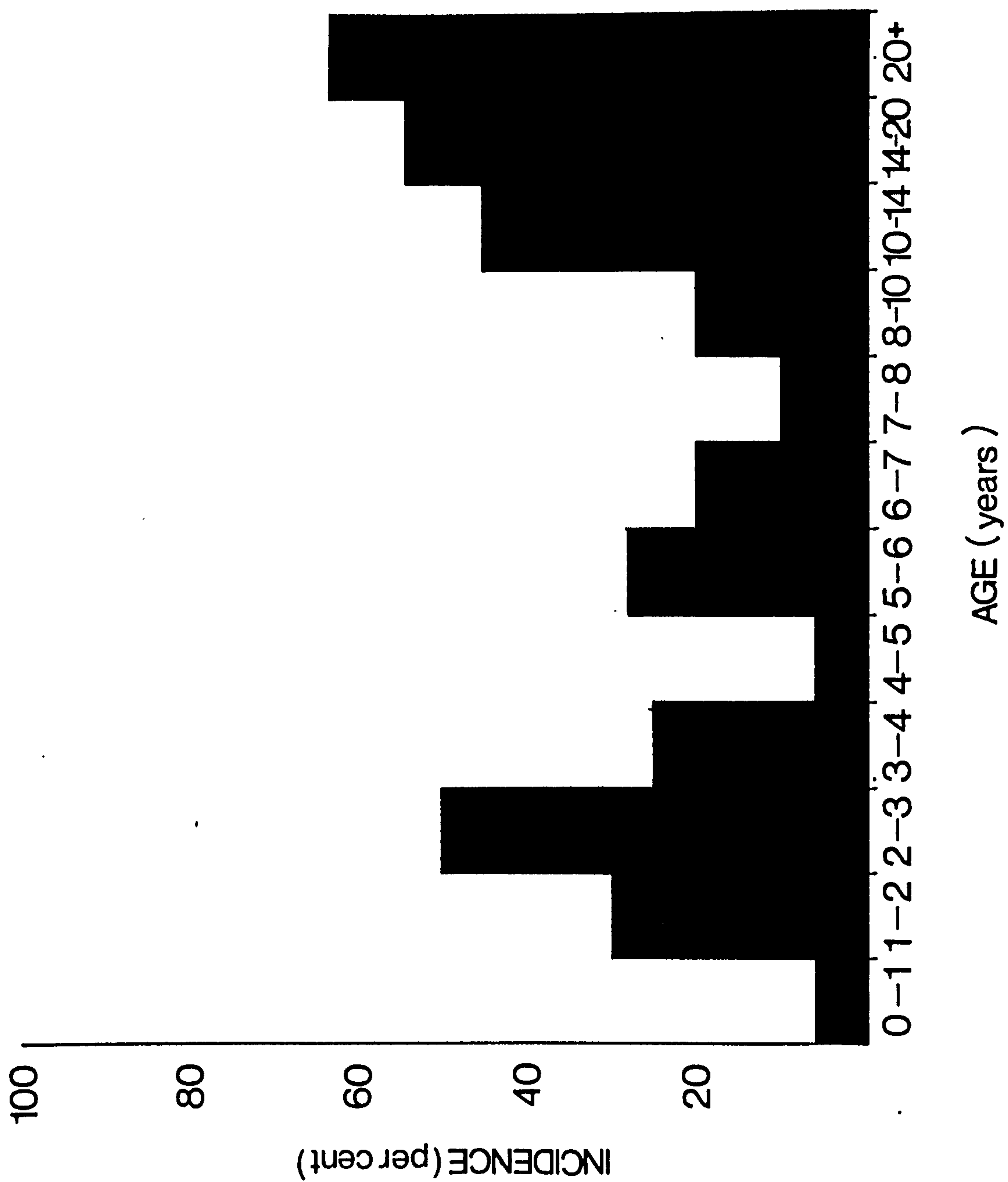
d) Periodontal Disease

Figure 23 depicts the incidence of periodontal disease found in the survey. During the period of eruption of the permanent dentition up to 50% of horses had Grade 2 lesions (erosion of gingival margins) involving the majority of the gum margins. In age groups from 5-10 years the incidence was reduced and then found to increase incrementally with age, so that horses over 20 years of age had an incidence of over 60%. Equally the severity of the lesions increased with age.

Periodontal disease, as seen in the horse, was inflammatory in nature and the initial lesion was found to be a marginal gingivitis with hyperaemia and oedema (Figure 24). When associated with erupting teeth the marginal oedema, erosion and alveolar osteitis extended along the whole arc of the erupting tooth. In established periodontal disease in older horses the gingivitis in the interproximal areas extended to erode the side of the gingival sulcus and a triangular pocket was found (Figure 25). This pocket was impacted with food material and the cycle of irritation, inflammation and erosion had been established leading to destruction of the gum tissue through to the lingual aspect of the tooth and deeper into the periodontal ligament (Figure 26). Ultimately there was gross alveolar infection and the tooth became rotated or lost.

Grade 4 lesions (gross periodontal pocketing and alveolar infection) were found in association with the more severe abnormalities of wear previously described. From measurements of crown overgrowths it was possible to estimate that the disease had been present for at least 3 years (Figure 27 showing 9 mm of crown overgrowth).

FIGURE 23: Dental Disease Survey:
Incidence of periodontal disease.



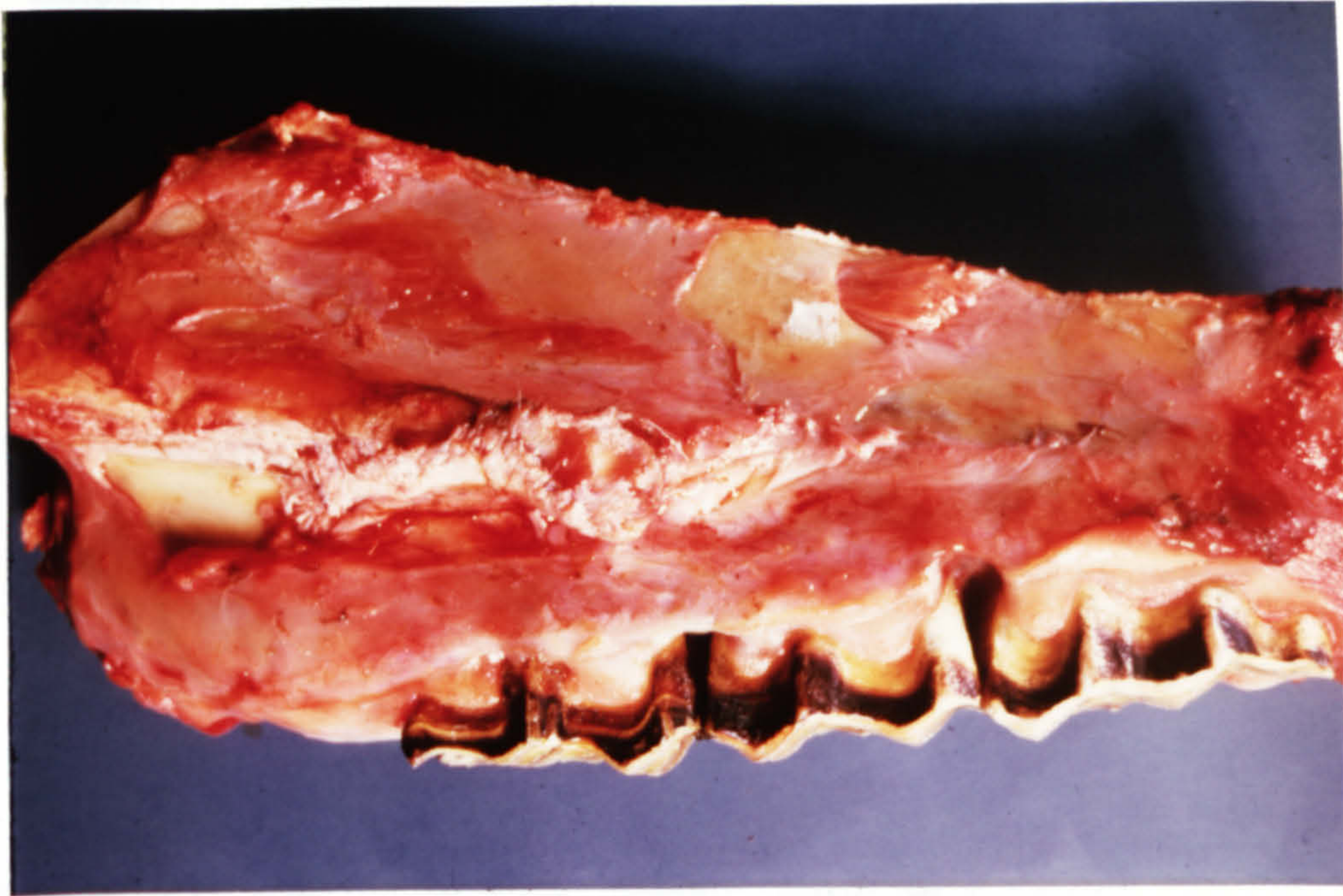


FIGURE 24: Marginal gingivitis between first and second maxillary teeth in a nine month old foal.



FIGURE 25: Severe periodontitis with interproximal pockets filled with food debris. 20 year old mare.

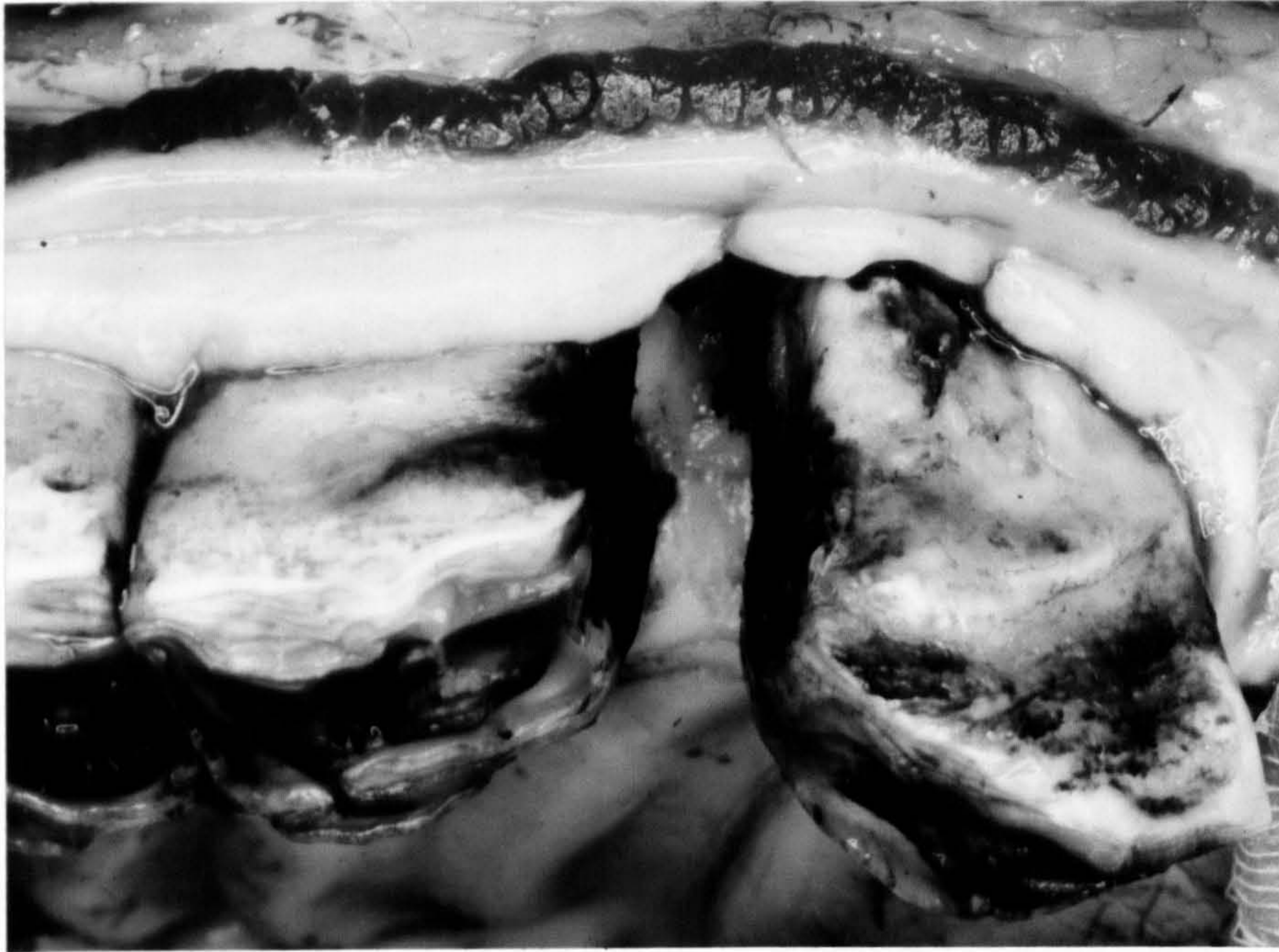


FIGURE 26: Loosening of maxillary tooth as a result of deep periodontal pocketing.



FIGURE 27: Acquired overgrowth of sixth maxillary tooth resulting from loss of sixth mandibular tooth.

Food impaction with local gingivitis was found in association with all lesions that interfered with normal mastication, e.g. broken teeth, wave mouth, unilateral facial and mandibular paralysis. In such cases the disease seen varied from Grade 4 at the locus of the initiating lesion, e.g. tooth fracture, to Grade 2 in adjacent interproximal areas and the apposing arcade.

An analysis of the 157 records of horses aged 14 years and over showed that although 60% had periodontal disease, only 6% were not affected by other dental disease.

Deposition of tartar on the labial aspects of the crowns was only seen on the cheek teeth in the presence of periodontal disease. The canine teeth of older males often carried a thick deposit of tartar, but periodontal infection was not seen around these teeth.

e) Disease of Tooth Substance

Using the grading system as devised by Honma et al. (1962) Figure 28 depicts the incidence of cement necrosis as found in the survey. In the histogram representation of incidence/year of age examined of the whole series it is seen that there is an increase in frequency of occurrence of cement necrosis with age, with over 70% of the specimens examined over 14 years of age showing lesions. It is possible that the 20% figure seen in the 8-10 years group is not wholly accurate in that 60% was seen in both the preceding and succeeding age groups, and the low figure may reflect the fact that only 19 skulls constituted this group (35 in preceding and 52 in succeeding group).

Figure 29 represents a detailed analysis of the incidence and severity of cement necrosis within individual teeth of the maxillary arcades. The fourth cheek tooth (M1) was the tooth most frequently

FIGURE 28: Dental Disease Survey:
Incidence of caries of cementum
(Cement Necrosis, Baker 1970).

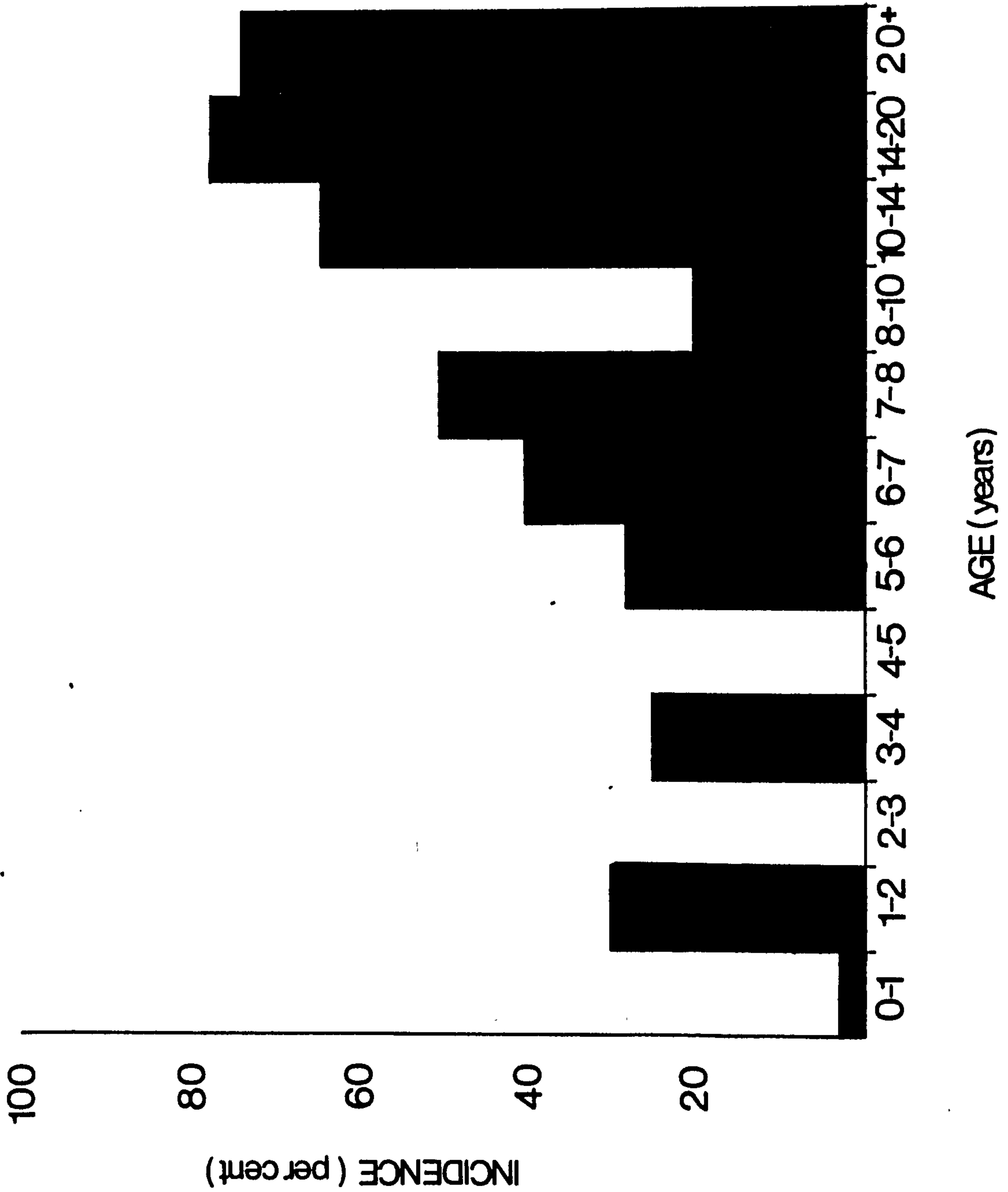
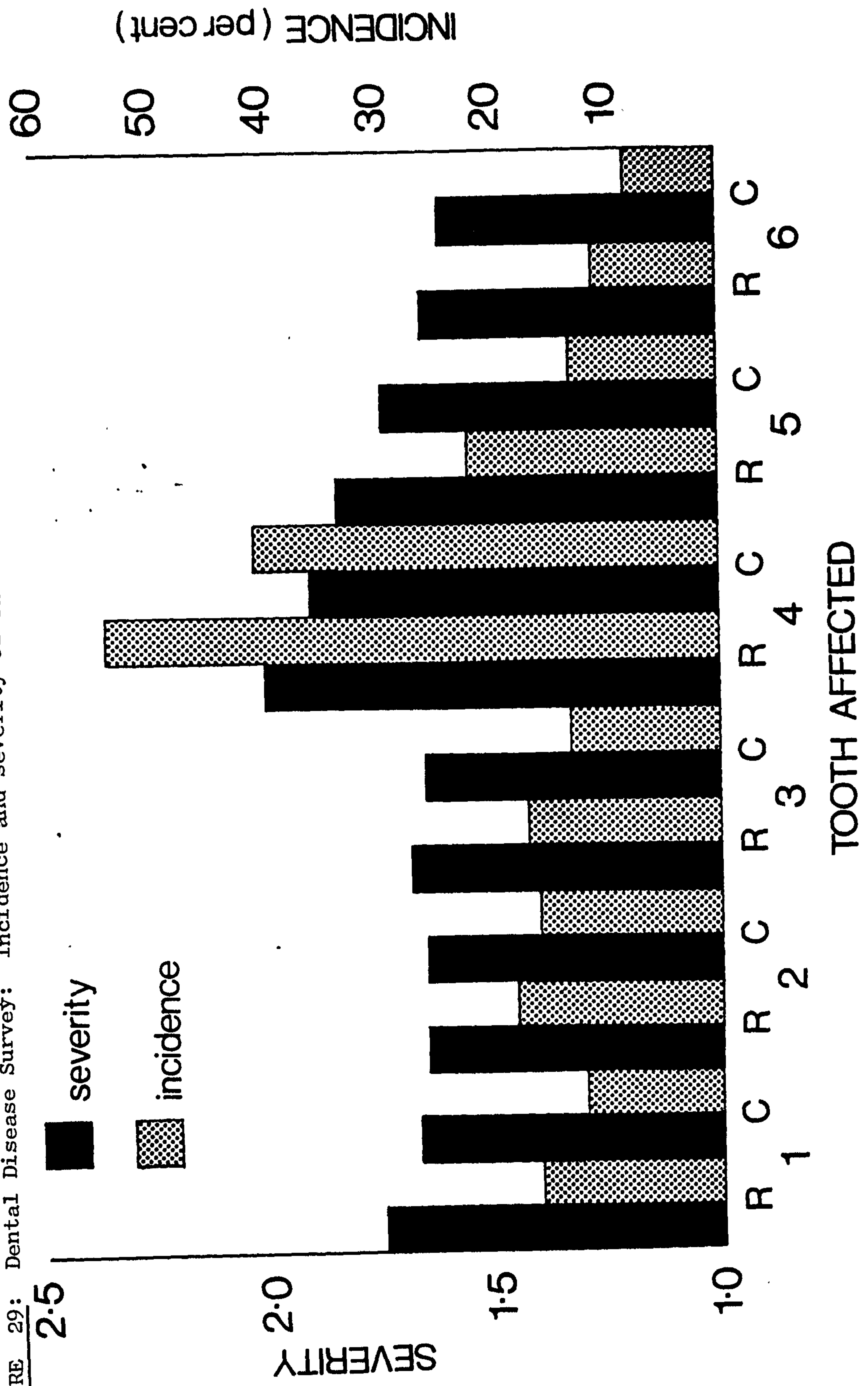


FIGURE 29: Dental Disease Survey: Incidence and severity of caries. R = rostral and C = caudal cement lake.



affected by cement necrosis and was also found to be more severely affected than other teeth. An overall analysis of the incidence showed two distinct types of arcades. The first in which nearly all the teeth were affected to some degree, and the second in which only a single tooth (commonly the fourth cheek tooth) was affected.

In many cases the lesions were confined to the cement lakes (Grade 1), and destruction of enamel (Grade 2) and dentine (Grade 3) were less commonly seen (Figure 30). The area of cement necrosis was centred on the infundibulum and expanded centrifugally within the enamel invaginations. The pockets thus formed were constantly packed with food debris. In advanced cases this expansion resulted in the coalescence of the area of the rostral and caudal cement lake necrosis so that a deep central valley was formed (Figure 31). Five cases were found with fracture of the fourth cheek tooth along such a necrotic valley and loss of portions of the tooth.

In old animals in which the lesion remained confined to the cement lake as attrition of the crown occurred and the enamel invagination was lost by wear, so the area of cement necrosis was also worn away, leaving an occlusal surface of polished dentine (Figure 32).

Ten randomly selected cheek teeth from each location that appeared to have normal cement lakes as viewed from the occlusal surface were selected. Using the diamond disc cutter 3 mm. transverse sections were prepared. In each specimen the depth of the enamel invagination was examined for evidence of cement necrosis (cement hypoplasia). It was found that of the 60 teeth examined 43% of the cement lakes examined were so affected (Figures 33a and 33b).

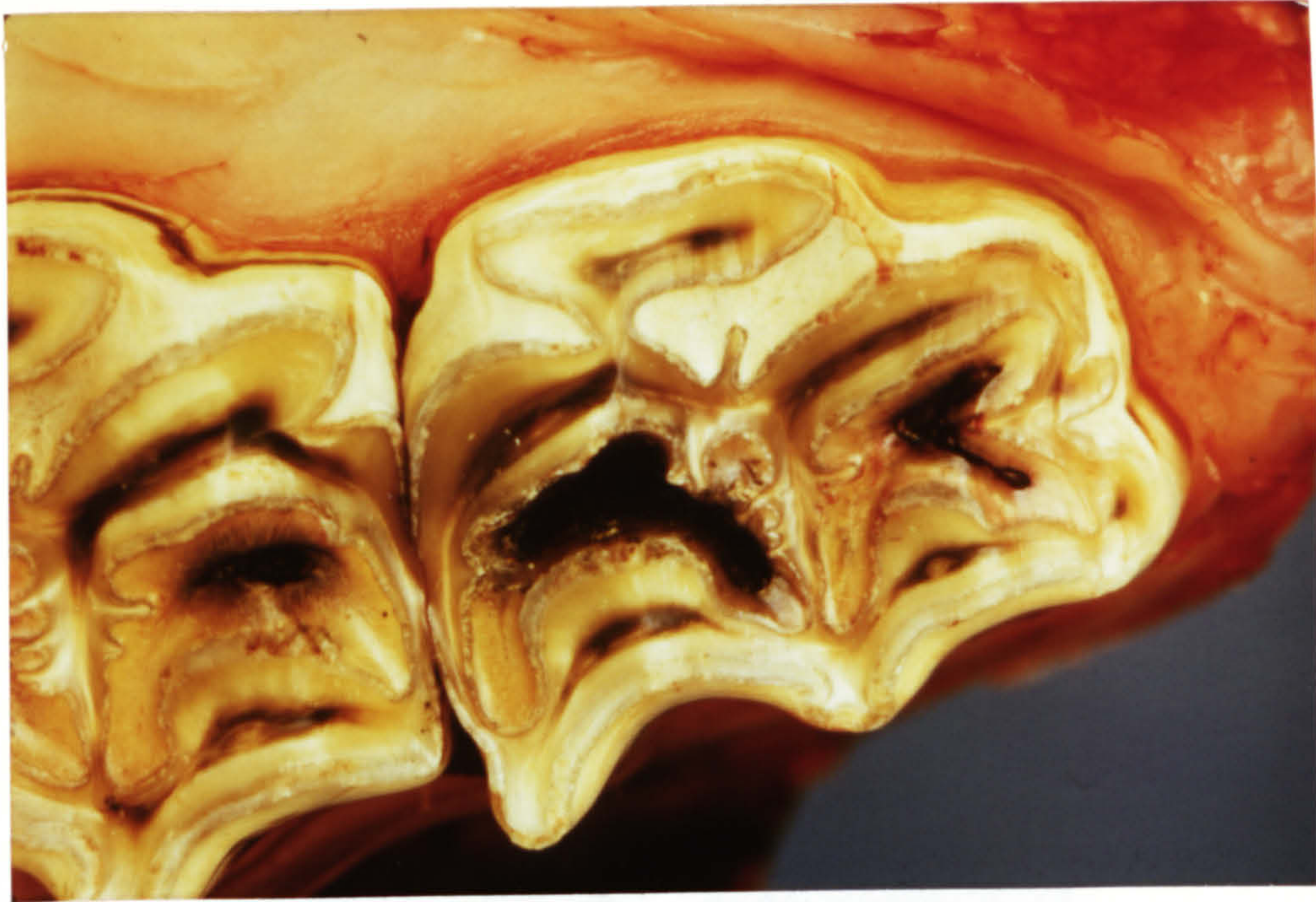


FIGURE 30a: Occlusal surface of first maxillary cheek tooth of a six year old horse with Grade 1 lesion of rostral and Grade 2 lesion of caudal cement lake.

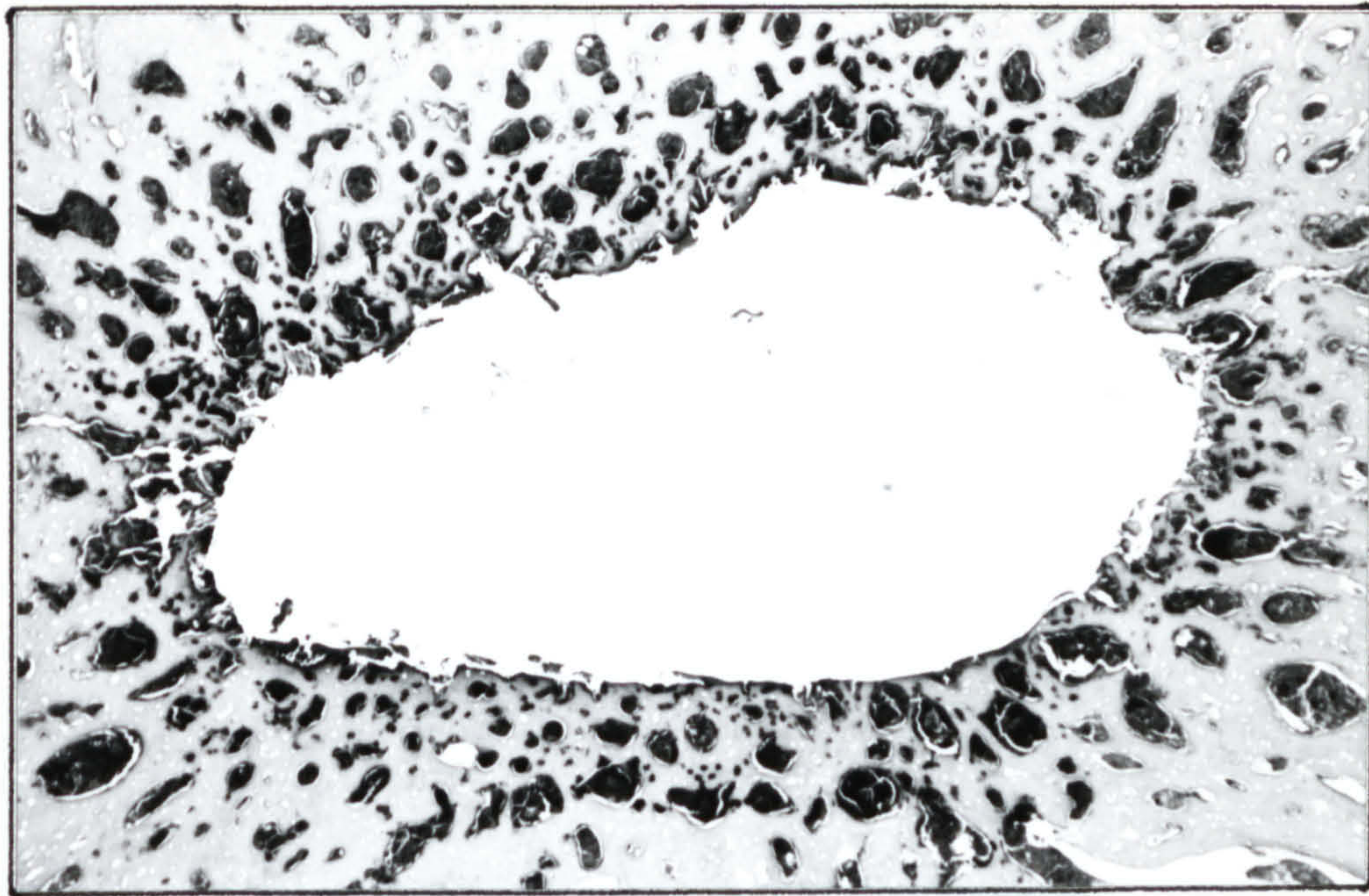


FIGURE 30b: Centrifugally expanding caries of cementum. Decalcified H and E x 14.



FIGURE 31: Coalescence of Grade 3 lesions to create central valley of necrosis.



FIGURE 32: Attrition of cement lakes and exposure of dentine. 20 year old mare.



FIGURE 33a: Transverse section of third cheek tooth one cm from occlusal surface.



FIGURE 33b: Transverse section 5 cm from occlusal surface with food debris and hypoplastic cementum of enamel invagination.

(4) Histology

In transverse decalcified and ground sections of teeth with diseased cement lakes the earliest changes appeared to be an enlargement of the central infundibulum with marked irregularity of its edges. The canaliculi were filled with organisms and food debris (Figures 34 and 35), and there appeared to be destruction of the cementum. Similar destruction was seen of both enamel and dentine in Grade 2 and Grade 3 lesions.

DISCUSSION

Only 13% of the specimens examined had erupted wolf teeth. No evidence was seen of healed sockets at this site in those without wolf teeth, indicating previous surgical extractions of these teeth. The standard reference texts indicate that there is some doubt as to which set of teeth the wolf tooth belongs. Sissons (1910), as in Chapter I, includes it as the vestigial permanent first premolar, but does not indicate how frequently it is found. The 13% frequency found in this survey is less than the occurrence rate observed by clinicians (B.E.V.A. personal communications).

Colyer (1931), after examining nearly 500 skulls of horses which had been worked in the London area, found that "approximately one-third presented with some degree of paradontal disease". This survey illustrates that the true incidence is likely to exceed this rate and is clearly age dependent. Colyer was of the opinion that the disease was initiated by injury to the gingivum caused by food. He concluded by stating that any departure from a "natural diet and conditions" would lead to periodontal disease. He had stabling and

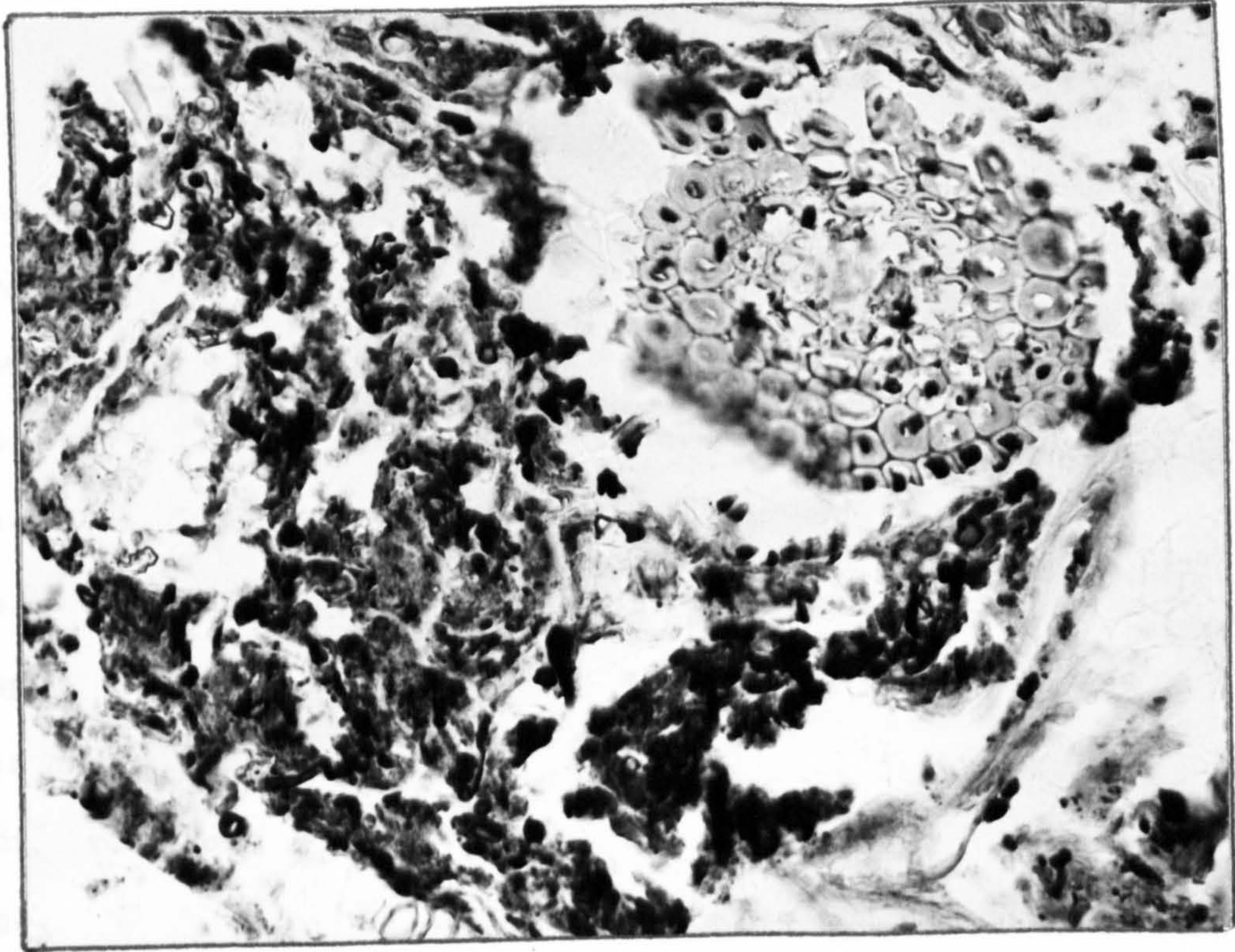


FIGURE 34: Food debris extending from infundibulum into cementum. Decalcified H and E x 350.

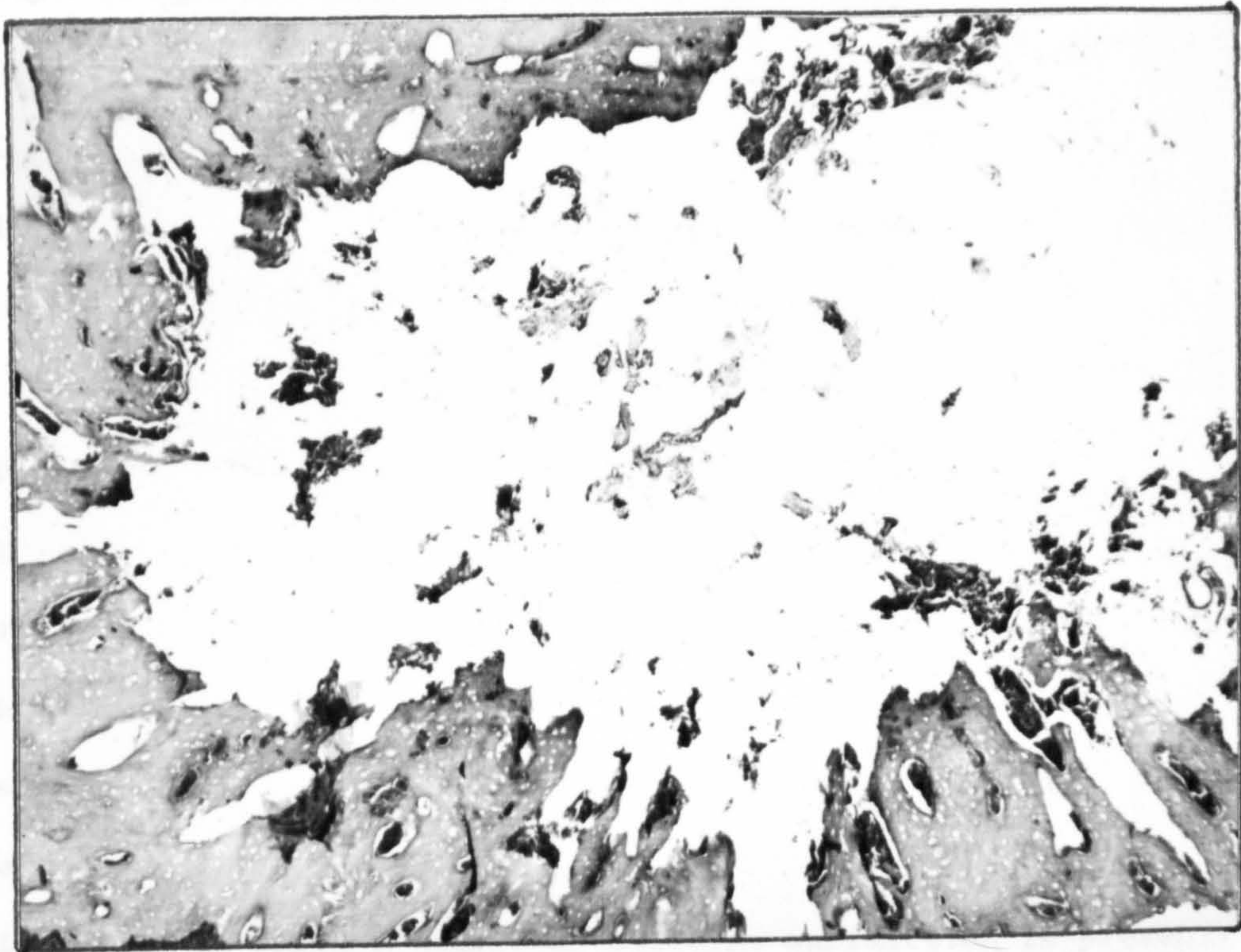


FIGURE 35: Destruction of cementum. Decalcified H and E x 55.

chaff feeding in mind. In this survey the trauma of tooth eruption was clearly implicated in the pathogenesis of periodontitis and alveolar osteitis in younger horses. Stagnation and impaction with food debris of areas of established periodontal disease was commonly seen in older specimens. Such impacted areas were more severe in association with conditions of gross malocclusion. Under such circumstances it is possible to speculate that the initiating factor in the cycle of irritation, inflammation and infection that is the basis of periodontal disease is a failure of the normal mechanisms of cleansing of the gingival crevice. Only 11% of all specimens examined with periodontal disease did not have other intercurrent forms of dental disease. This figure is reduced to 6% if the tooth eruption ages are excluded. Such evidence would support this theory. The role of the cleansing function of saliva was discussed by Voss (1937) and it was his opinion that interference with normal salivary flow was inevitably followed by periodontal infection.

It has been noted by King (1947) using ferrets, and Brown and Park (1968) using dogs, that the frictional forces associated with chewing hard materials (bones) were effective in keeping the teeth free of tartar and the gums healthy. It is noted in this survey that dental scale (tartar) is not frequently seen on the cheek teeth of the horse. It was seen on the surfaces of the canine teeth and in the presence of dental disease, i.e. under circumstances where a degree of occlusal malfunction is present. Leue (1941) showed the abnormalities in grinding patterns that occurred in the presence of polyodontia, fractured teeth and other

irregularities of the dental arcade. Colyer (1931) cites H.K. Box as defining physiological occlusion as "a condition in which the systems of forces acting upon the tooth during occlusions are in a state of equilibrium and do not and cannot change the normal relationship existing between the tooth and its supporting structures". Box also defined traumatic occlusion as "being a relationship in which there is overstress on all or part of the pericementum" (periodontal ligament).

From this survey it would appear that in older horses the aetiological agent in the pathogenesis of periodontal disease is traumatic occlusion.

In the literature review it was seen that the terms caries, caries of cement, dental necrosis of herbivores, alveolar periostitis, peridentitis, pericementitis and pulpitis have all been used to describe the lesions found in the cementum of the enamel lakes of the maxillary cheek teeth. Skewes (1962) expressed doubt that dental caries exists in the horse. Baker (1970) suggested that until further studies had been made that the term infundibular necrosis might be more appropriate. Evidence has been presented which suggests that the lesions seen may be of two types. In most instances the lesions are confined to the enamel invaginations and do not actively expand. It is suggested that such lesions are the direct result of the developmental fault of cement hypoplasia (Chapter II), and they appear as the normal crown is worn away, hence the greater frequency seen in the fourth cheek tooth and in older horses. With review the use of the terms infundibular or cement necrosis would not seem to be applicable. In adult horses the cementum in the enamel invaginations is not a

living structure, and therefore to describe this as undergoing necrosis (death) is inappropriate. This type of lesion should therefore be described as hypoplasia of cementum or disintegration of cementum.

Some specimens however clearly showed an active inflammatory process expanding the lesion through the enamel into dentine and pulp. Many authors (Merillat, 1921; Colyer, 1931; Hofmeyr, 1960) have stated that food material becomes packed into the "open infundibular canals" and subsequent fermentation leads to the dissolution of cementum and the surrounding enamel and dentine by acid substances. Under such circumstances the term caries or caries of cementum could be applied to all lesions seen above Grade I in the Honma system of classification.

CONCLUSIONS

Periodontal disease is inflammatory in nature in the horse and is commonly seen during the eruption of teeth and under circumstances of traumatic occlusion.

Hypoplasia of cementum is found in over 60% of horses' teeth and it is suggested that it is from such areas of hypoplasia that true dental caries develops.

CHAPTER IV

ORAL BACTERIOLOGY AND pH STUDIES

INTRODUCTION

For over 100 years oral bacterial flora studies have been the keystone of much of the dental and periodontal research in man. The basic studies of Miller (1889), with in vitro incubation of tooth slices and saliva, demonstrated that cariogenic acid was produced by bacterial fermentation and subsequently the investigation of Lactobacillus acidophilus, Streptococcus salivarius, Streptococcus mutans and the Strep. viridans group has been well documented (Green and Dodd, 1956). In man, the bacteriological examination of unstimulated saliva reveals the presence of a wide range of streptococci, Neisseria spp., Fusobacterium spp., Leptotrichia spp., salt-tolerant micrococci, Candida spp., coliform organisms, Veillonella spp. and lactobacilli. Numerous workers have shown statistically significant changes in the flora depending upon sampling and culturing techniques, diet and the caries and dental disease status of the subject (Richardson and Jones, 1958).

The pH of saliva has been the subject of intensive investigation in man. It appears to vary within a narrow range and most of the studies dealing with the relationship between salivary pH and dental caries have shown no positive correlations.

Studies on the rate of salivary secretion, its antibacterial and buffering properties have given widely varying results and McDonald (1950) reviewed the problems of these investigations.

The role of the oral flora and saliva is a relatively unexplored field as far as the natural history of spontaneous dental diseases in animals is concerned, although much use has been made of gnotobiotic animals in studies of the epidemiology and pathogenesis of dental diseases. Voss (1937) expressed the opinion that periodontal disease in the horse resulted from impaired salivary flow, implying that under normal conditions salivary washes maintain the integrity of the gums and periodontal structures. Both Merillat (1921) and Hofmeyr (1960) stated their opinions that necrosis of the maxillary cheek teeth was brought about by the bacterial fermentation of food material within the infundibulum of the central lakes of cementum.

This chapter describes observations made on both the bacterial flora of the horse's mouth and of the pH of saliva in the anaesthetised horse.

MATERIALS AND METHODS

Disclosing Techniques

In the washed mouth, observations were made of the location, quality and quantity of dental plaque and calculus after the application of a specific disclosing agent (Displac)* or vegetable food dyes.** The quantity of plaque and calculus were scaled on a zero to three grading (Figure 36). Observations were

* Displac, Pacemaker Corps, Oregon, U.S.A.

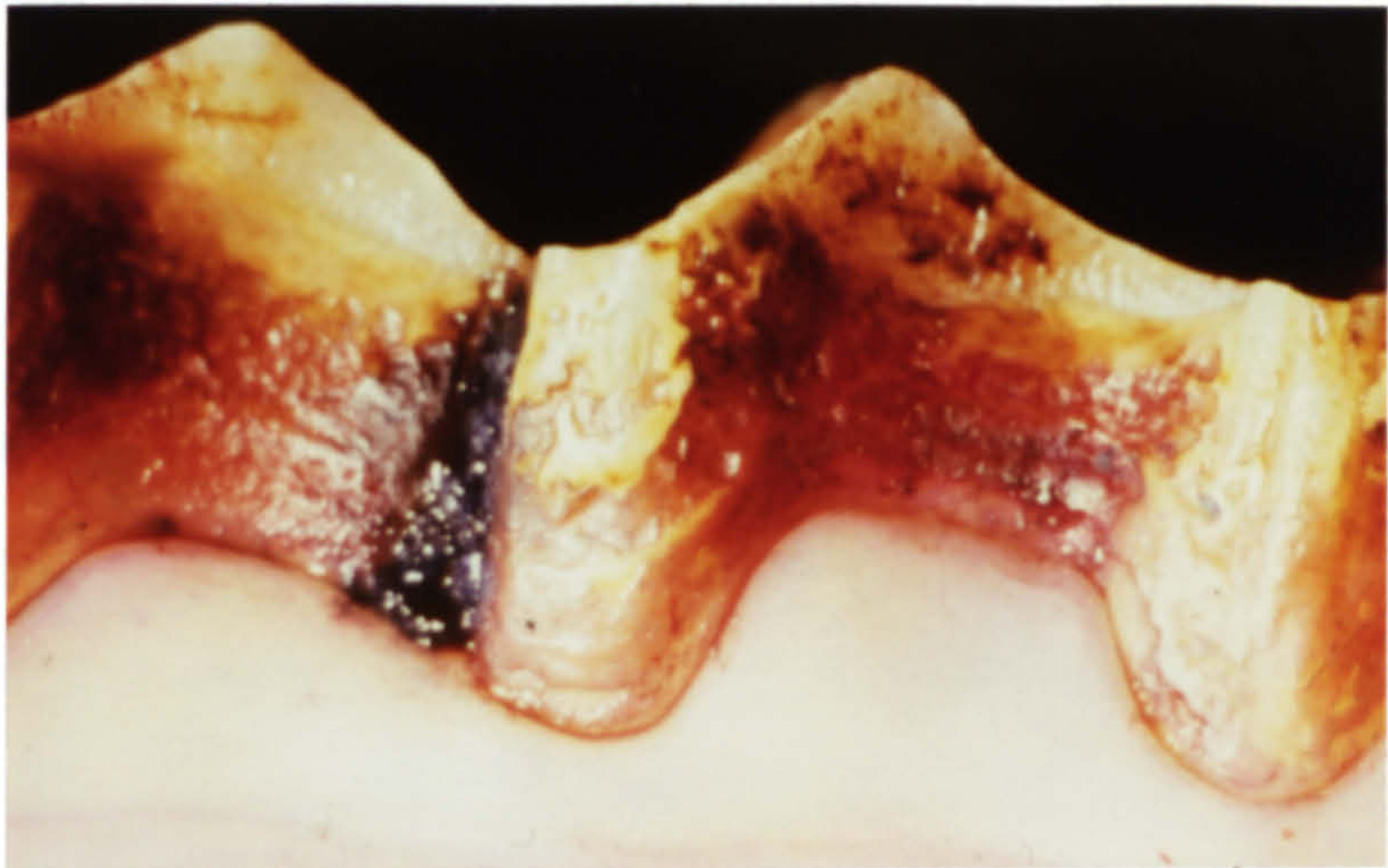
** E142 Greens and E124 Ponceau 4R, Clayton & Jowett, Blue Seal Colouring, Liverpool.



A



B



C

FIGURE 36:

- Disclosure of dental plaque**
- A. Pre-disclosure.
 - B. Grade 1 plaque on incisor and Grade 2 on canine.
 - C. Grade 3 plaque adjacent to periodontal pocket.

made in horses free from dental disease, horses with clinical dental disease and in those skulls in the Dental Disease Survey section (Chapter III) that were obtained in a fresh condition.

Sampling and Culture Techniques

Preliminary samples showed that bacterial colony counting and identification was rendered difficult by overgrowth of the culture plates by aerobic, spore-bearing organisms and fungi. These organisms originated from food-stuffs such as hay, corn, nuts and straw. Consequently the following technique was developed to overcome this problem.

The subjects were stabled on peat moss rather than straw and were muzzled for at least 12 hours (usually 17 hours) to prevent eating and drinking (Figure 37). Two groups of animals were used. The first were selected individuals undergoing general anaesthesia in the University of Glasgow Veterinary Hospital that proved to be free of dental disease on oral examination (15 animals). These served as the control group and were selected to match the dental disease group as closely as possible, e.g. management regimes, whether at grass or stabled, and were also age and sex-matched (15 animals).

Under anaesthesia the gingival margin, crevice and tooth surface on the buccal aspect of the first upper cheek tooth (PM2) was irrigated with a 20 ml spray of sterile water from a syringe and 19 gauge needle (Figure 38). Using a sterilised dental probe with a beaked margin the gingival crevice and tooth surface were scraped along a two cm length (Figure 39).

The scraped material was suspended immediately in 10 ml of 0.1% peptone water at a pH of 7.2. After mixing, serial dilutions



FIGURE 37: Design of muzzle to prevent oral contamination with ingesta.



FIGURE 38: Irrigation of gingival crevice with sterile water.

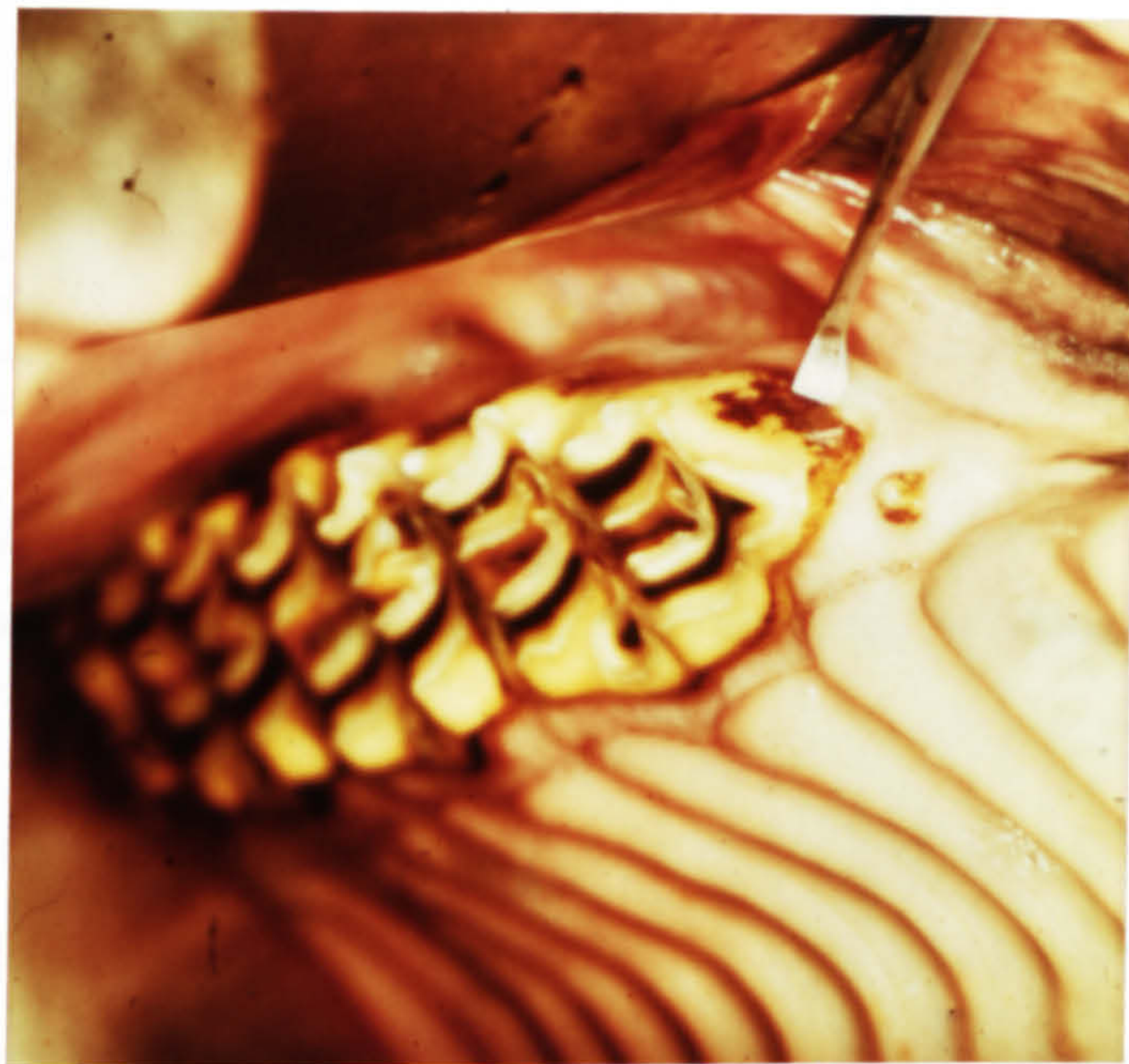


FIGURE 39: Collection of plaque sample.

were made into 9 ml portions of peptone water within two hours. In this manner three cultures of 10^{-2} , 10^{-3} and 10^{-4} dilutions were prepared. One ml of the selected log dilution was mixed with 20 ml of liquid medium* at 52°C and immediately poured into standard 90 mm plastic petri dishes. The pour plates were used for the enumeration of total aerobes, anaerobes, Strep. salivarius, Veillonella spp., lactobacilli and H_2S producing organisms. Spread plates of 0.1 ml diluted material were used for the enumeration of total streptococci, starch hydrolyzers, Neisseria spp., Fusobacteria spp., Leptotrichia spp., micrococci, coliforms and Candida spp. Care was taken to ensure sterility by autoclaving the media just prior to use.

All cultures were made both aerobically and anaerobically at 37°C for four days. Anaerobiosis was achieved using jars that were evacuated then filled with carbon dioxide and hydrogen. Tomato juice agar** was used for the culture of lactobacilli.

The results were expressed as numbers of colonies counted on each plate using an illuminated colony counter*** (Figure 40).

After subculture of selected colonies, bacterial identification was based upon aerobic/anaerobic culture, colony shape and size, glucose utilisation, catalase and oxidase tests, gram staining and the form and motility of the organisms (Cowan, 1974).

* Oxoid blood agar base No. 1 containing 5% sheep blood. Oxoid Ltd., Basingstoke.

** B.B.L., Becton, Dickinson & Co., Wembley.

*** Colony counter, Gallenkamp, London.



FIGURE 40: Colony counting.

Cultures were also prepared from the material found within the depths of the infundibular canal of sectioned maxillary cheek teeth.

pH Measurement

In the anaesthetised horse the pH of the gingival crevice and saliva were measured using an antimony/KCl microelectrode (Figure 41). The saliva was aspirated with a 5 ml syringe placed in a 5 cm glass saucer dish and CO₂ evaporation was prevented by a film of oil. A standard pH meter was used and calibrated as per manufacturers instructions.* Measurements were made at 15 minute intervals.

Freshly extracted maxillary teeth with normal occlusal surfaces and diseased teeth (caries of cementum) were sectioned in a vertical plane through the cement lake. pH measurements were then made within the depths of the enamel invagination (see Figure 13).

RESULTS

(1) Dental Plaque and Calculus

In man Displac stains bacterial deposits dark blue and superficial protein precipitates are stained pink. When used in the horse this distinction between "old" and "new" plaque was not seen and Displac proved to have no advantage over food colouring dyes (green or scarlet).

Plaque was found on all surfaces of normal teeth with greater accumulations on the canine teeth when these were present, on the lingual aspect of the incisor teeth and at the interproximal areas of the cheek teeth at the gum margins. In all teeth the

* pH Meter 2.5, Radiometer, Copenhagen.



FIGURE 41: Antimony/KCl micro-electrode for gingival crevice and salivary pH measurements.

depth of deposit decreased from the gum level to the crown. No marked variations were found along the length of the dental arcade and in particular the amount of plaque deposit was not related to the position of the parotid salivary outflow papillae at the level of the third upper cheek teeth.

In cases of periodontal disease the interproximal areas were commonly plugged with foodstuffs, even after 17 hours muzzling. In these areas the disclosing solutions demonstrated soft plaque over the whole of the buccal surface of the tooth.

With the exception of the canine teeth in males the teeth of the horse were generally found to be free from both supra- and sub-gingival calculus deposits. In all cases the canine teeth carried deposits of firm yellowish-brown calculus that increased in volume with age. The mandibular canine teeth had more calculus than the upper canine teeth.

In cases of periodontal disease noticeable deposition of calculus was found in 15 per cent of cases (39 horses or skulls out of 260 examined). The calculus was made of calcium carbonate contaminated with food and pigment deposits.

(2) Oral Bacteriology

Colony counts have been grouped into three categories according to their incidence (high, intermediate and low numbers) and the results shown in Table 6. The gram positive cocci, mainly streptococci, micrococci and starch hydrolyzers were the most frequently found organisms and up to 100 colonies were found in dental plaque diluted as described in Materials and Methods, i.e. a concentration of 10^{-5} organisms per gram net weight. Although as careful a technique as possible was used when sampling

HIGH COUNT	Streptococci
(number of colonies	Micrococci
at 10^{-4} dilution)	Starch Hydrolyzers
INTERMEDIATE COUNT	Anaerobes
(number of colonies	<u>Veillonella</u> spp.
at 10^{-3} dilution)	H ₂ S producers
LOW COUNT	<u>Lactobacillus</u> spp.
(number of colonies	<u>Fusobacteria</u> spp.
at 10^{-2} dilution)	Coliforms

TABLE 6

Oral Bacteriology: Colony Counts
of Bacteria Present in Equine
Dental Plaque

horses with dental disease many unsatisfactory cultures were made. As has been described the gingival margins were frequently contaminated with food material in cases of periodontal disease and such contamination resulted in overgrowth of the cultures by aerobic, spore-bearing organisms and fungi. Consequently the results obtained could not be subjected to statistical analysis. The predominant organisms in isolates from infected gums were streptococci with the appearance of coliforms and Fusobacteria at 10^{-3} dilutions. Spirochaetes and campylobacters were also seen on direct smears.

As with the cultures from diseased gums the results obtained from culturing suspended material from cases of caries of cementum (hypoplasia of cementum or disintegration of cementum, Chapter III) were unsatisfactory, there being either only a few streptococcal and micrococcal colonies or contaminated plates (aerobic, spore-bearing organisms and fungi). Cultures from dental fistulae produced mixed growths of streptococci, staphylococci, coliforms and actinomycetes.

(3) pH Measurements

pH measurements were made over periods ranging from one hour to 13 hours, the majority being taken over a period of two hours. A total of 18 horses were examined and observations of the gingival crevice pH showed an alkaline reading (7.4 mean, range 7.1-7.7). After two hours anaesthesia the pH fell to a mean of 7.3 (range 6.9-7.6).

The pH of salivary aspirates roughly paralleled these readings (7.54 mean, range 7.3-7.9). In some cases it was not possible to aspirate samples at 15 minute intervals throughout

the two hour observation period but no decrease in pH was seen in those animals in which eight readings were made.

The antimony probe of the electrode was just fine enough to be inserted into the softer tissues of the hypoplastic carious cementum of the sectioned maxillary cheek teeth. The results were as follows:-

	<u>pH Mean</u>	<u>Range</u>
Normal teeth (10)	6.9	6.6-7.2
Caries of cementum (10)	7.1	6.8-7.4

DISCUSSION AND CONCLUSIONS

In man it has been recognised for many years that dental plaque (microbial or bacterial plaque) is a structure of major significance as a contributing factor to the initiation of dental caries. In the horse occlusal caries is very rarely seen. It has, however, been suggested that the caries of cementum commonly seen in the maxillary cheek teeth (see Chapter III) arises as a result of fermentation of food material within the infundibulum (Merillat, 1921; Colyer, 1931; Hofmeyr, 1960), and the formation of cariogenic (cementolytic) acid. Baker (1970), after studies on cementogenesis in the maxillary cheek teeth, felt that if such lysis occurred it was secondary to the developmental defect of hypoplasia of cementum.

It was hoped to show in these studies the precise role of bacteria in the pathogenesis of caries of cementum of the maxillary cheek teeth. Although acid was found within the depths of the cement lake of some individual teeth, there was no clear correlation between the degree of caries of cementum present and either the acidity of the environment or the number of cariogenic

bacteria isolated (streptococci). This was probably the result of the technique used in that as the teeth were sectioned the surface was washed with a water spray to prevent overheating of the diamond disc and the specimens. In man it has been possible to investigate pH changes across the depth of the dental plaque and to study its permeability to sugar solutions (Stephan, 1940; Stralfors, 1948). Such techniques are not practicable in the horse. Similarly, indwelling dental electrodes have been used in man but cannot be used in the horse.

It is interesting to note that calculus and plaque deposits were maximal in association with poor occlusal contact and in the canine teeth where no occlusal contact is made. Such observations support the inference that the normal masticatory forces are instrumental in cleaning the teeth. It suggests that the application of strain gauges to the lateral surfaces of the teeth might yield valuable information on the scale of the shearing forces present on the horse's tooth during mastication.

Very large volumes of saliva are produced by herbivorous animals when compared to the scale of salivary flow in carnivores or omnivores. Afonsky (1961) produced an extensive review of the literature dealing with saliva in man covering such aspects as its composition, pH, quantity, viscosity, antibacterial properties and its buffering capacity. It has been estimated that the horse produces up to 40 litres of saliva in a 24 hour period and that 40% is produced by the parotid salivary glands (Phillipson, 1977). No figures are available for the changes in either the quality or quantity of saliva associated with dental disease in the horse, although it is recognised that there is an increase in quantity

of saliva held in the mouth (as distinct from an increase in total volume) associated with the eruption of teeth, periodontal disease and broken and grossly decayed teeth. Voss (1937) was of the opinion that salivary washings were essential for the maintenance of healthy gums and periodontium. Other workers (King, 1947; Brown and Park, 1969; Baker, 1970) feel that it is not possible to separate the effects of salivary flow from the frictional forces of mastication.

Specific bone infections associated with dental disease and actinomycete or nocardial infections have been described in the horse (Tritschler and Romack, 1965). No cases were found in this series from either the specimens examined in the dental disease survey or in the cases of clinical dental disease (Chapters III and V), although actinomycetes were seen in direct smears of swabs taken from some mandibular fistulae.

CHAPTER V

CLINICAL ASPECTS OF DENTAL DISEASE

INTRODUCTION

Comment has already been made that in the British Equine Veterinary Association's survey of diagnoses made after requests for veterinary attention in the year 1962-63, attention to teeth accounted for nearly 10% (1,608 out of 17,268) of the consultations. It is clear therefore that dental diseases and dental prophylaxis form an important part of equine clinical practice. It has also been recognised that some dental malformations are inherited and parrot mouth is listed as a condition for which a stallion can be refused a licence in the United Kingdom.

MATERIALS AND METHODS

Detailed case records have been made of all cases of dental disease in horses referred to the author since 1965. In the period 1965-69, 68 horses and ponies with dental disease were examined in the Department of Surgery, Royal Veterinary College Field Station, North Mymms, and from 1969-1977 a further 163 cases have been treated in the Department of Surgery, University of Glasgow Veterinary Hospital.

The history of each horse was obtained from the owner and a complete physical examination, including the mouth, teeth and lips, was made. In some fractious animals the oral examination could only

be completed with the aid of ataractic sedation, narcosis or general anaesthesia. In those cases that were anaesthetised, the visual findings were complemented by endoscopic examination of the dental arcades using a rigid endoscope* and by radiography.

All extracted teeth were measured and details of dental disease recorded. The teeth were sectioned in both the transverse and saggital planes and prepared for histological examination as described in Chapter I.

Radiographic Techniques

The majority of radiographic examinations were carried out under general anaesthesia with the jaws held apart by a gag between the incisor teeth. In those horses in which the dental disease was associated with maxillary sinus empyema erect lateral skull films were taken in the tranquillised, conscious horse to demonstrate the presence of sinus exudate.

In order to produce radiographic films giving detail of changes in the structure of the reserve crown, the roots and the alveolar bone, the problems of superimposition were overcome by employing a 30-40° oblique beam, which projected the image of the normal arcade away from the diseased area (Figure 42). Better results were obtained when the arcade under investigation was adjacent to the film or cassette. The tube was angled from the dorsal side for the maxillary arcade and from the ventral aspect for the mandibular arcade. It was also found to be of value to have a similar projection of the normal arcade for comparison. The sites of mandibular or maxillary dental fistulae were demonstrated by the insertion of metal probes.

* Rhinolaryngoscope, Hauptner, Berlin.

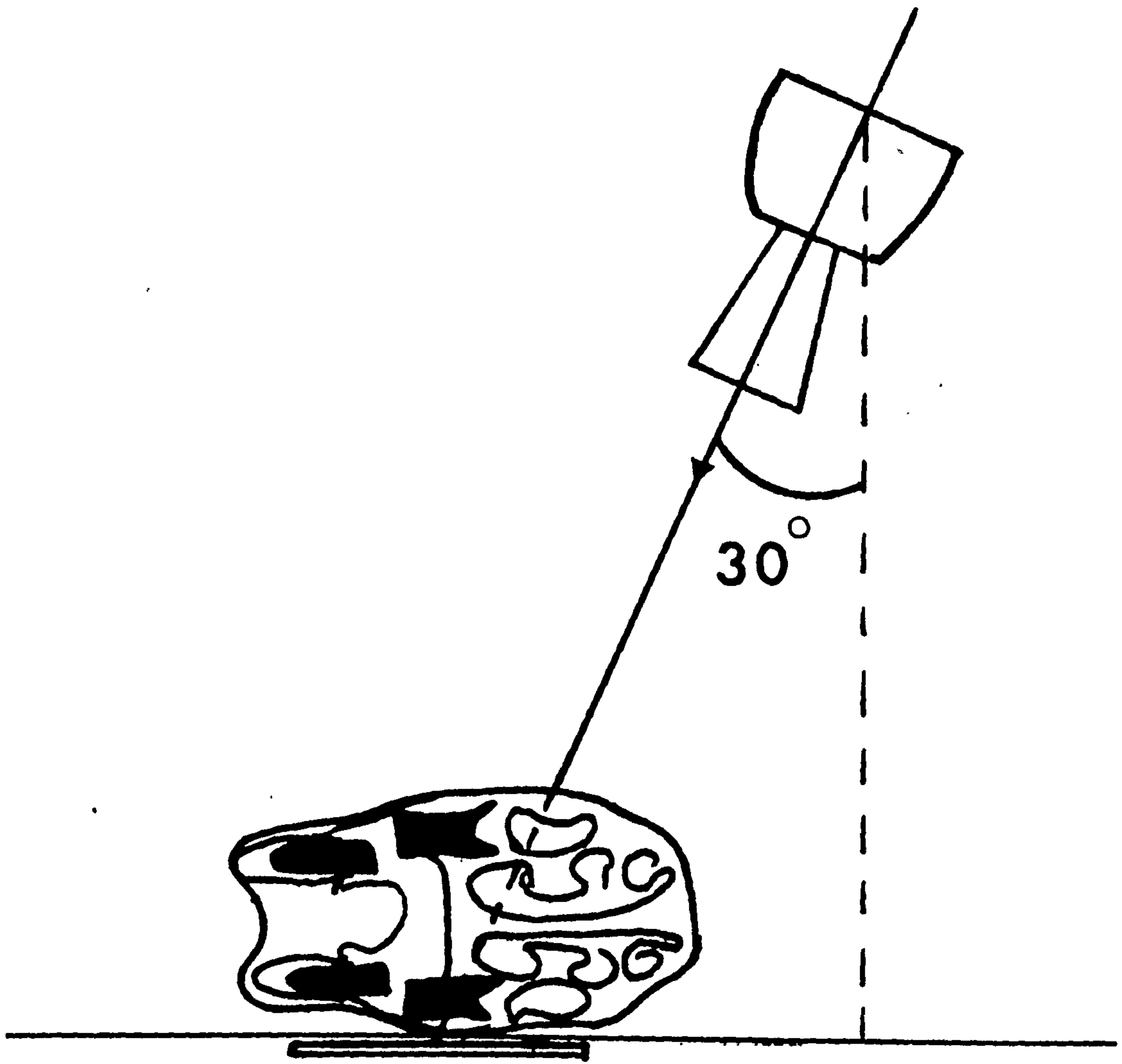


FIGURE 42: Radiographic technique using an oblique beam to overcome superimposition.

Intra-oral, double-wrapped dental films were used to obtain "cone-downed" views of individual tooth roots. For the upper arcade the film was wedged at 45° across the hard palate with the tube head correspondingly angled. For the mandibular arcade a 10.5 x 8 cm film, placed between two perspex sheets to prevent ensalivation and to keep the film flat, was wedged vertically between the tongue and the ramus of the mandible.

Using a film focal distance of 100 cm, gridded cassettes (8/1 ratio) with regular intensifying screens and Kodak X-0 mat films, satisfactory radiographs were obtained with 76 Kv and 25-50 mAs exposure factors.

RESULTS AND TREATMENTS

During the period 1965-1977, 231 cases of equine dental disease were examined and a total of 139 teeth were extracted (Table 7). The cases reviewed have been subdivided into eight clinical categories (Table 8). Four equine types were represented in the survey and Table 9 shows the breakdown of cases into type and sex.

Category 1: Bitting and Riding Problems

Ten animals were examined in both the conscious and anaesthetised state, five were treated under ataractic sedation. Owners and riders complained that the horses were either difficult to ride, hard mouthed, unbalanced or had developed sore mouths.

Enamel points were rasped and hooks removed. In eight horses wolf teeth were extracted using a dental elevator and incisor dental forceps. The crowns of the canine teeth of the eight male horses and ponies were reduced and made smooth using

	<u>No.</u>	<u>%</u>
Incisors	2	1.5
Wolf	14	10.0
Maxillary	56	40.3
Mandibular	67	48.2
	—	—
	139	100.0
	—	—

TABLE 7

Clinical Dental Disease:
Teeth extracted

<u>Category</u>	<u>Diagnosis</u>	<u>No.</u>	<u>%</u>
1	Biting and Riding Problems	15	5.9
2	General Anaesthesia for Dental Prophylaxis	22	8.7
3	Major Irregularities of Arcades	26	10.3
4	Apical Infections (135)		
	a) Maxillary (60)		
	(i) Sinus Empyema	24	
	(ii) Periostitis	36	23.7
	b) Mandibular (75)		
	(i) Dental Fistula	52	
	(ii) Periostitis	23	29.6
5	Delayed Eruptions		
	a) Wolf	4	
	b) Canine	2	9.9
	c) Cheek Teeth	19	
6	Palatal Displacement of 3rd Cheek Tooth	4	1.6
7	Tumours	4	1.6
8	Miscellaneous	22	8.7
TOTAL		253	100.0

TABLE 8 Clinical Dental Disease: Classification of Cases
into eight Clinical Categories

Category	Ponies		Hunters/Cobs		Thoroughbred		Heavy Horses		Total
	M	F	M	F	M	F	M	F	
See Table 8									
1	2	-	6	7	-	-	-	-	15
2	1	-	8	6	4	2	1	-	22
3	6	3	8	7	-	-	1	1	26
4	21	11	40	35	14	11	-	3	135
5	6	8	7	4	-	-	-	-	25
6	-	-	3	1	-	-	-	-	4
7	1	1	-	-	2	-	-	-	4
8	8	5	3	4	-	-	-	2	22
	45	28	75	64	20	13	2	6	
Totals	73		139	33	8				253

TABLE 9 Clinical Dental Disease: Classification of Cases,
Type and Sex of Horses examined.

a rasp.

Follow up reports were available from eleven owners at intervals ranging from six weeks to six months after treatment. Significant improvement in riding and dressage work was reported by six owners and no significant improvement in five cases. It was possible to separate horses that had shown previous normal behaviour from novice or newly broken horses using their anamneses. There was, however, no difference between the response to treatments as reported by the owners.

Category 2: Dental Prophylaxis

In each of the 22 cases in this category it had been impossible to rasp the animals' teeth in the conscious state, even with physical restraint and ataractic sedation. Consequently general anaesthesia or chloral hydrate narcosis was used to facilitate the routine rasping of the dental arcades.

Enamel points were made smooth by longitudinal strokes of the rasp in a rostro-caudal manner and angled so as to ride along the labial edge of the upper arcade and the lingual edge of the lower arcade. Acquired hooks on the first upper and last lower cheek teeth were knocked off, by a clean blow from a stone mason's chisel and mallet followed by short transverse strokes of a small dental rasp. Dental calculus was scraped from the canine teeth and their crowns reduced in size to prevent labial ulcers forming from bit pressure. No after care was given apart from the appropriate tetanus protection (vaccine or antitoxin).

Category 3: Major Irregularities of Dental Arcades

The mean age of the 26 horses in this group was 13.6 years (range 5 years - 28 years). Six animals were found to have such

severe dental malocclusion that euthanasia was recommended because of the resulting gross periodontal disease (see Figure 21). Of the remainder, four animals were treated for primary irregularity of the incisor teeth (Figure 43) and secondary waves of the cheek teeth. In these cases a hacksaw was used to level the crowns of the incisor teeth.

Problems were encountered in developing techniques to correct major step defects, severe wave formation, shear teeth (parvignathism) and other arcade defects. In many old horses (20 years and over) attempts to level molar overgrowths using the chisel and mallet technique resulted in the whole tooth being knocked out. As a precaution radiographs were taken to evaluate the root and reserve crown before striking such overgrowths. Mechanical dental drills and oscillating bone saws proved to be incapable of sectioning the cheek teeth of the horse and air turbine drills and cutters were not available for this work. Consequently the methods employed were limited chiselling followed by hand rasping to try and restore normal occlusal contacts. Following these initial operations owners were advised to seek biannual dental examinations and treatments.

Category 4: Apical Infections

Apical infection was present in 135 horses with an age range of three months to 24 years. The mean age of maxillary apical infections was 10 years and that of mandibular infections was 6 years.

Tooth fractures were responsible for 26 (17.2%) apical infections of the cheek teeth. Of these fractures, 20 (77%) involved the maxillary arcade and only 6 (23%) were found in the



FIGURE 43: Irregular incisors.
17 year old Polo Pony.

mandibular arcade. In addition there were two upper incisor fractures with associated apical periostitis. More than one tooth was involved in 12 cases giving a total of 142 primary apical infections (Table 10).

a) Clinical Signs Of the 135 cases examined signs of dental disease (e.g. mandibular or maxillary swelling and/or fistula formation) had been present from the time of purchase in 19 animals (14.1%). The duration of clinical signs in other cases ranged from four weeks to over two years (mean 5.4 months). Loss of condition was the primary complaint in a further 19 cases (14.1%) and was associated with difficulty in eating, quidding (improperly masticated food bulging the cheeks and dropping from the mouth) as in Figure 44 and halitosis. However, in the majority of the animals (85.9%) body condition was not affected by the presence of apical infections. In these cases the main clinical signs were facial swelling, nasal discharge, buccal ulcerations, mandibular swelling and dental fistula formation (usually single). Such local reactions were often sensitive to palpation and percussion.

Table 10 details the sites of apical infections in the 135 cases (142 teeth). Maxillary sinus empyema accompanied the disease process in 24 cases.

b) Radiographic Features The main radiographic features of apical infections in the horse and pony may be summarised as lysis of periapical alveolar bone and localised osteitis together with changes in the shape of the root apex (Figure 45). Some cases showed sclerosis of the bone surrounding such areas of apical osteitis and the formation of periodontal cysts (Figure 46).

Cheek Tooth	1	2	3	4	5	6	Totals					
Root	R	C	R	C	R	C	R	C				
Maxillary	2	7	9	9	10	11	7	8	0	0	0	0
Total	9	18	21	15	0	0	0	0	63			
Mandibular	0	4	18	22	13	10	5	3	3	1	0	0
Total	4	40	23	8	4	0	0	79	142			

R = Rostral Root
C = Caudal Root

TABLE 10 Clinical Dental Disease: Category 4,
Apical Infection Sites. 135 clinical cases (142 teeth)



FIGURE 44: Quidding, maxillary swelling and
right sided nasal discharge.
10 year old Pony mare with fracture
of third maxillary tooth and apical
infection.



FIGURE 45: 15 year old Pony gelding.
Clubbing of anterior lateral root of second
cheek tooth.

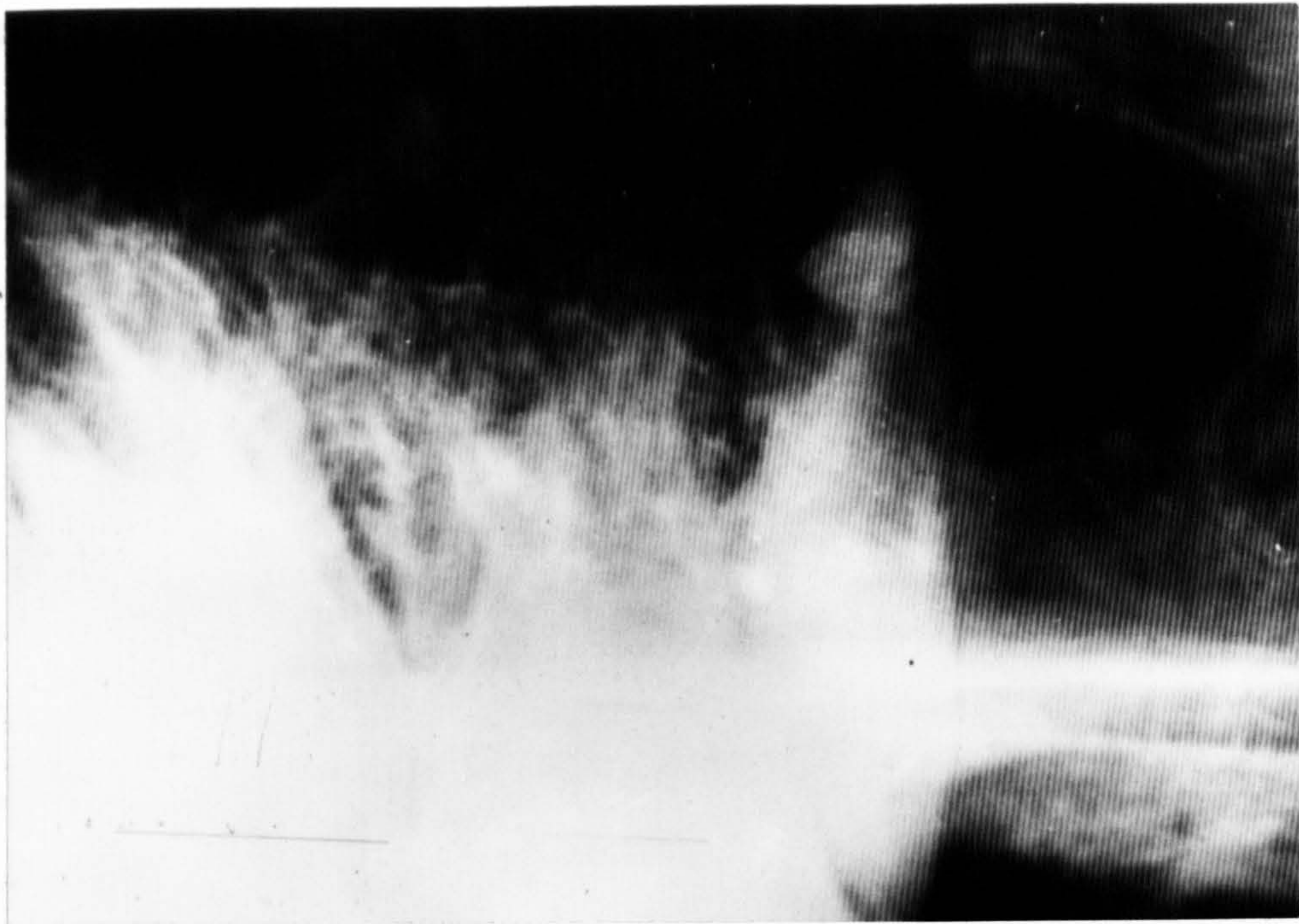


FIGURE 46: 12 year old Pony mare.
Cystic apical osteitis and root nodule of
cementum.

Destruction of the root apex resulted in the formation of clubbed roots and nodules of cementum (Figure 47). In some areas more diffuse ossifying alveolar periostitis was seen (Figure 48).

When the roots of a tooth within the maxillary sinus were diseased the effect was a diffuse ground glass appearance rather than a distinct fluid line on erect lateral films. In chronic maxillary sinus empyema of dental disease origin there may be maxillary bone hyperplasia (Figure 49). Dental fistulae were more readily detected when the mandible was involved (compare Figure 50 and Figure 51). Typically the fistulae had a narrow tract extending from the diseased root to the ventral mandibular margin with a loss of definition of the lamina dura and in some cases localised mandibular osteitis and proliferative periostitis (Figure 52). In a three month old Palomino filly a periodontal pocket between the first and second mandibular cheek teeth resulted in gross alveolar bone lysis and fistula formation. In this case the intra-oral dental film proved to be invaluable in showing the full details of the lesion (Figures 53a and 53b).

c) Surgical Techniques Each case was evaluated after a clinical and radiological examination and the appropriate surgical treatment carried out under general anaesthesia. The affected tooth was manipulated by hand and by dental forceps to ascertain if an oral forceps extraction was possible. In grossly diseased and fractured teeth extraction could frequently be effected per os (26 cases). Incisor teeth were easily removed using a dental elevator to divide the periodontal ligament and loosen each tooth prior to its extraction. Forceps and elevator extraction of the majority of the cheek teeth proved to be more



FIGURE 47: Eight year old Hunter gelding with root fragments and nodules of cementum.

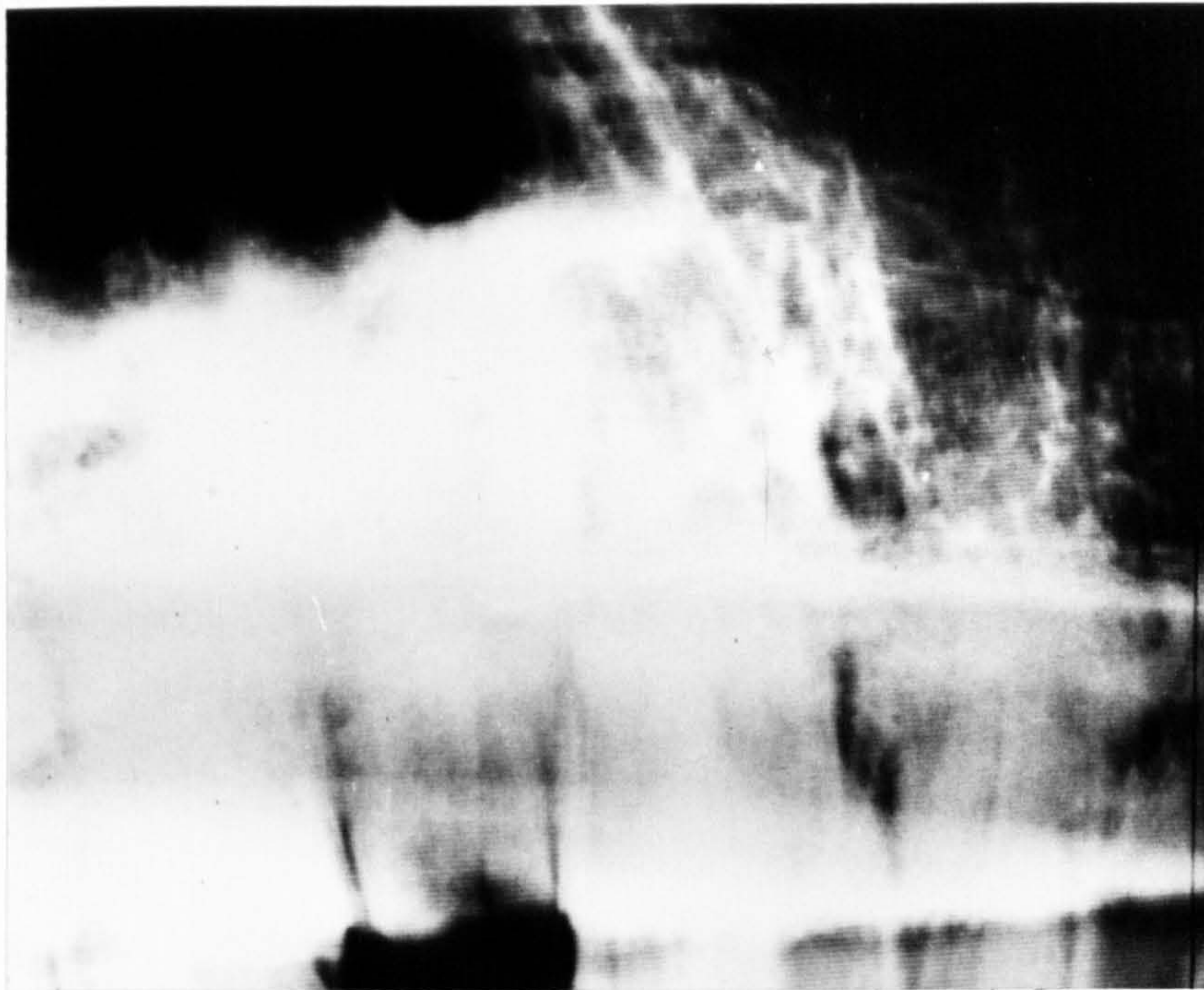


FIGURE 48: 10 year old Pony mare.
Diffuse maxillary alveolar periostitis.
Third tooth has been extracted.

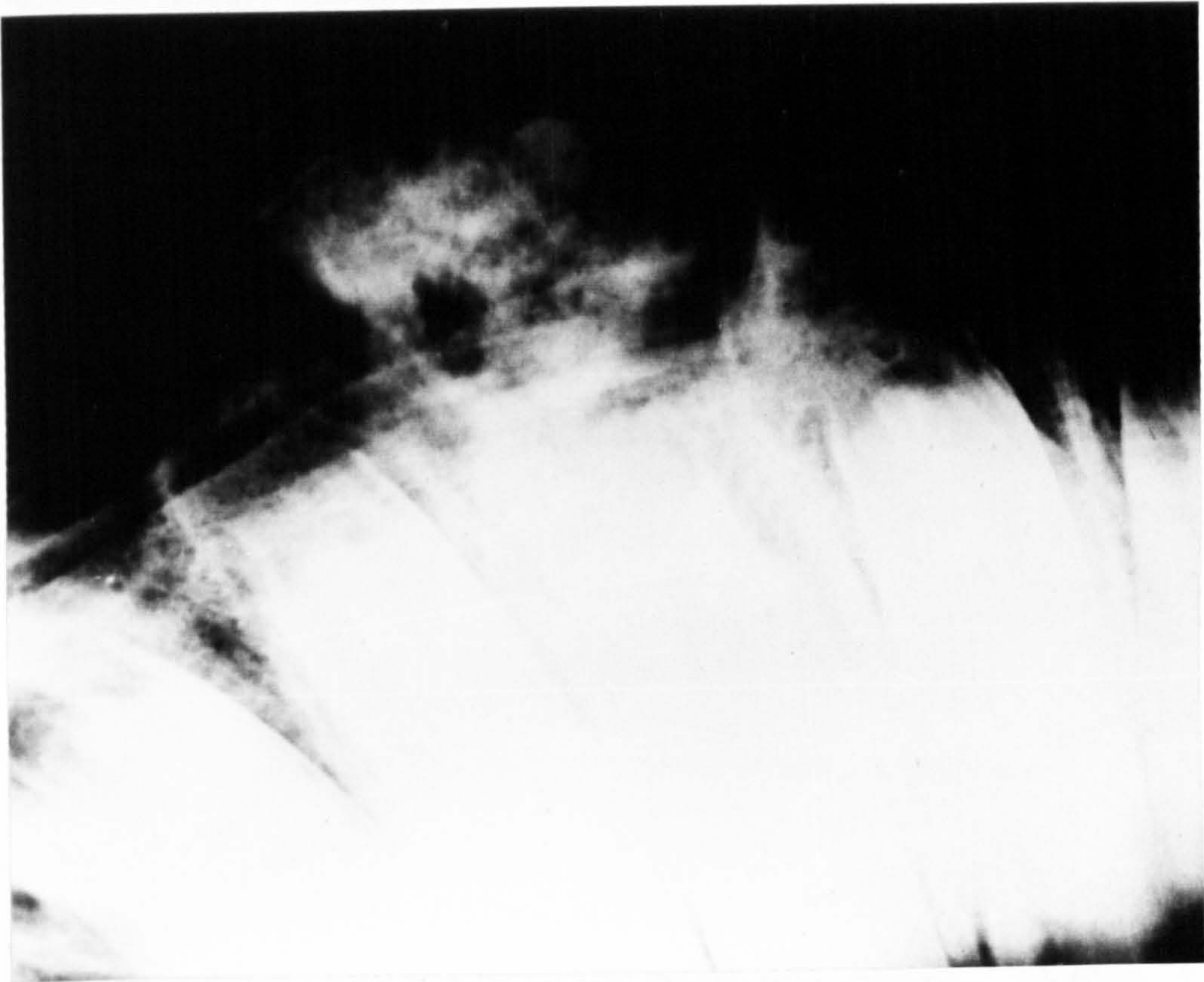


FIGURE 49: Five year old Welsh pony gelding with apical osteitis of fourth tooth, sinus empyema and maxillary bone hyperplasia.



FIGURE 50: Six year old Connemara gelding.
Dental fistula from rostral root of second tooth.
Note loss of lamina dura and sclerotic area of
cementosis.

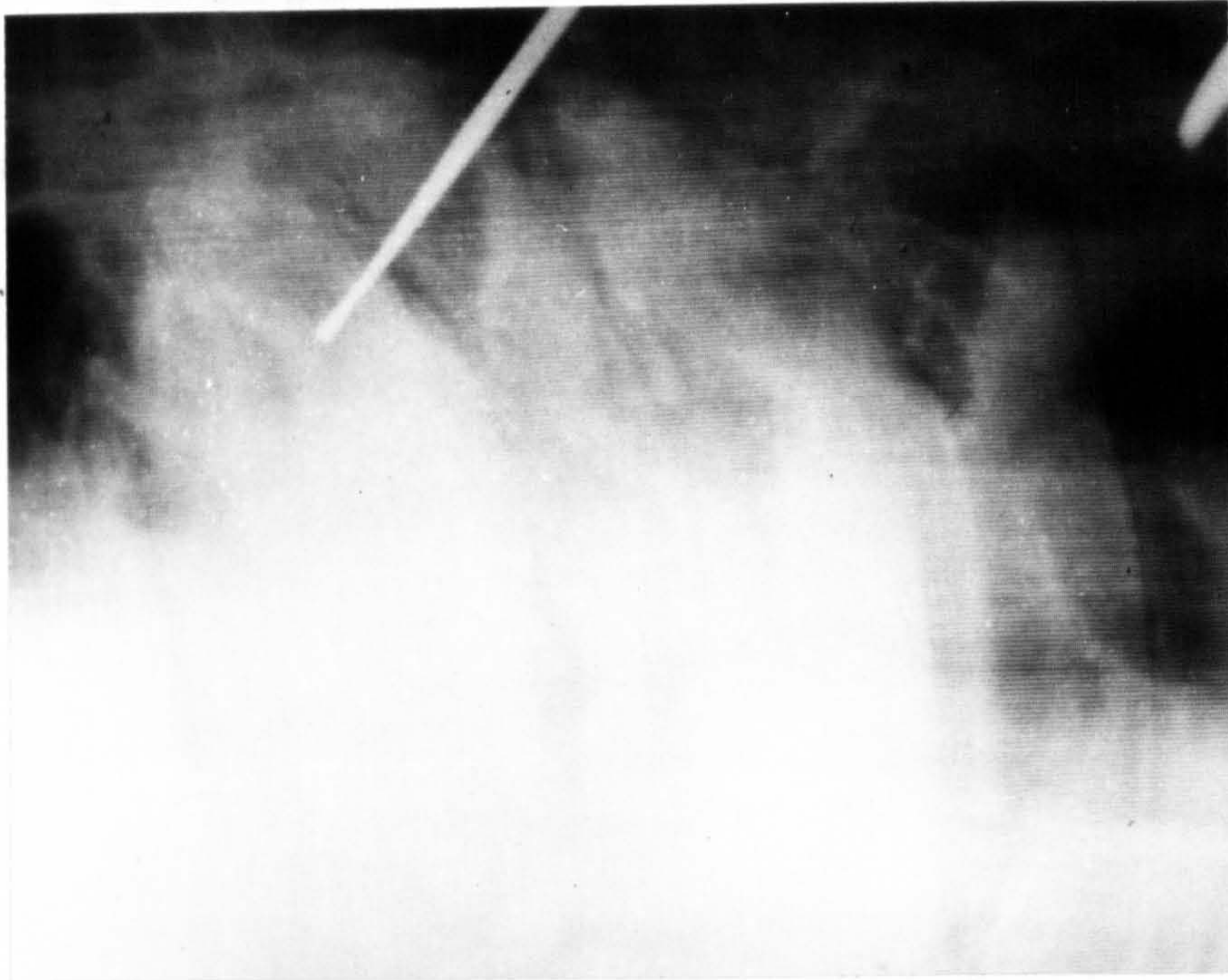


FIGURE 51: Eight year old Thoroughbred.
Probe extends from facial fistula to apical
osteitis of rostral root of third cheek tooth.

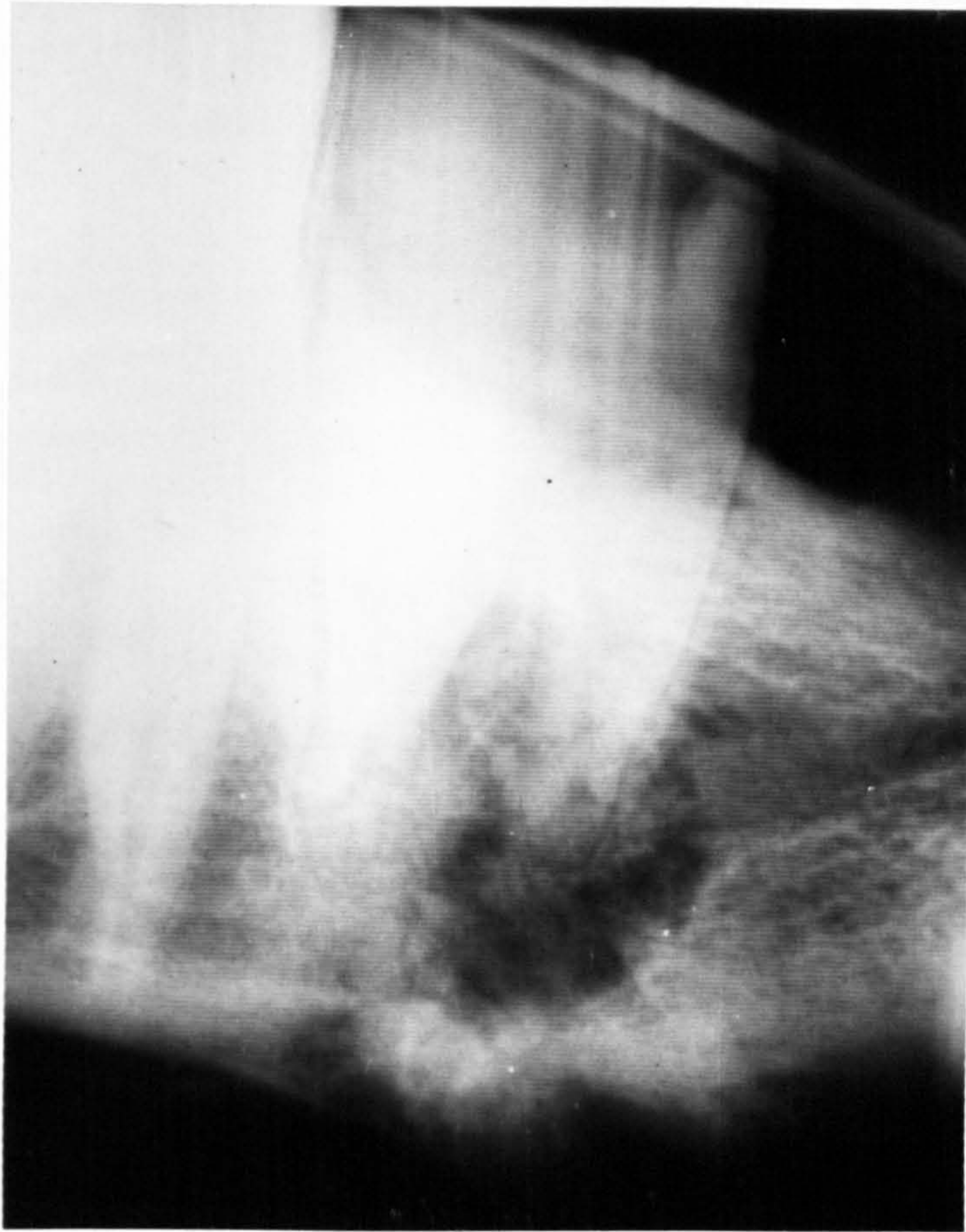


FIGURE 52: Eight year old Hunter mare.
Mandibular osteitis and periostitis
from rostral apical infection.



FIGURE 53a: Three month old Palomino filly.
Ventral fistula formation and mandibular periostitis.



FIGURE 53b: Intra-oral film of 53a.
Alveolar bone lysis between first and second
deciduous teeth.

difficult except in those cases in which the tooth was already loosened by the dental disease process or for the more rostral of the cheek teeth (first or second).

If the diseased tooth could not easily be loosened the technique of mandibular or maxillary bone trephination and extraction by repulsion was carried out. The site of trephination was selected from anatomical landmarks and radiographs of the case. The site was closely clipped, washed with a chlorhexidine/cetremide* mixture and dressed with povidone-iodine solution.** The skin over the site was incised and the periosteum of the underlying bone reflected. A disc of bone was removed by the trephine to expose the apical infection (Figures 54a, 54b and 54c). An 8 mm x 25 cm stainless steel punch and a heavyweight orthopaedic steel mallet were then used to repulse the tooth. Care was taken to ensure that the angle of repulsion was such that the punch made continuous contact with the tooth. This prevented the punch from sliding off the diseased tooth either laterally or medially and damaging the alveolar bone or the palatine artery.

Following extraction of the tooth thorough curettage of the alveolus was carried out. Necrotic bone and remnants of the apical granuloma were debrided as were fragments of tooth root and nodules of cementum. Post-operative radiographs were used to check that all such potential sequestra had been removed. The socket was then irrigated with a one per cent hydrogen peroxide solution followed by normal saline. A plug of dental wax***

* Savlon, I.C.I., Macclesfield, Cheshire

** Pevidine, Berk Pharmaceuticals Ltd., Shalford, Surrey.

*** Pink Modelling Wax, Cottrell & Co., London.



FIGURE 54a: Site of incision along ventral border of mandible.



FIGURE 54b: Reflection of periosteum and exposure of mandibular granuloma.



FIGURE 54c: Mandibular trephine exposes apical granuloma.

was then moulded into the socket from the mouth to prevent food material contaminating the socket. The wax plug was mushroomed over the socket and arranged so that no occlusal contact was made (Figure 55 and Figure 56). The trephine site was packed initially with sterile guaze and then treated as an open wound by daily irrigation. Particular care was taken in the post-operative irrigation of the maxillary sinuses after trephination and extraction of those cheek teeth with roots and reserve crowns within the paranasal sinuses. In such cases the animals were exercised daily to encourage the sinus exudate to drain into the nasal cavity and, if necessary, follow up examinations were made under general anaesthesia to ensure that the sinuses were not contaminated by food material.

Post-operative injections of penicillin and streptomycin were given for five days as well as the appropriate antitetanus prophylaxis (vaccine or antitoxin).

In four cases a flap of alveolar bone was removed to expose the infected root apex and an attempt was made to conserve the tooth after apical curettage as described by Eisenmenger (1959) (Figure 57). In three of the four cases the teeth were subsequently extracted because of recurrent swelling and discharge.

d) Healing of Extraction Wounds After removal of a tooth the unpacked area of the extraction wound filled with blood. The blood coagulated and, even in irrigated wounds, the clot began to be replaced by granulation tissue within three days. The dental wax oral plug was made redundant by 21 days, when the socket had been filled with organised fibrous tissue (Figure 58). The gingival epithelial margins migrated across the defect so that

FIGURE 55: Position of wax plug in mandibular tooth socket.

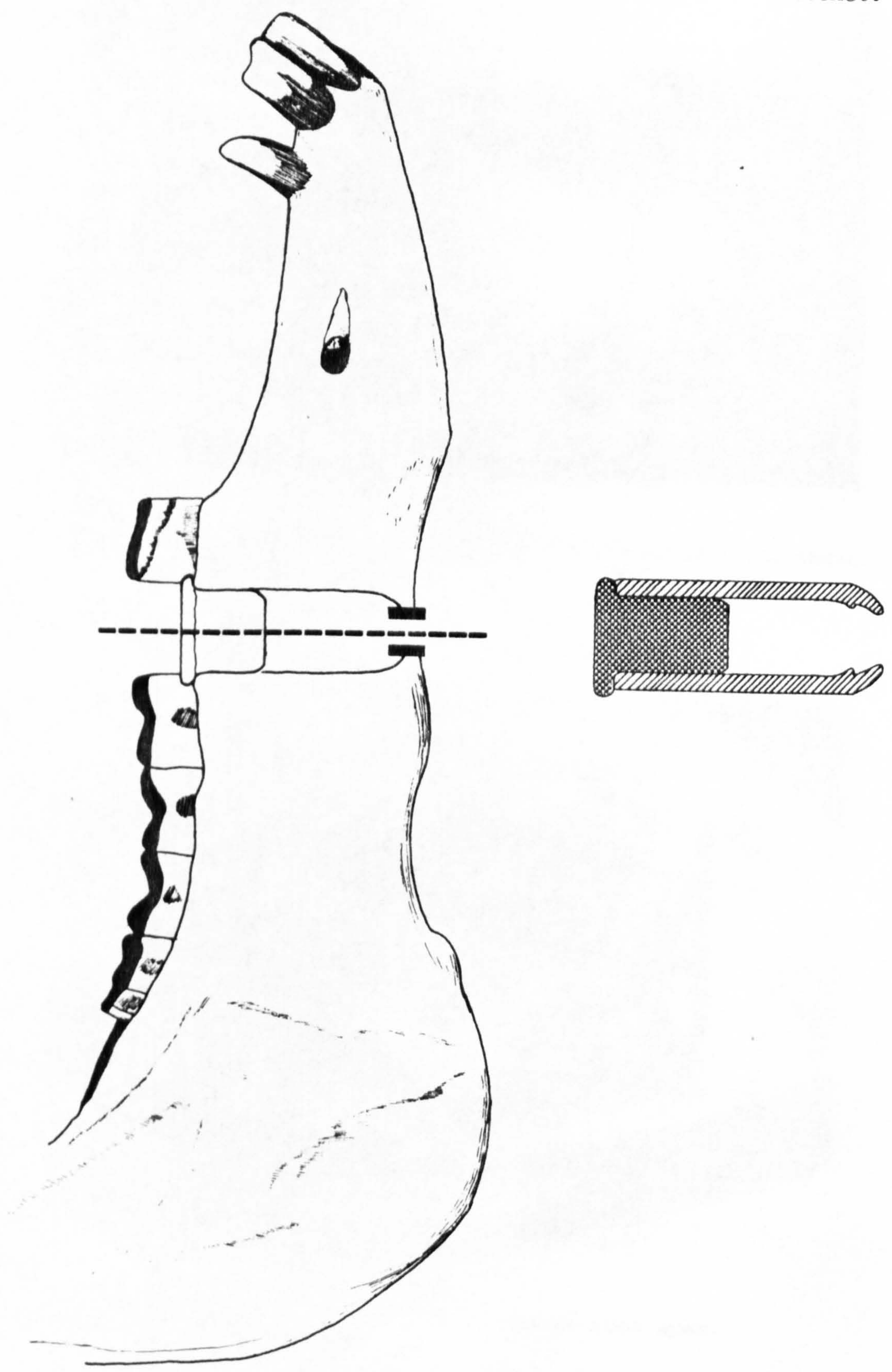




FIGURE 56: Gutta percha plug after extraction of fifth tooth.

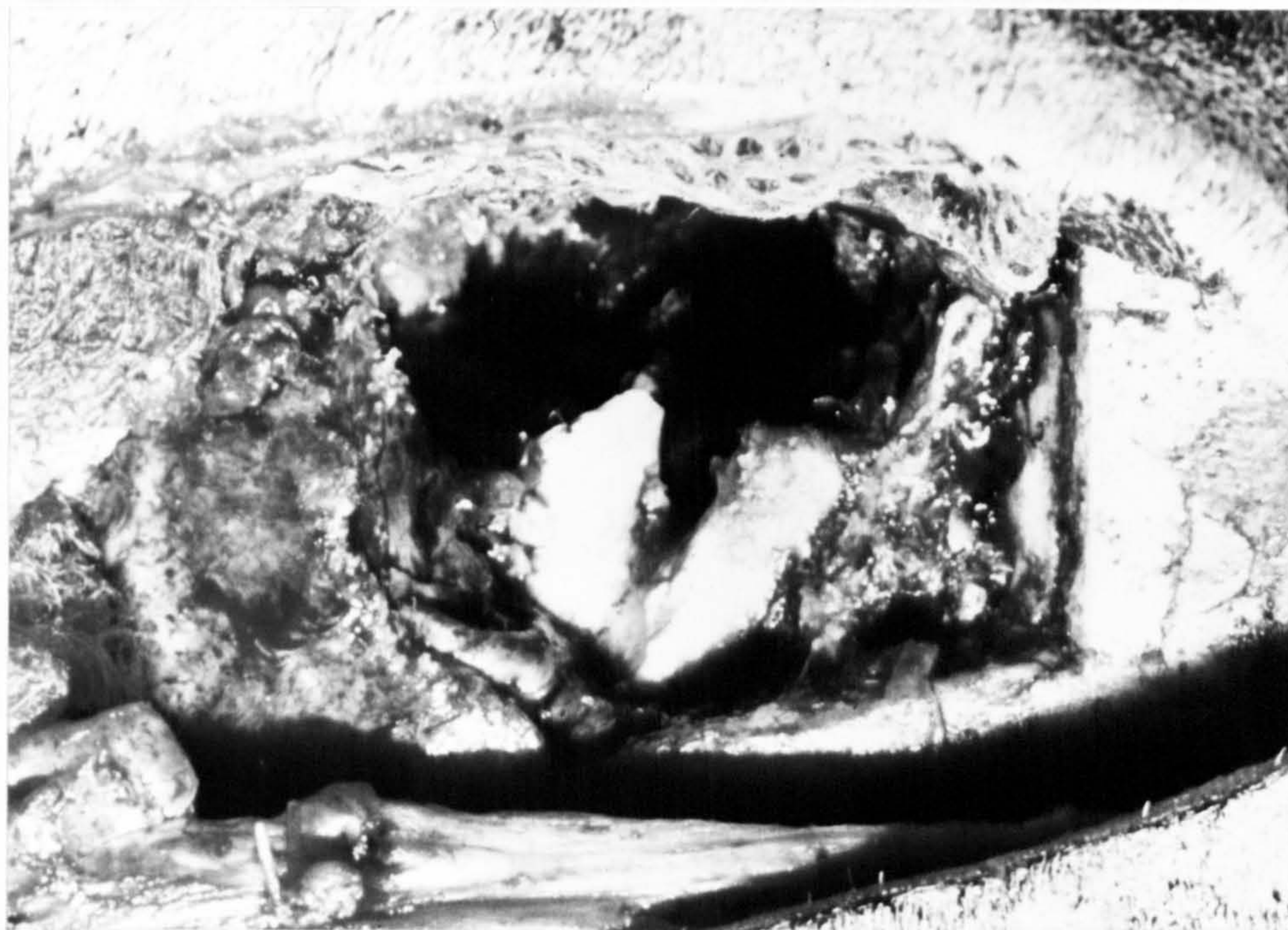


FIGURE 57: Maxillary bone flap to expose root apex.

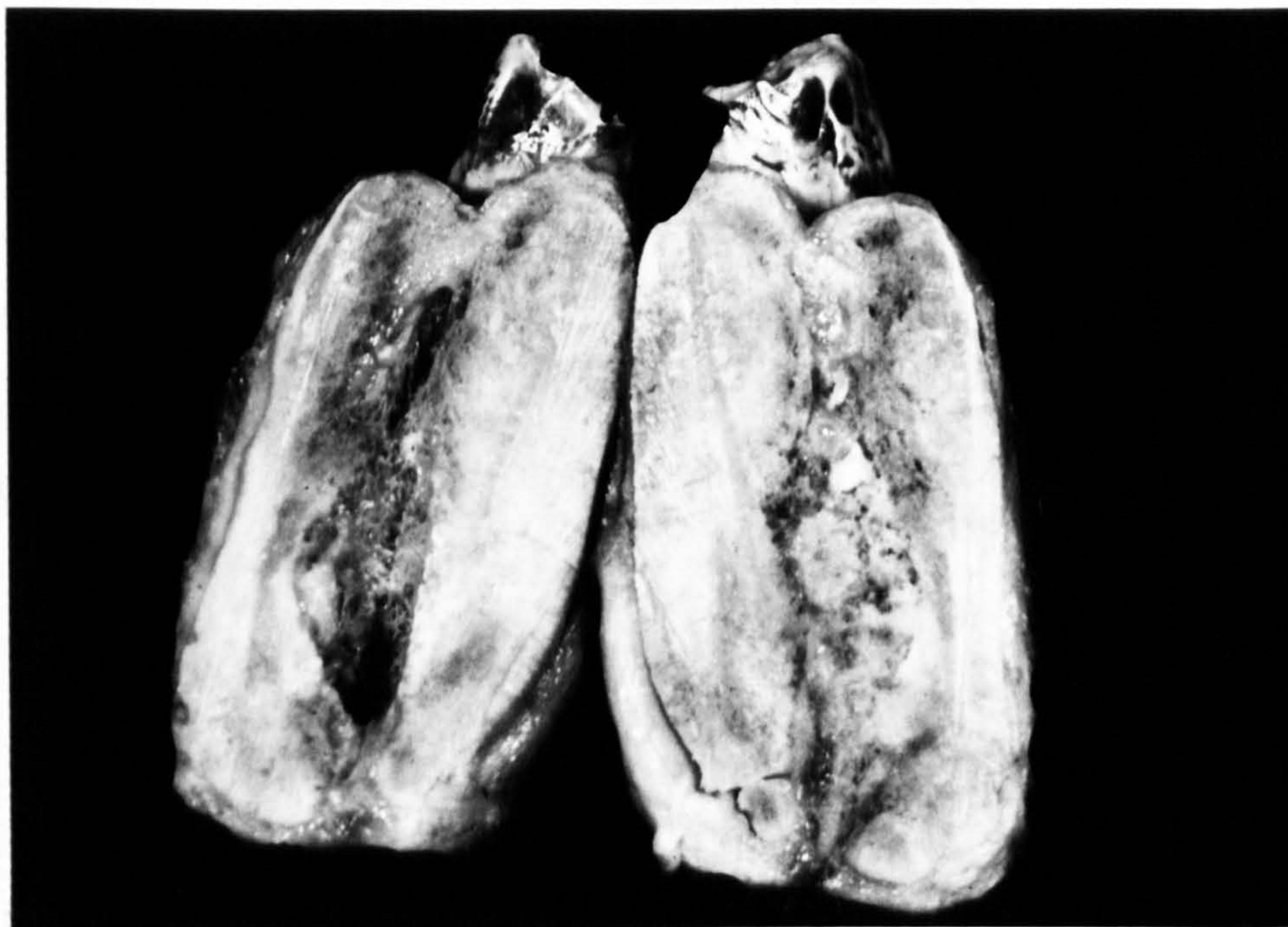


FIGURE 58: Vertical section of healing mandibular tooth socket.



FIGURE 59: Healed maxillary socket.

epithelialization was completed within 30 days (Figure 59). By this time considerable osteoclast activity had taken place in the alveolar wall and many micro-sequestra were removed. As the socket filled with new bone the adjacent teeth rotated rostrally and caudally to narrow the gap in the dental arcade (Figure 60).

e) Complications of Dental Extractions In this series by far the most common complication was the development of persistent socket infection with a discharging fistula at the trephine site. It was found that 20 per cent of horses required further socket curettage after extractions. In a number of cases root fragments and/or sequestra of bone and cementum, not visible on the immediate post-operative radiographs, were detected as nodules on the second series of films as they enlarged due to the deposition of cementum on the bone fragment. After decalcification and the preparation of histological sections stained with haematoxylin and eosin, it was found that these nodules, which could be seen on some radiographs of infected teeth (as in Figure 47) or which appeared after extraction, were made of cementum (Figure 61).

One eight year old hunter mare developed a cervical vertebral abscess four months after extraction of the third left mandibular cheek tooth. The mare had a stiff neck and was unable to feed or drink from the ground. She showed progressive ataxia and had great difficulty in rising. Post-mortem examination showed C3/C4 vertebral osteomyelitis with cord pressure (Figures 62a and 62b). No bacterial growth was seen from a culture of the mandibular socket. The vertebral abscess yielded a mixed infection of staphylococci (aerobic, coagulase negative, β haemolytic) and streptococci (anaerobic, non-haemolytic).



FIGURE 60: Healed mandibular socket with rotation of adjacent teeth.

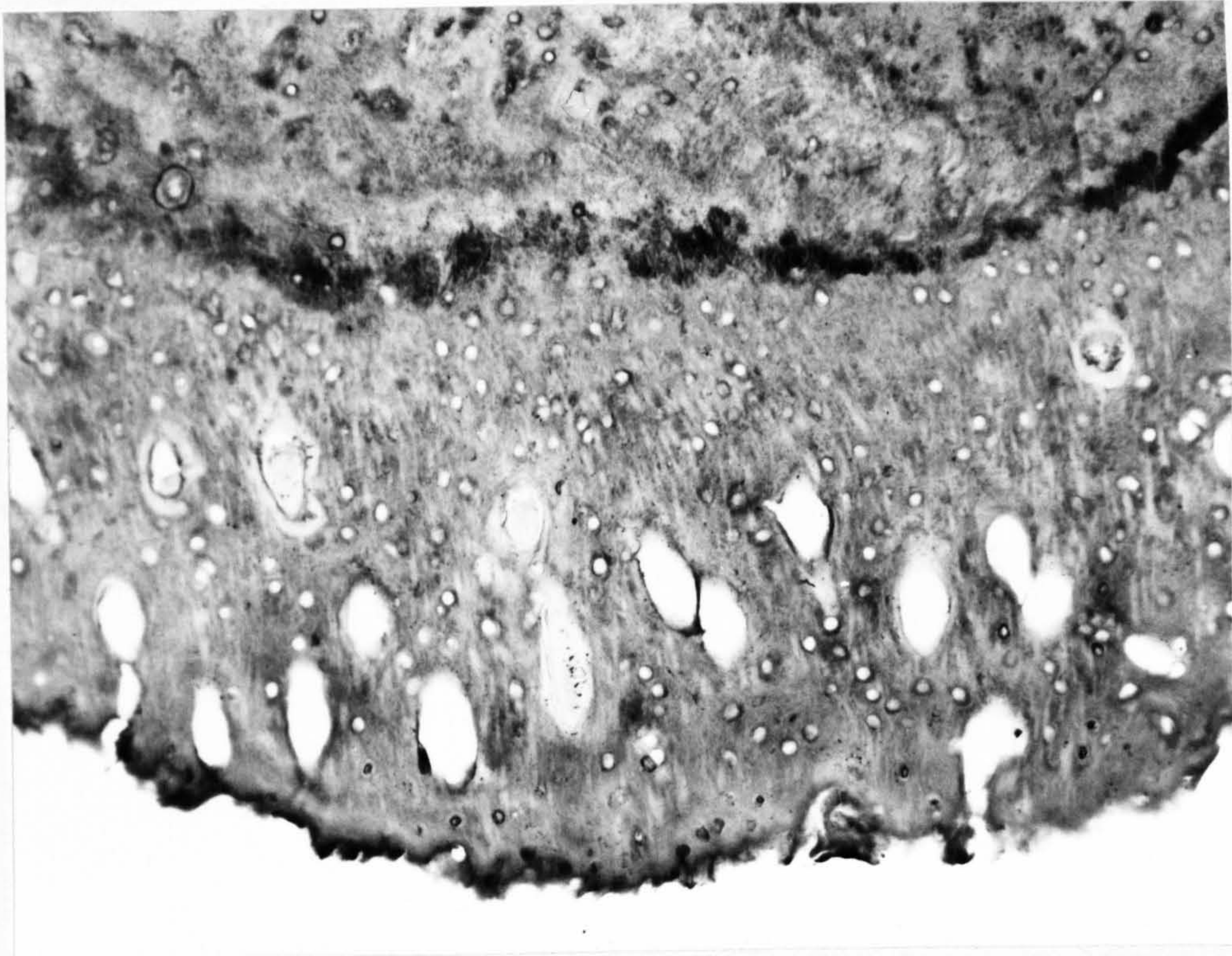
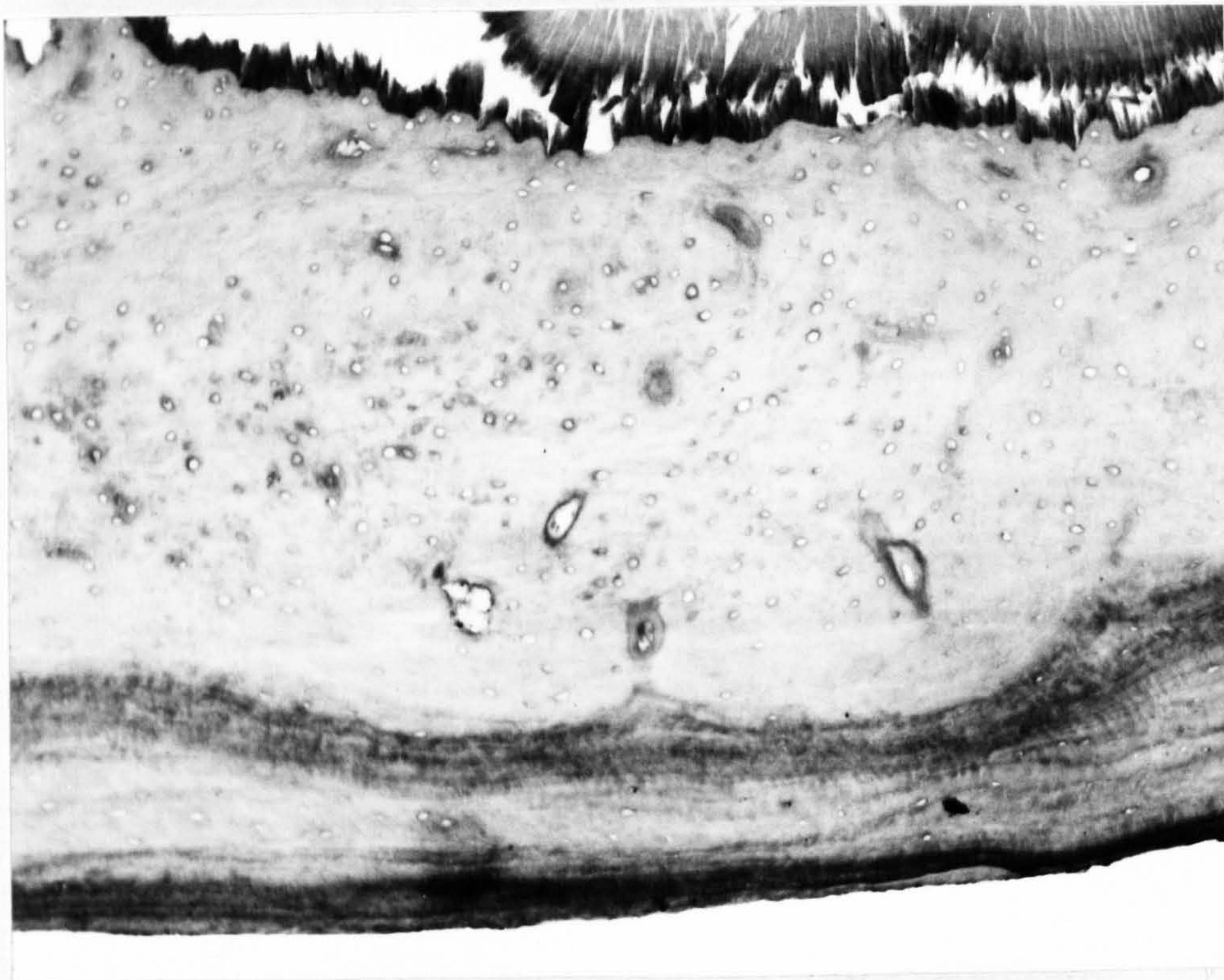
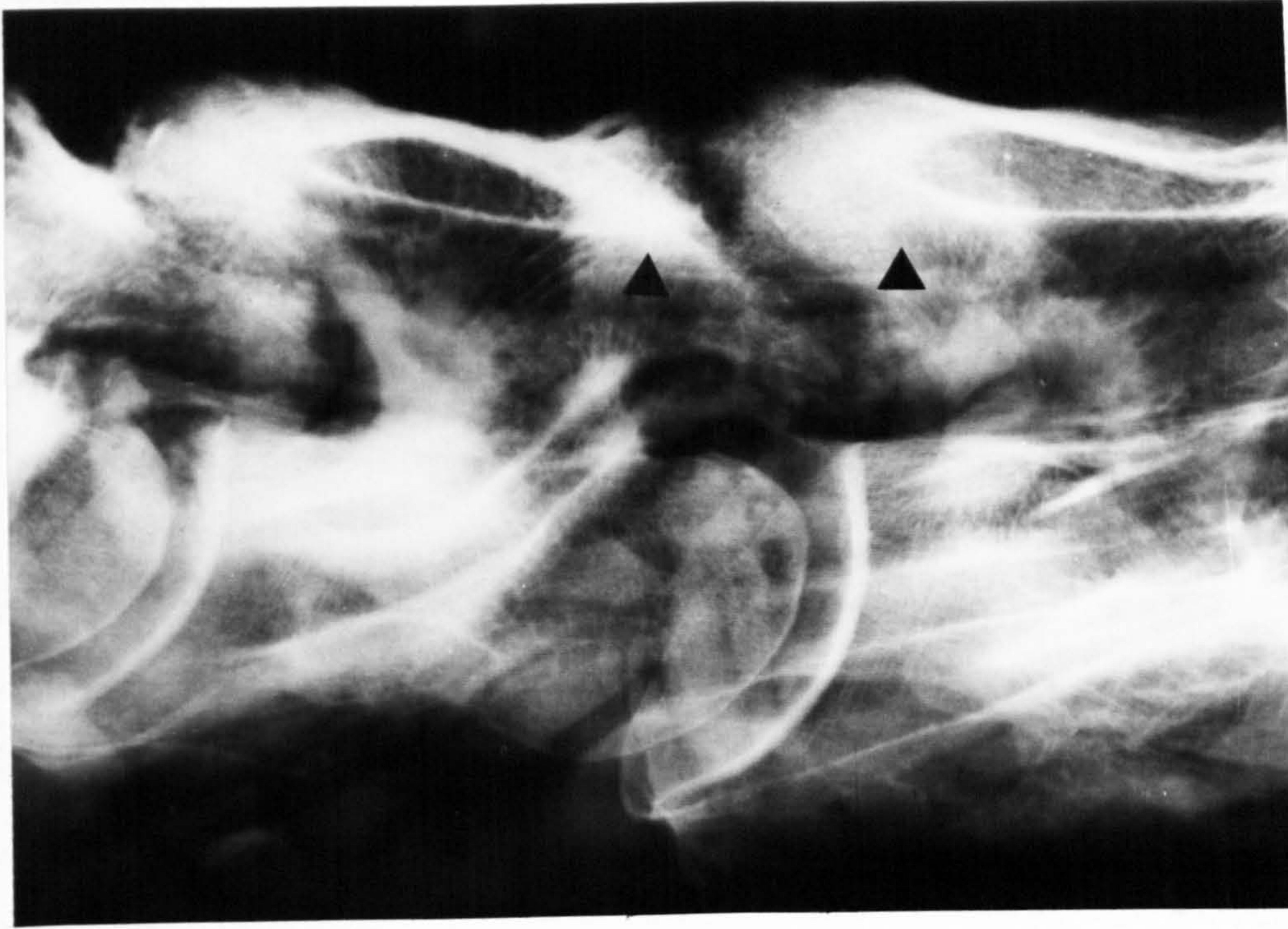
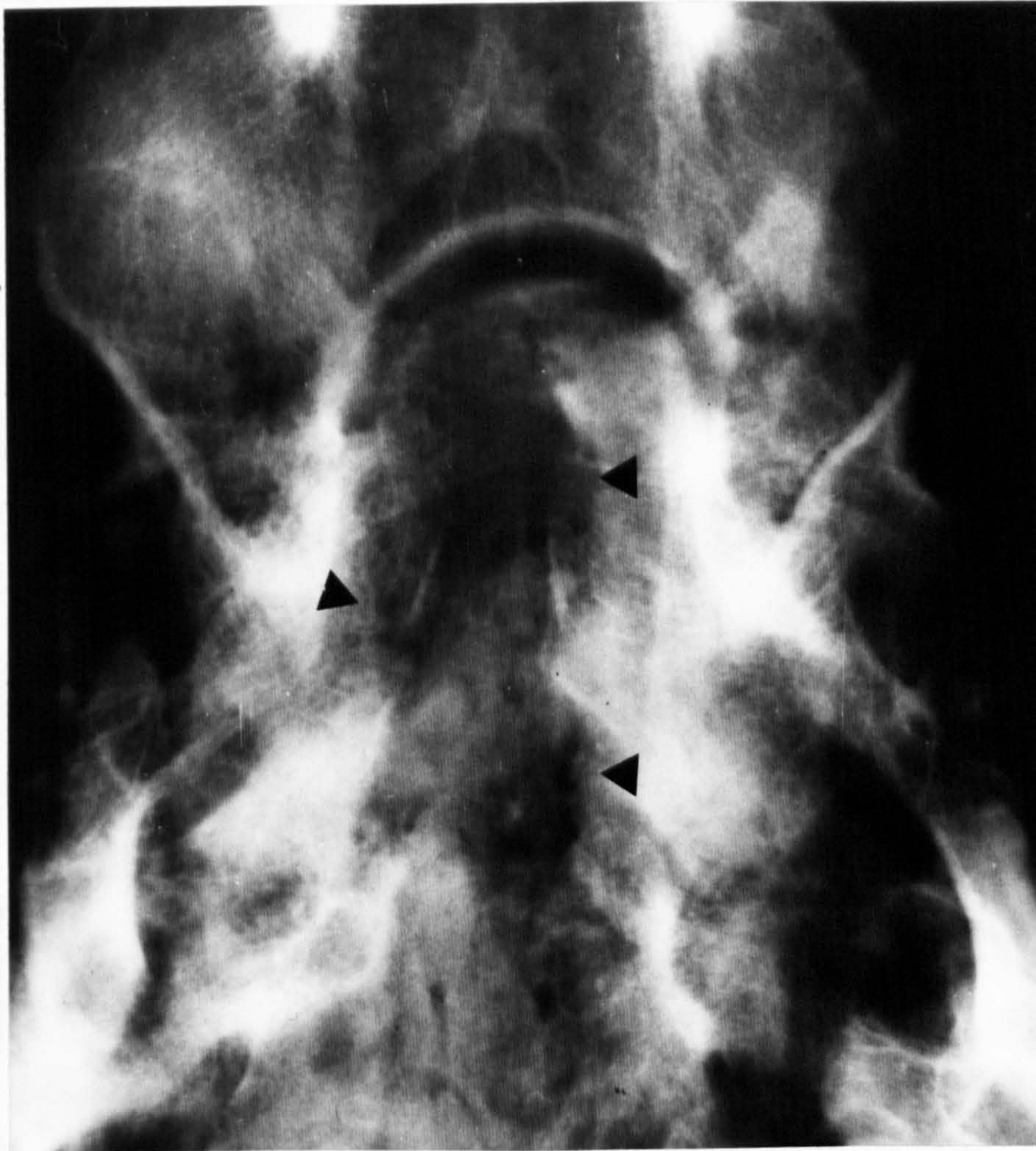


FIGURE 61: Nodule of cementum from apical infection.
Compare with compact cementum of tooth crown (below).
Decalcified H and E x 110.





FIGURES 62a and b: Lateral and ventro-dorsal radiographs of C₂/C₃ vertebral abscess.



No cases of "dry socket" i.e. alveolar osteitis or alveolitis sicca dolorosa (Shafer, Hine and Levy, 1963) were encountered in follow up examinations or reports from owners.

In a few cases the dental surgery resulted in excessive oral trauma. There was severe haemorrhage from the palatine artery in two cases and although the artery could not be ligated the haemorrhage was controlled by pressure from the wax plug. Large fragments of mandibular alveolar bone were lost in four cases but no major mandibular fractures developed.

Following dental extraction of the primary apical infection there was modification of occlusal contact to a greater or lesser degree. Care was taken to treat enamel points and acquired overgrowths at the time the diseased tooth was extracted and subsequently six monthly dental prophylaxis was recommended to prevent the formation of large hooks.

f) Pathology of Extracted Teeth A total of 123 cheek teeth were available for study (56 maxillary and 67 mandibular teeth). A considerable number were removed in multiple fragments and were grossly contaminated following fracture and fragmentation of the crown. Consequently only 69 teeth (26 maxillary and 43 mandibular) were examined in detail.

Twenty of the maxillary teeth (76.9%) were found to have hypoplasia or caries of cementum with extension of the disease process into the pulp and in some cases the coalescence of the disintegration of the rostral and caudal lesions resulted in fracture of the tooth (Figure 63 and Figure 64). In the majority of mandibular teeth no primary factor was found for the presence of the pulpitis and apical infections. The sequelae of pulpitis

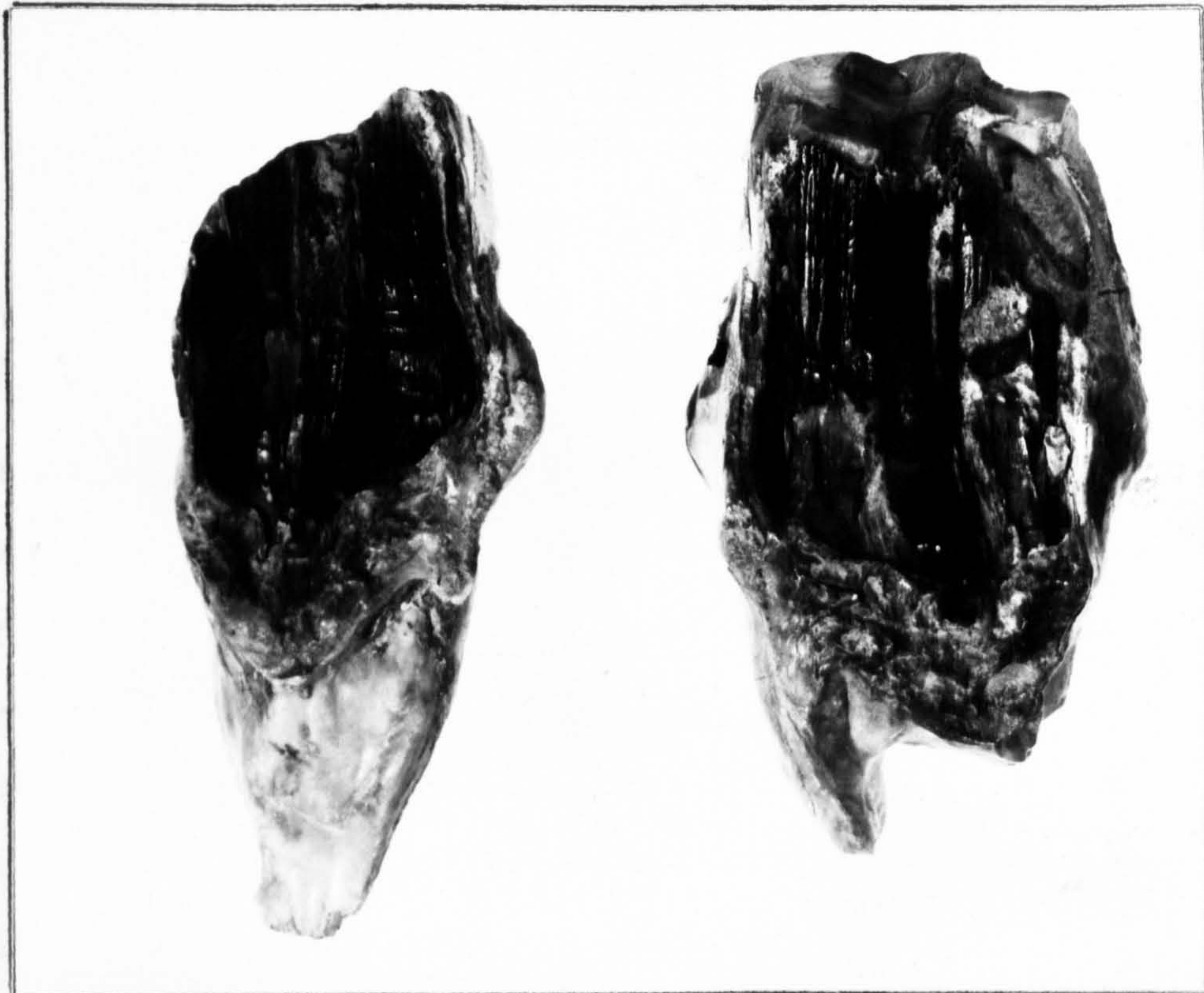


FIGURE 63: Fourth cheek tooth from an eighteen year old pony mare.
Fracture of tooth as a result of caries of cementum.

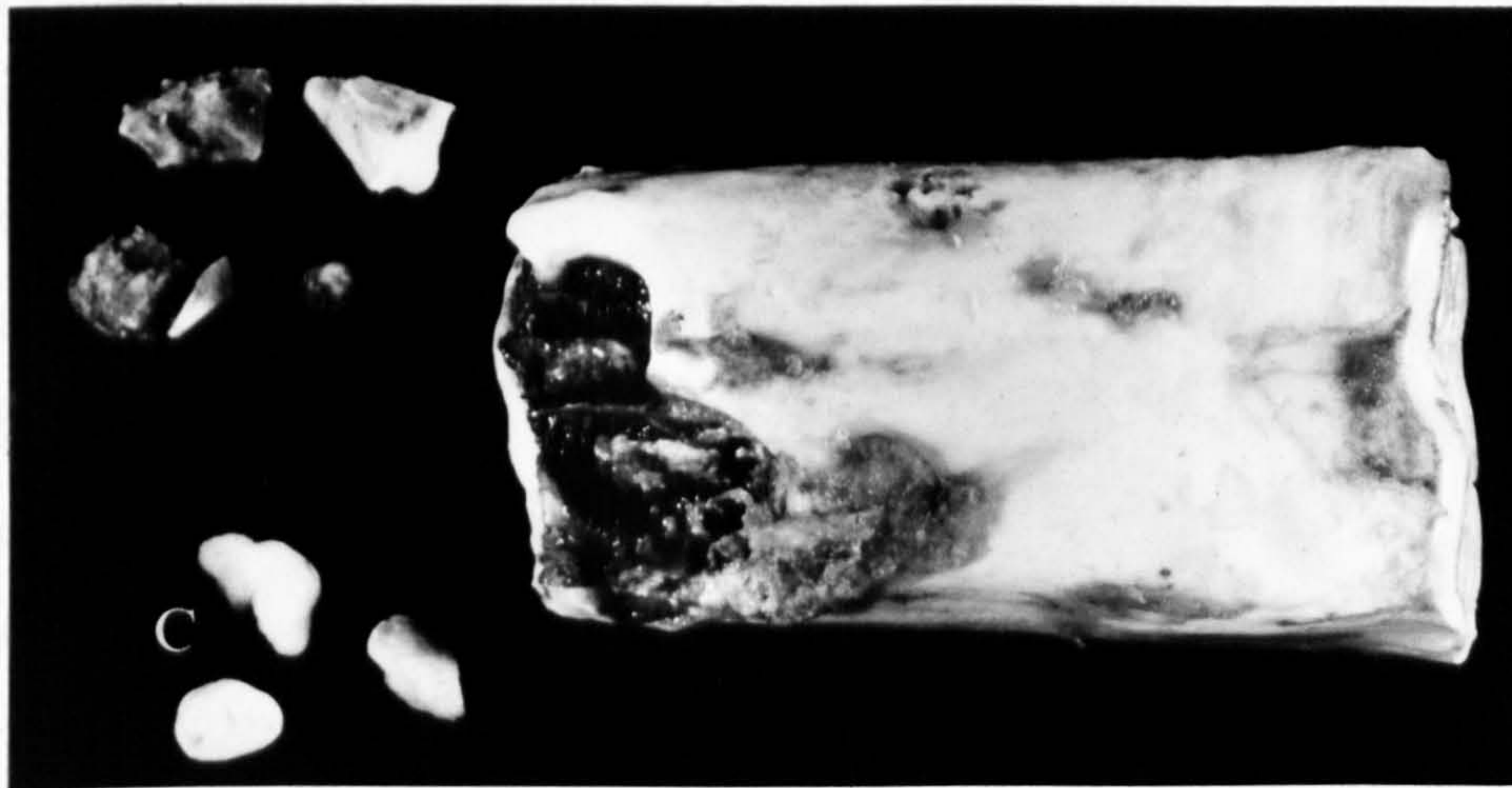


FIGURE 64: Second mandibular tooth from a five year old Thoroughbred.
Tooth fragments and nodules of cementum (C).

of the cheek teeth of the horse are summarised in Figure 65.

The pulp of the extracted teeth was malodorous and filled with decaying food material so that most of the tooth substance was dead (Figure 66). There was evidence of peripheral cementoblast activity leading to thickening of the peripheral cementum, which exhibited a smoothly irregular surface when compared with that of a normal tooth (Figure 67). Secondary dentine formation inside the pulp cavity was minimal in most cases. In many decayed teeth secondary fractures occurred through the dentine plane, detaching a lateral slab of enamel and dentine from the labial aspect of the tooth.

Category 5: Delayed Eruptions

A total of 25 animals were diagnosed as suffering from retarded or delayed eruption. The four wolf tooth cases and two canine tooth cases were regarded as primary delayed eruptions and were seen as gingival swellings, within which the dental crown could be palpated (Figure 68). The wolf tooth cases were 2-6 years of age (normal eruption time 9 months) and the retarded canine teeth were both found in 5 year olds (normal eruption time 4-5 years). The wolf teeth were extracted and the gingivum incised over the canine teeth and their subsequent eruption was uneventful.

Delayed eruption of the permanent cheek teeth was usually the result of a degree of dental overcrowding so that the crown of the third cheek tooth was impacted. This resulted in the formation of "eruption cysts" (Figure 69), with marked mandibular and facial swellings in three year old animals. Whenever possible the eruption was assisted by cutting the interproximal edges from

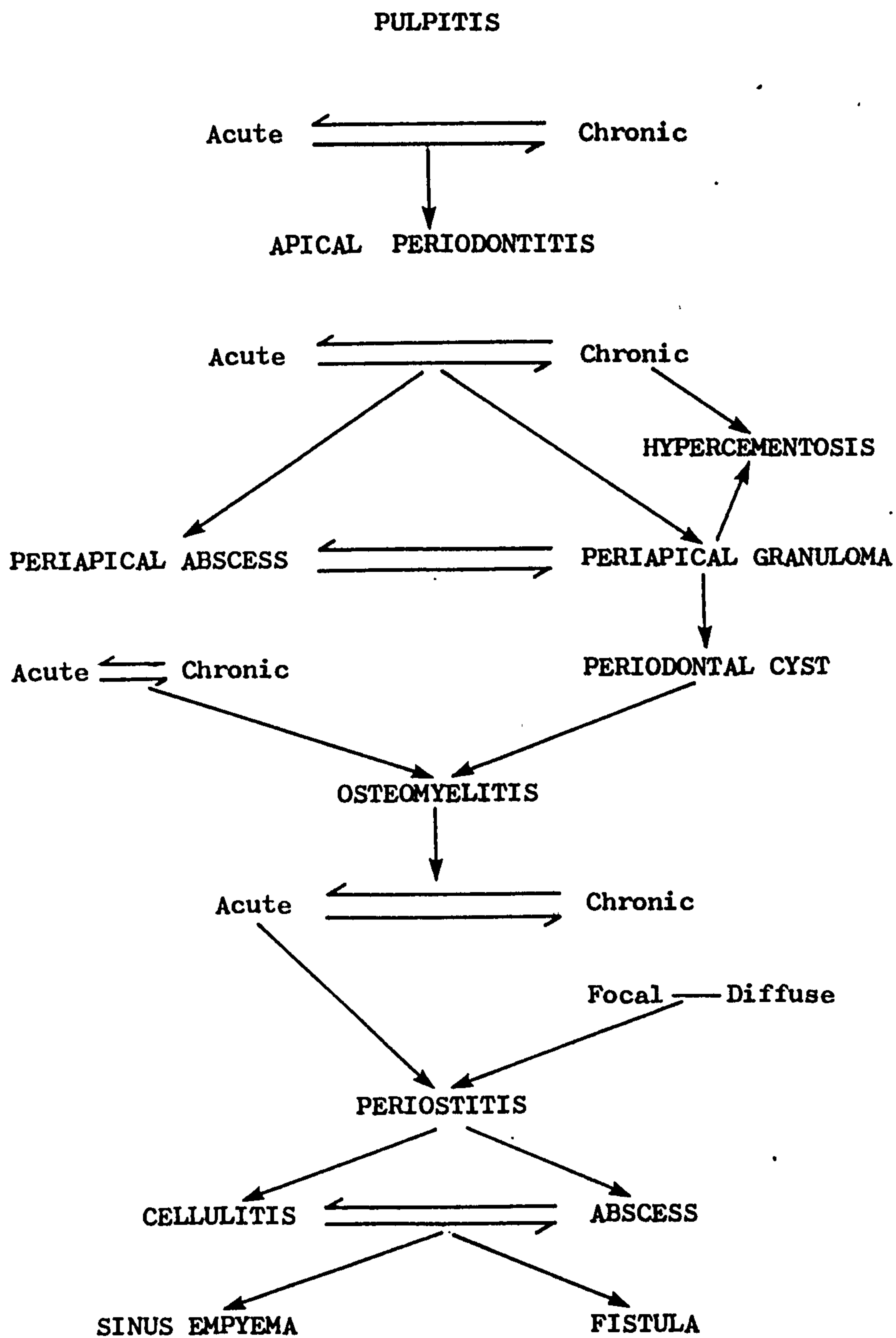


FIGURE 65 Inter-relationships of Periapical Infections in the Horse (Modified after Shafer, Hine and Levy, 1963)

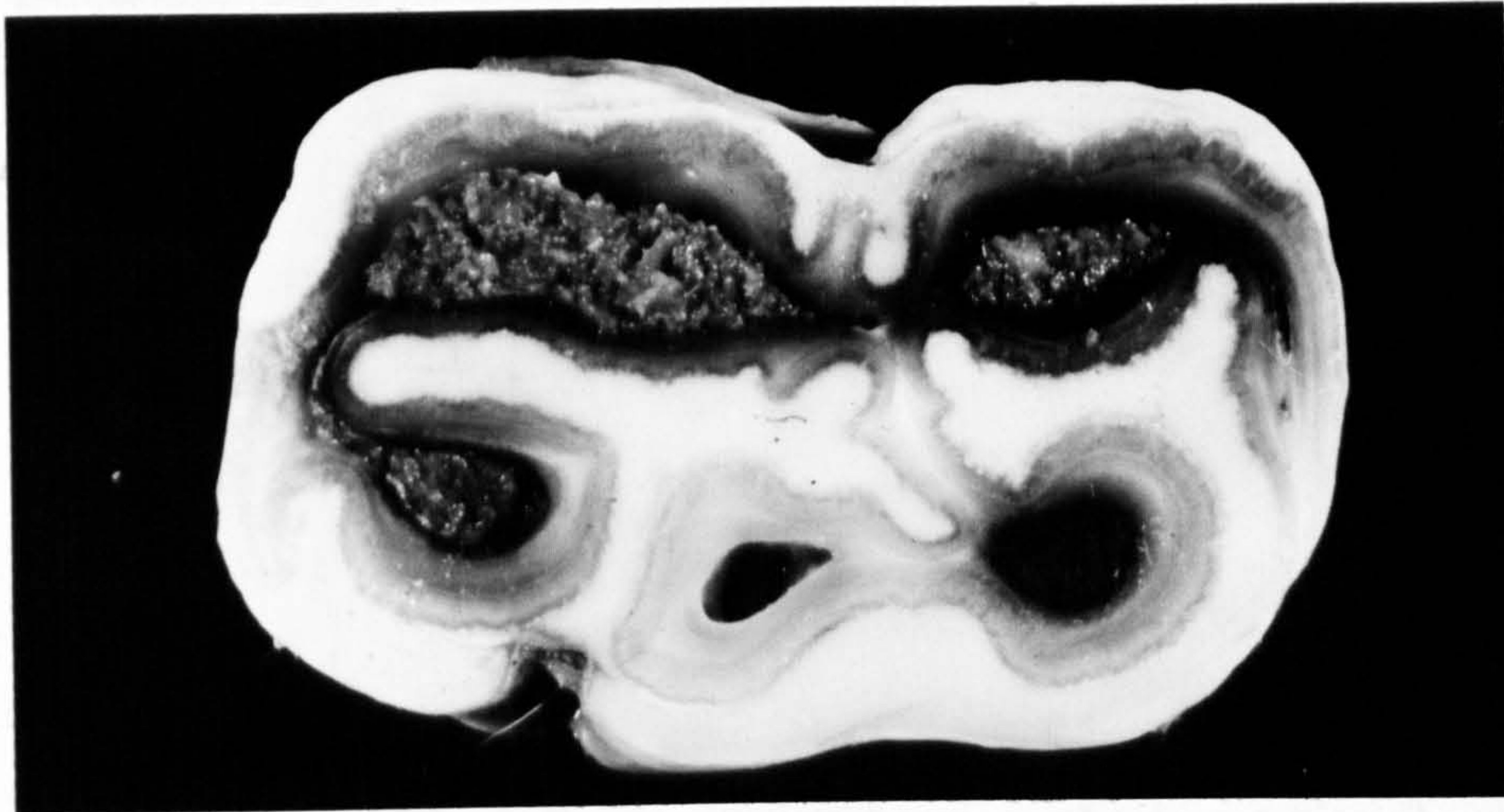


FIGURE 66: Transverse section of decayed mandibular tooth.
Food debris packed into pulp cavity.

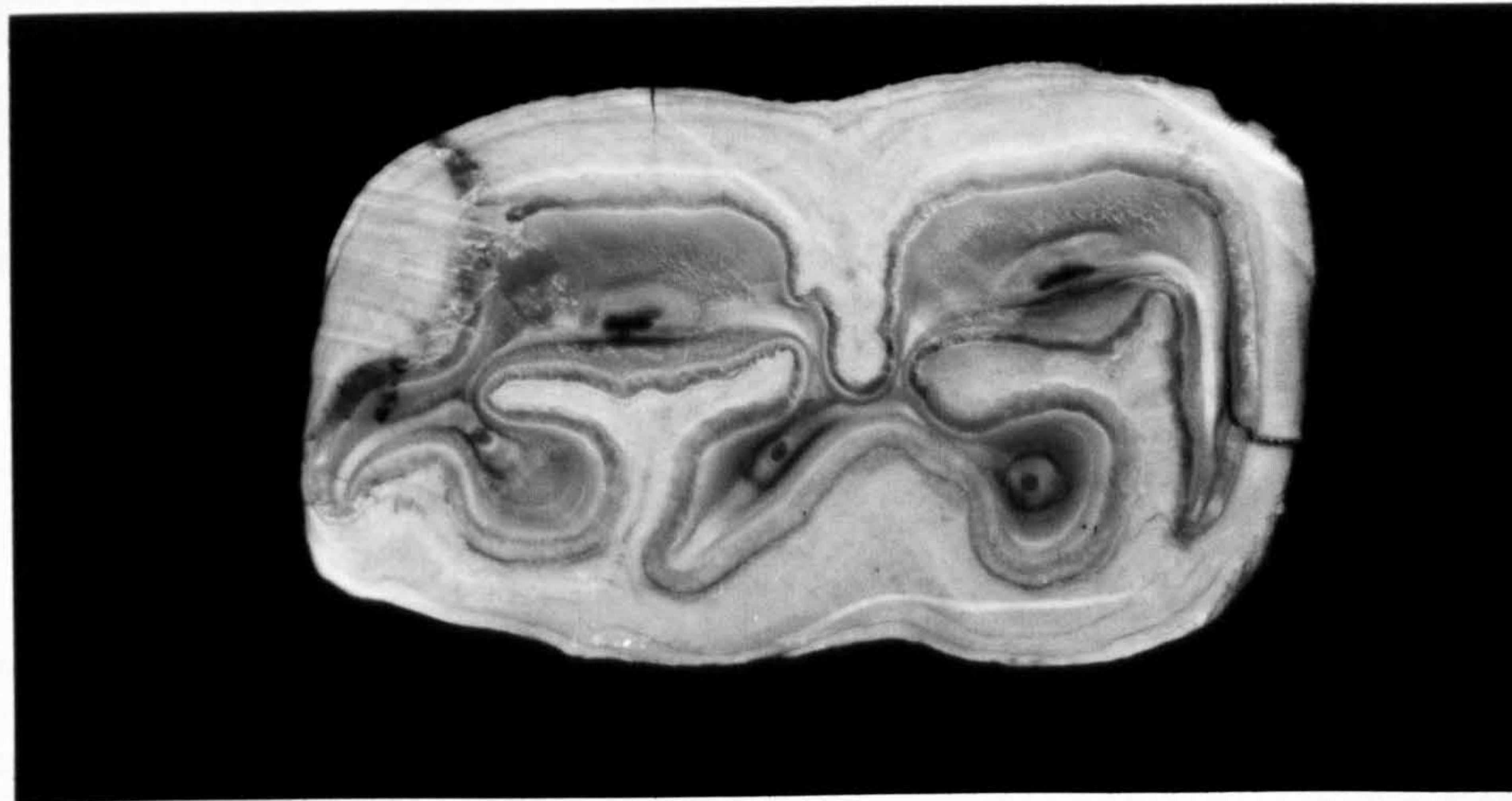


FIGURE 67: Transverse section of normal mandibular tooth
for comparison with Figure 66.



FIGURE 68: Impacted wolf tooth. Five year old Cob mare.

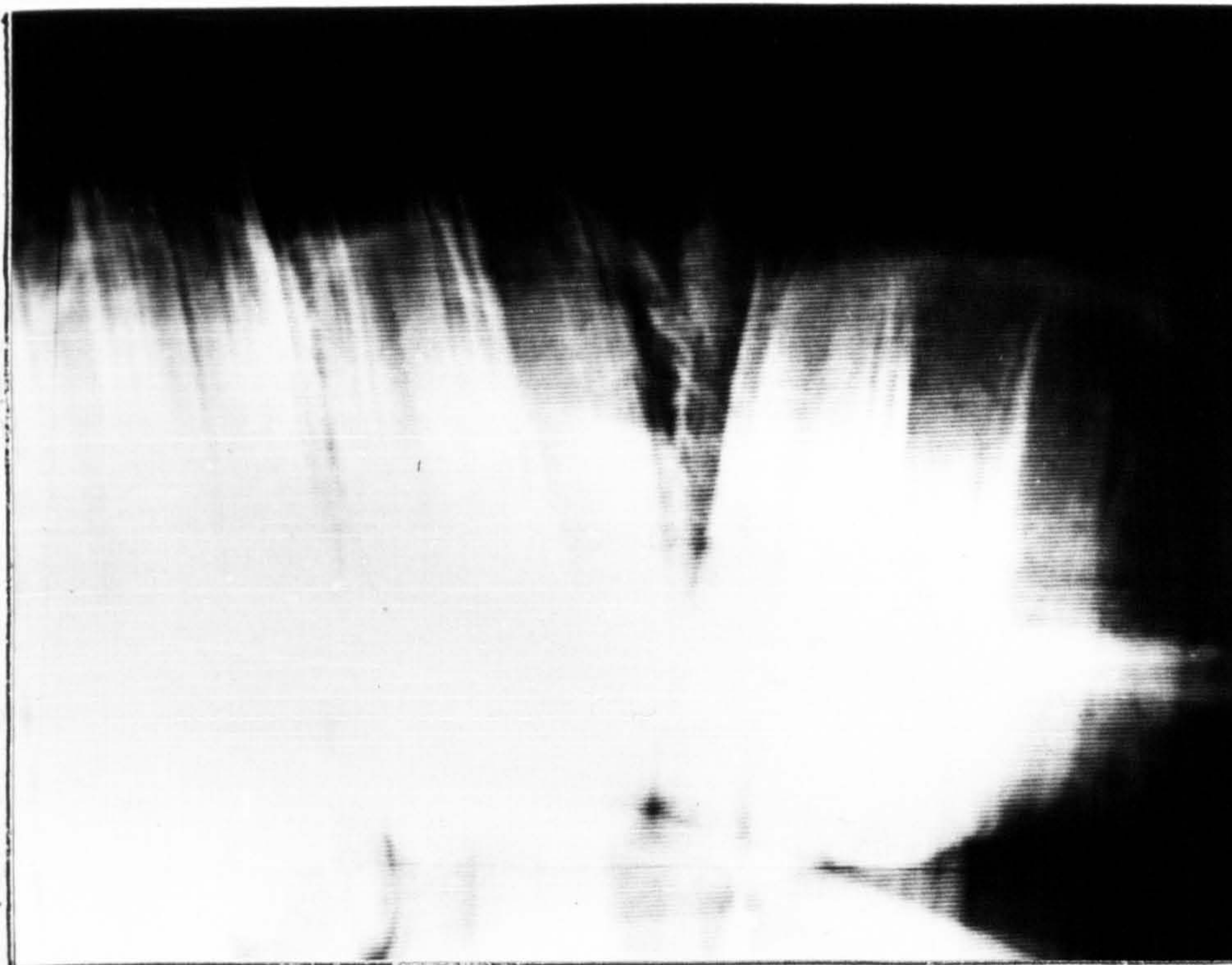


FIGURE 69: Three year old Highland pony with maxillary swelling associated with "eruption cysts".

the adjacent crowns using a dental drill. This was not effective in all cases and subsequently dental fistulae developed up to two years later (Figures 70a and 70b). In the majority of cases, however, the mandibular and facial swellings (and rhinitis) resolved spontaneously as root formation and eruption took place.

Category 6: Palatal Displacement of Third Cheek Tooth

Four cases of this specific maleruption were seen with clinical signs of improper mastication and occasional quidding (Figure 71). The only treatment given was to ensure that the adjacent teeth were cut back at their interproximal edges.

Category 7: Tumours

One congenital maxillary cyst was seen in a one day old Thoroughbred foal with left-sided facial distortion that prevented it from sucking. Radiographic examination and dissection of the specimen proved the disease to be multiple with a large left-sided cyst, a smaller right-sided cyst and early cystic degeneration of both mandibles around the third deciduous tooth (Figures 72a and 72b).

Two cases of ameloblastic odontoma were diagnosed in ponies aged nine months and 15 months, but only one of these specimens was available for detailed study. The tumours presented as slowly enlarging maxillary swellings causing nasal obstruction and discharge. Radiographs showed a variegated, radiodense mass from which normal tooth structures were absent. After decalcification, haematoxylin and eosin sections showed tissues of both epithelial and mesenchymal origins (Figures 73a and 73b). The tumour was seen to be made of epithelial follicles consisting of central stellate cells bordered by columnar cells, resembling the enamel



FIGURE 70a: Five year old part Arab mare.
Painful swelling of mandible and distension of
lamina dura of second tooth.

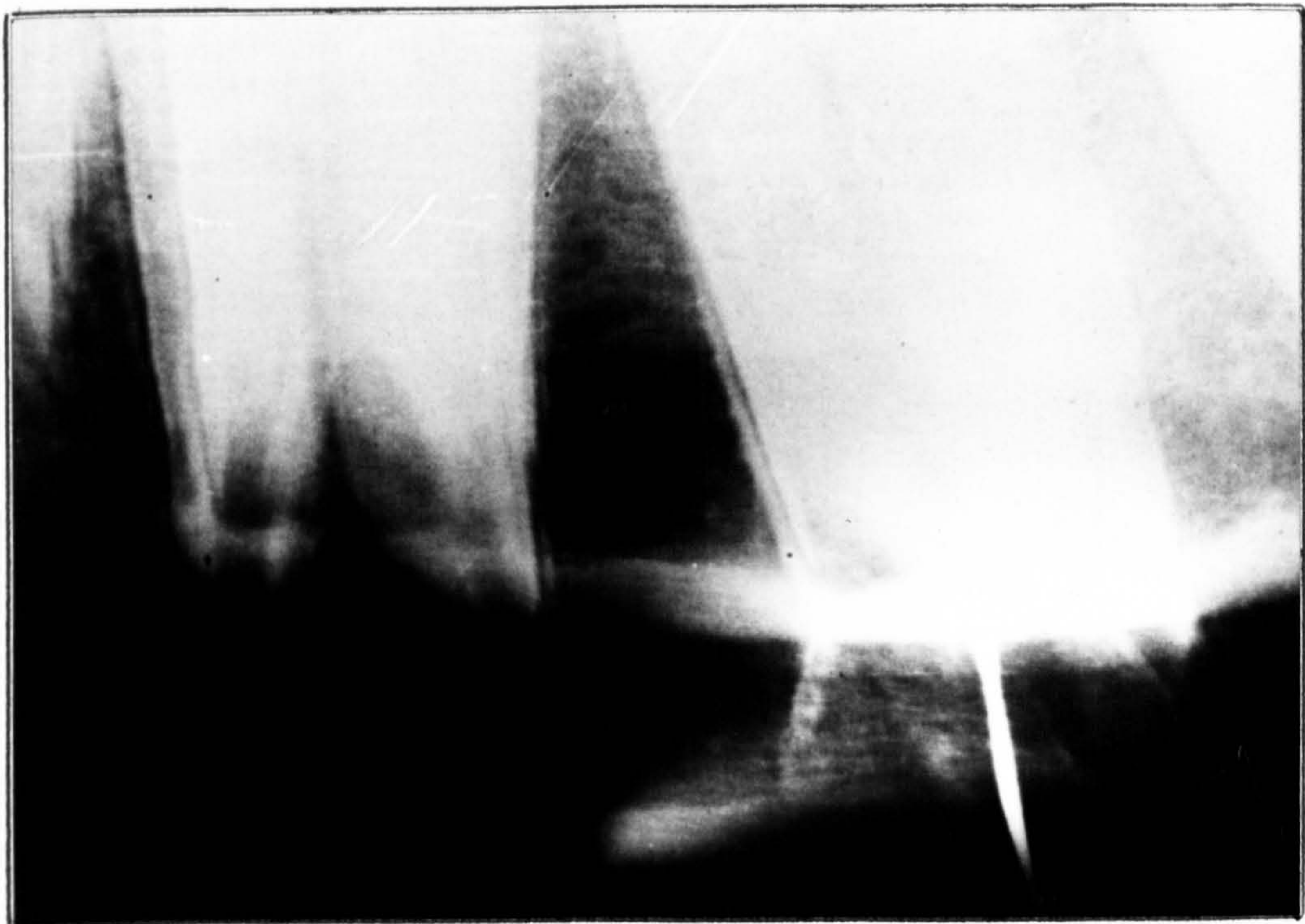


FIGURE 70b: After two years the swelling has regressed
but a dental fistula has developed.

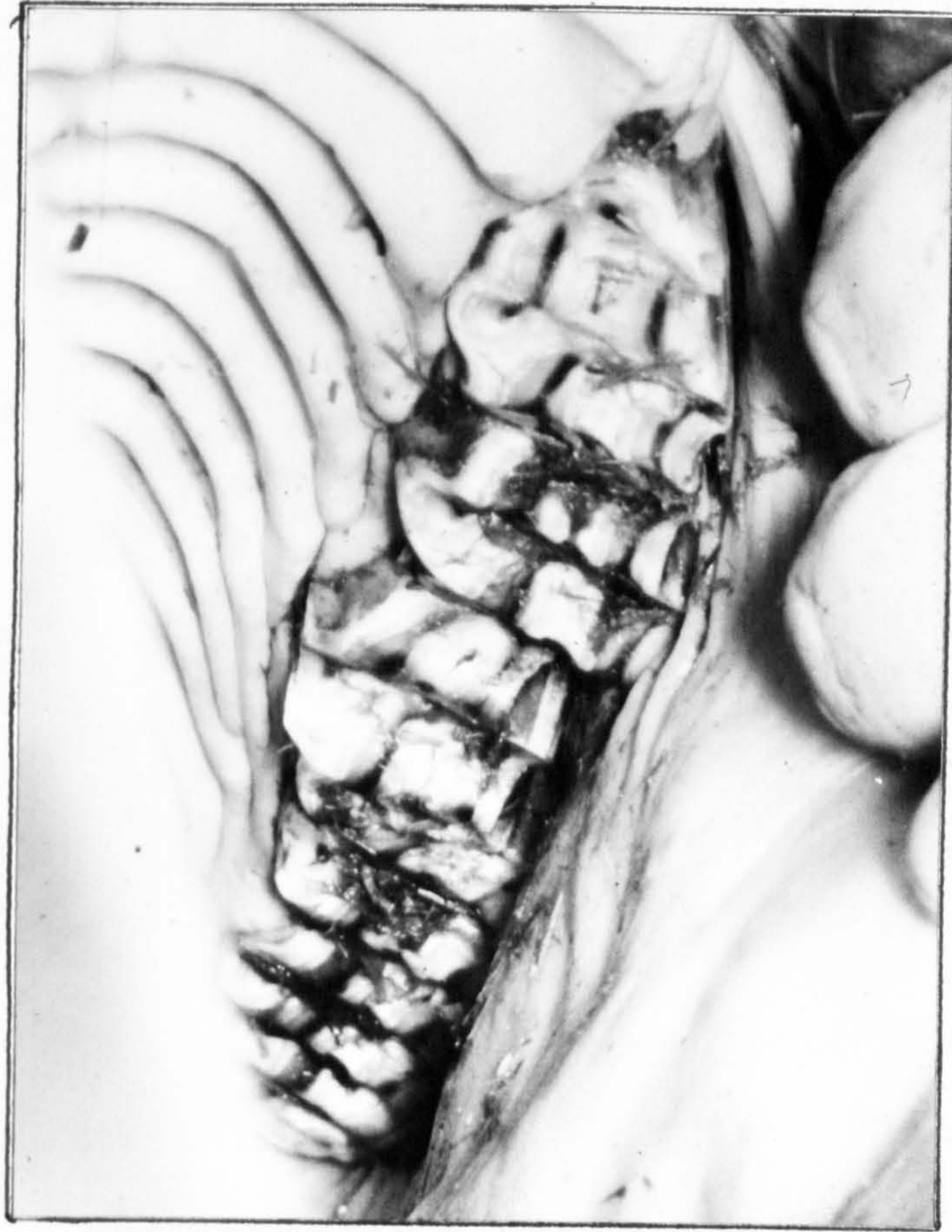


FIGURE 71: Palatal displacement of the third cheek tooth.



FIGURE 72a: One day old Thoroughbred with multilocular maxillary cyst.



FIGURE 72b: Radiograph of 72a with displacement of cheek teeth.

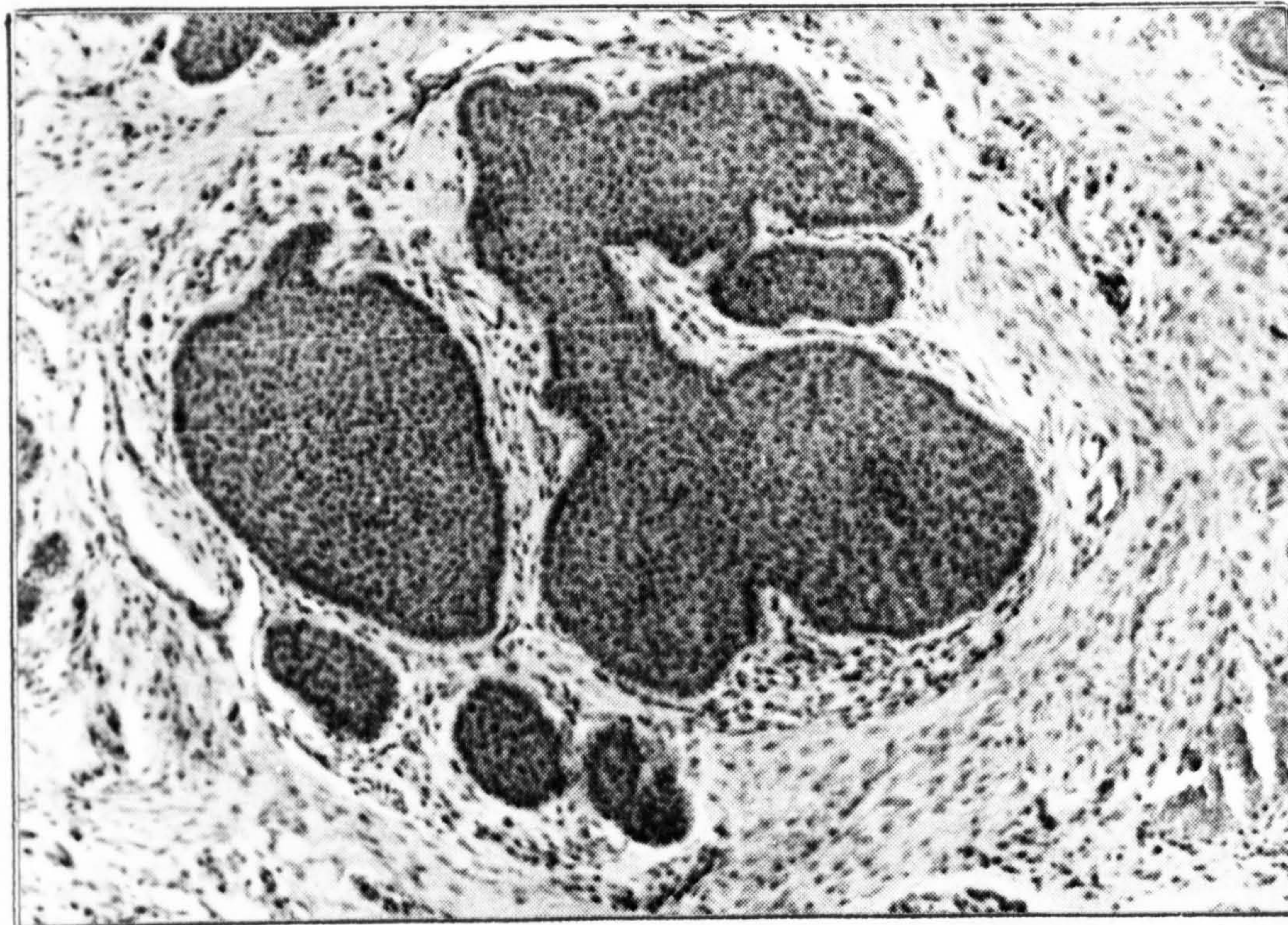


FIGURE 73a: Ameloblastic odontoma in a nine month old Shetland pony. Epithelial and connective tissue tumour elements. H and E x 110.

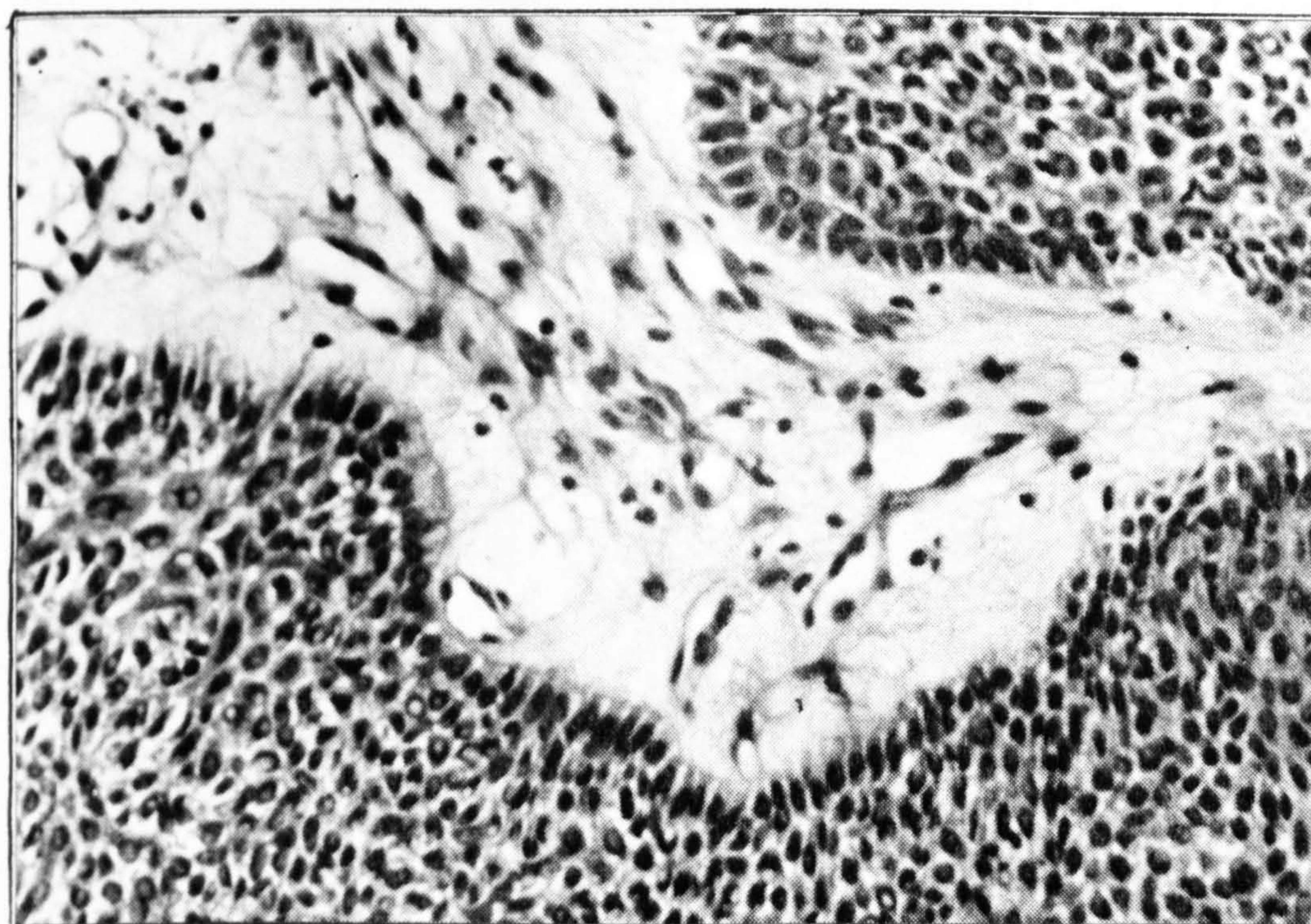


FIGURE 73b: Epithelial elements resembling ameloblasts. H and E x 250.

organ. There was a stroma of fibrous tissue and calcified dentine.

A single temporal teratoma (aural cyst, dentigerous cyst, dental teratoma) was seen in an eight month old Thoroughbred colt foal. The lesion measured 7 x 5 x 4 cm and discharged a sterile mucoid fluid from the caudal margin of the ear. The tumour was explored and a molar tooth-like structure, measuring 2 x 1.5 x 1.5 cm, was excised from the temporal bone (Figures 74a and 74b).

Category 8: Miscellaneous

Table 11 lists the cases examined in this category.

a) Campylorrhinus lateralis Major facio-maxillary reconstruction was undertaken in a six month old Cleveland Bay colt foal. The nasal bones and septum were sectioned and the maxilla divided submucosally in a vertical plane rostral to the first cheek tooth. Steinmann pins supported by a fibre glass cast were used to hold the maxilla in its corrected alignment (Figures 75a and 75b). The foal was nourished by an indwelling oesophagostomy tube, but died 14 days after surgery following a peracute episode of colic. Post-mortem examination revealed gross tympany of the small intestine. The second case in an Arab filly foal was untreated.

b) Labial Tumours and c) Granulomata The diagnosis of melanomata was based on their occurrence in grey animals and the pigmented appearance of the tumour tissue. No biopsies were taken and no treatment given. Contact was maintained with only one case, a nine year old Highland pony with a diffuse plaque of melanotic tumour originating at the commissure of the mouth on



FIGURE 74a: Temporal teratoma in an eight month old Thoroughbred colt.

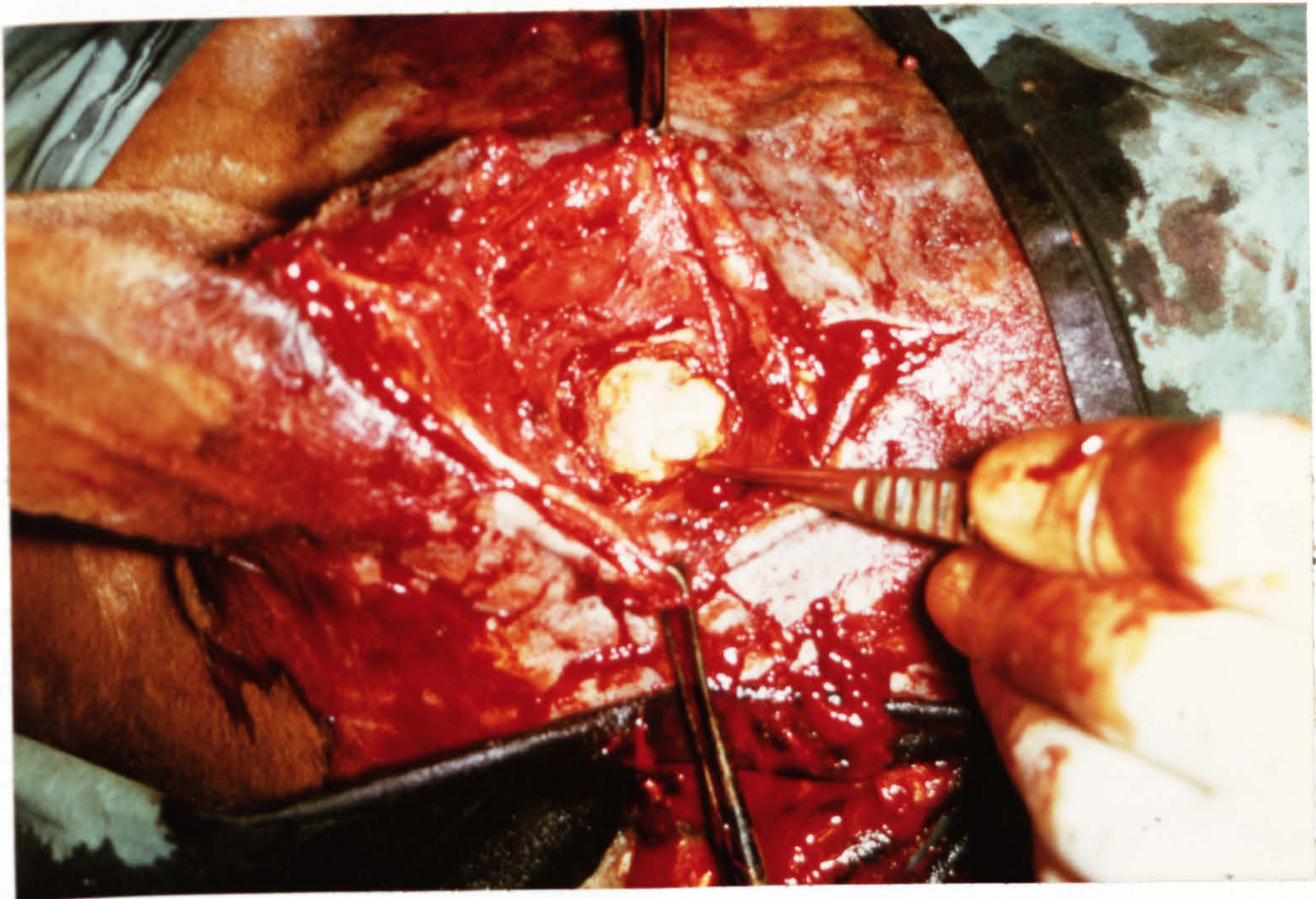


FIGURE 74b: Surgical exposure of temporal tooth and aural fistula.

		<u>No. of Cases</u>
a)	<u>Campylorrhinus lateralis</u>	2
b)	Labial tumours Melanomata	4
	Mast cell tumour	1
c)	Granulomata Actinobacillosis	2
	Inflammatory	2
d)	Fractures Incisor area	6
	Mandible	3
	Maxilla	2
	TOTAL	— 22 —

TABLE 11 Clinical Dental Disease:
Category 7, Miscellaneous Diagnoses

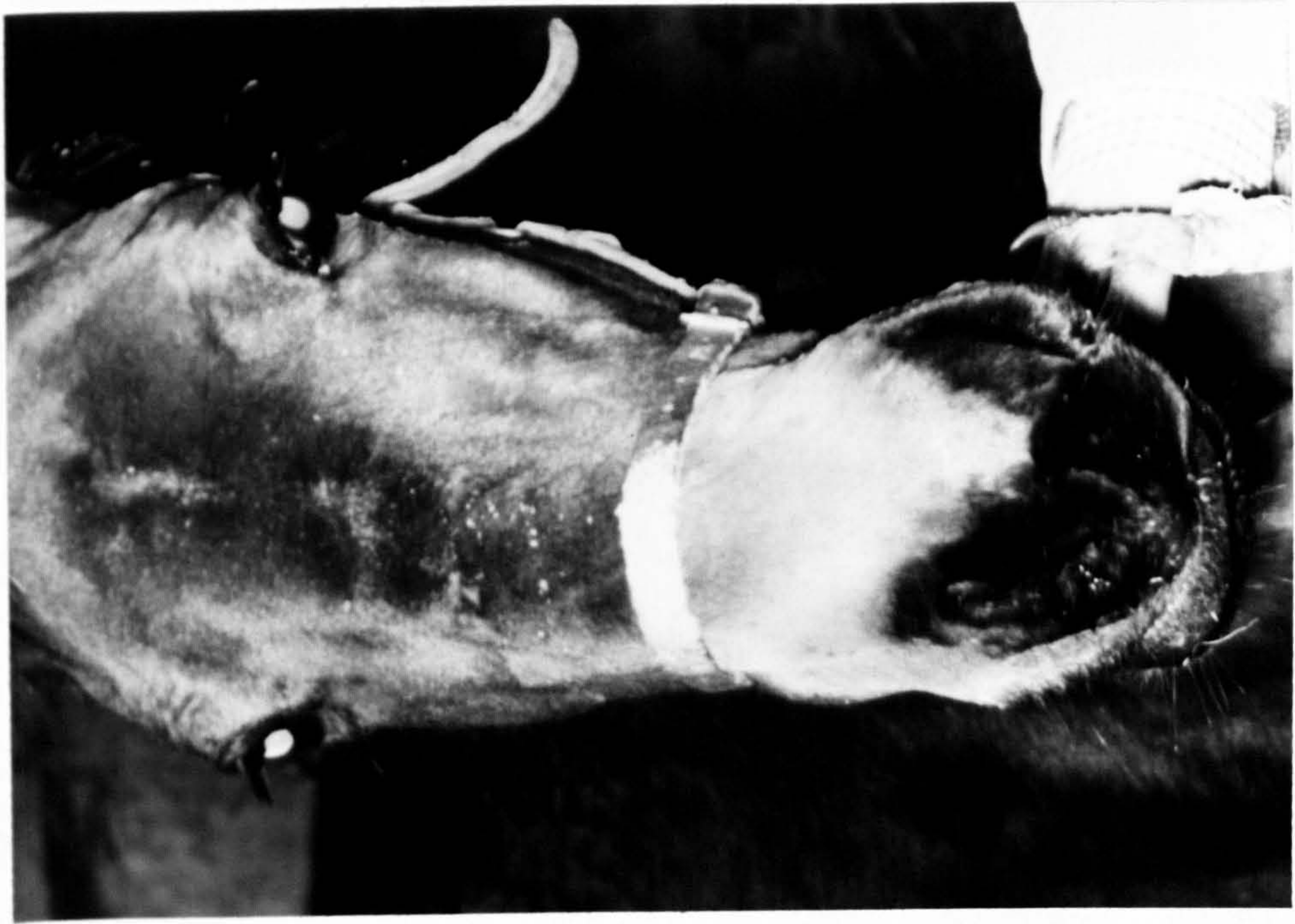


FIGURE 75a: *Campylorhynchus lateralis* in a six month old Cleveland Bay colt.

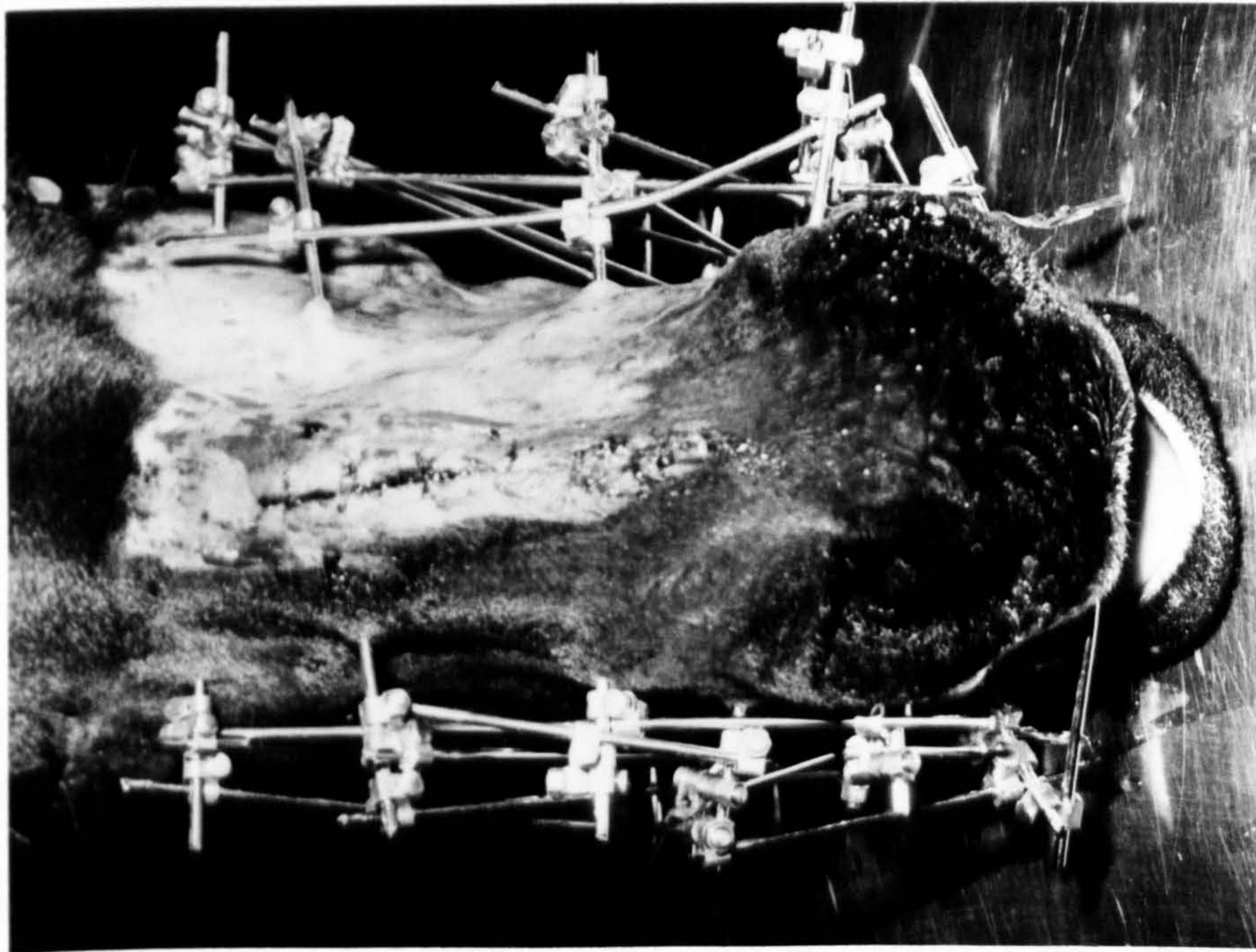


FIGURE 75b: Post operative appearance.

the left side and extending both rostrally into the chin and caudally into the cheek. The tumour mass prevented normal bit contact and the animal was destroyed after nine months due to local growth. A post-mortem examination was not carried out.

Excision and/or excision and drainage of the other lesions, together with antibiotic therapy, was effective.

d) Fractures Of eleven fracture cases two animals were destroyed on humane grounds and nine were treated by conventional osteosynthetic techniques using wires and short steinmann pins.

DISCUSSION

Most of the cases reviewed in this chapter were referred to the author by veterinarians in general practice. Consequently the classification of cases seen was inevitably biased towards the more complicated or difficult dental problems. In general practice dental prophylaxis (i.e. routine rasping of dental arcades, etc.) is by far the commonest task of the practitioner (B.E.V.A. survey 1965). In this chapter categories 1 (bitting and riding problems), 2 (general anaesthesia for dental prophylaxis), 3 (major irregularities of arcades), 5 (delayed eruptions) and 6 (palatal displacement of third cheek tooth) could be included under the broad subject heading of dental care. Some of the cases illustrated the extreme forms of irregularity of wear that occur with years of dental neglect. Such cases formed the material from which earlier authors (Little, 1913; Harvey, 1920; Merillat, 1921; Pillers, 1933) suggested that many systemic disease problems stemmed from oral disease. Pillers (1933) quotes Cadiot and Dollar as stating that chronic dental affections in the horse and

all large animals favoured obstruction of the large intestine and may bring about impaction of the caecum. Leue (1941) established a correlation between dental diseases and digestive disorders in some of the horses he studied. However, in this series no evidence was found to support Cadiot and Dollar, although six animals were destroyed because of starvation resulting from gross malocclusion. Horse owners, riders and trainers frequently complain of biting and riding problems that are attributed to enamel points, dental eruptions, maleruptions, retention of deciduous caps and wolf teeth. In many cases, however, it has not been shown that improvement in performance stemmed from treatment of a specific dental problem - with the exception perhaps of misplaced wolf teeth and retained deciduous caps that rotate and severely ulcerate the buccal mucosa causing painful bit contacts. Such problems occur in younger horses at a time when they are likely to be undergoing schooling or re-schooling. The author feels, from personal daily observation of the feeding and riding behaviour of a small number of horses, that good bit contact is hindered by sharp points and such horses tend to be messy feeders. In these cases there was an immediate improvement after dental treatment.

The clinical signs of apical infections have been described and the relationship between a specific symptom and the anatomical position of the decayed tooth has been confirmed e.g. dental fistulae were more commonly associated with mandibular apical infections. There were 24 cases of maxillary sinus empyema, which was 40% of all cases of maxillary dental disease.

In many cases the connection between caries of the central

cementum and pulpitis was clearly shown in the maxillary teeth. In the mandibular cheek teeth no clear aetio-pathogenesis was established for apical infection and dental decay. There was a marked difference in the mean age of the horses with diseases of the mandibular arcade (mean 6 years) compared with those involving the maxillary arcade (mean 10 years). This might suggest that the origin of mandibular infections was a delayed eruption and subsequent degeneration of the eruption cyst described by Eisenmenger (1959) as alveolar periostitis in young horses (Figure 4 and Figure 66). In both the upper and lower jaw the majority of infected teeth were rostral in position, with 63 of the 79 infected mandibular teeth being either the second or third tooth. It could be implied that this is further evidence to support the theory that delayed eruption or overcrowding is of primary aetiological importance in the development of mandibular apical infections as these are the teeth most commonly seen with large "eruption cysts". In the upper jaw no apical infections of the two caudal cheek teeth were seen. Descriptions of the particular techniques (using a wry-necked punch) necessary to repulse either the fifth or sixth cheek tooth have been recorded (Cook, 1965), but few such cases have been listed. Hofmeyr (1960) was of the opinion that the fourth cheek tooth was more frequently diseased because it erupted earlier than the other cheek teeth (at 10-12 months of age) and consequently was subjected to greater trauma. Of the maxillary tooth root infections described here the third tooth was more frequently diseased than was the fourth (Table 10), and it could again be suggested that delayed eruption and subsequent apical osteitis might be important in the pathogenesis of maxillary dental decay.

In this series all dental extractions were carried out under general anaesthesia and the clinical importance of radiography, both pre-operatively as an aid to diagnosis and post-operatively to check on root and socket sequestra, has been emphasised. Section of a number of these root nodules proved that they were discreet nodules of cementum presumably forming as a protective mechanism. Similarly secondary dentine was found lining diseased pulp cavities.

Dental wax as a plug for the alveolus was found to be more effective and easier to use than was gutta percha. In some cases as the trephine site was irrigated daily, partial displacement of the wax plug and loss of its integrity as a protective barrier was noted. In these cases the animal was re-anaesthetised and the plug replaced. It was also noted that despite thorough alveolar curettage up to 20 per cent of horses required further socket surgery after a period of up to 15 months in some cases.

CONCLUSIONS

In a referral equine clinic the dental problems most frequently encountered were the result of apical infections, pulpitis and dental decay. During the treatment of 135 cases of apical infection, 123 diseased teeth were extracted. The clinical signs associated with dental disease in the horse may be summarised as follows:

- 1) delayed eruption or maleruption
- 2) irregularities of wear leading to malocclusion and quidding
- 3) apical infections associated with dental fistulae or sinus empyema and facial swelling
- 4) congenital or neonatal dental tumour development.

GENERAL DISCUSSION AND CONCLUSIONS

The early Eocene ancestor of Equus caballus was only 26 inches in height and had simple teeth. An animal twice this height (52 inches or 13 hands) is not twice but is approximately eight times heavier and therefore needs about eight times as much food to maintain this body weight. As the environment changed to provide food of the appropriate quality and quantity, there were corresponding modifications of tooth structure involving the processes of molarization, reserve crown formation and continuous eruption with the development of crown cementum. These changes allowed the horse to become a grazing rather than a browsing animal and to grow up to 60 inches high in the wild. There is evidence that dental evolution is still continuing in the domestic horse as shown by the regression of the first premolar (or wolf) tooth. Only 13 per cent of specimens had wolf teeth in the dental disease survey and comment was made that this low incidence could have been the result of previous dentistry, since the routine extraction of wolf teeth is a common practice. However, of the Thoroughbred foetal skulls examined radiographically only 20 per cent contained wolf tooth anlagen whereas an incidence of almost 100 per cent was seen in the adult skulls of the Plains Zebra (Cook, W.R., 1974, personal communication). In this species it was not uncommon to find that

the maxillary wolf teeth were large enough to make occlusal contact with the first mandibular cheek tooth (PM2) and were therefore still functional, but had often been lost by 18 years of age.

It has been suggested (Little 1913; Harvey, 1920; Merillat, 1921; Colyer, 1931, Shuttleworth, 1948, Hofmeyr, 1960) that domestication, with the development of chaff, grain and bran feeding of working horses, was responsible for the occurrence of many severe forms of primary dental and secondary systemic diseases. Although there have been further changes in management practices in the past few decades, the results of the dental survey presented here highlight the importance of periodontal disease just as did Colyer's survey of draught horses in London in 1931. Any factors interfering with normal occlusion have been shown to result in degeneration of the gums and periodontal structures. Therefore, irregularities of wear of the dental arcades with the formation of enamel points and buccal ulcerations should be routinely corrected to prevent stagnation of food and the initiation of the inflammatory process that is the basis of periodontal disease in the horse. Both Harvey (1920) and Baker (1970) noted a peak in the incidence of periodontal disease during the period of eruption of the permanent dentition with a subsequent amelioration of the condition (Figure 23).

The results presented in Chapter II (cementogenesis), Chapter III (dental disease survey) and Chapter V (clinical dental disease) confirm that the disease, previously described as caries (Colyer, 1931; Hofmeyr, 1960) is a unique and very common disease of the maxillary cheek teeth of the horse. In agreement with Joest (1926) the author has concluded that the

condition is best described as caries of cementum. Epidemiological studies in man are aimed at introducing methods of controlling mouth acids or strengthening enamel but prevention along these lines is unlikely to be feasible in the horse. It has been shown that hypoplasia of the central cementum is a very important factor in the high incidence of the disease. The cheek teeth of the Plains Zebra show central cement lake lesions identical to those of the domestic horse (Cook, W.R., 1974, personal communication). In Equus caballus all the developing upper cheek teeth examined contained immature or hypoplastic cementum in the depths of the enamel invagination. Hofmeyr (1960) suggested that the fourth cheek tooth (M1) was more severely effected because of its early eruption and its subsequent exposure to a longer period of contact with fermenting foodstuffs. This appears to be unlikely and it is suggested that its early eruption in fact enhances the degree of hypoplasia of cementum present and thus predisposes the fourth tooth to a more severe lesion. Since it has been seen that the formation of cementum is incomplete at the time of eruption of the maxillary cheek teeth and that occlusal contact breaks the gubernacular blood supply thereby inhibiting further cementogenesis within the enamel invaginations.

Fortunately the caries lesion is usually benign in that it is confined to the cementum of the enamel invagination but expansion of the lesion may lead to pulpitis. It is thought that such expansion is probably due to the action of cementolytic acids produced by the fermentation of foodstuffs but this hypothesis has not been proved in these studies.

In the cases of clinical dental disease apical and periapical infections were found in almost equal numbers in upper (60) and lower jaws (75). Support is given to the observations of Paine (1909), Eisenmenger (1959) and Hofmeyr (1960) that many cases of so-called alveolar periostitis in young horses are the result of retarded eruption and subsequent cystic periapical degeneration. Although Cook (1965) classified such infections into three clinical categories i.e. mandibular fistula, maxillary osteitis and maxillary sinus empyema, and the cases described here could also be sub-divided into these categories, it has been shown quite clearly that the pathology of the periapical reaction was the same in each class. Periapical changes associated with impaction consist of sterile granulomata which open into fistula, develop secondary infections and spread to a pyogenic pulpitis and tooth necrosis.

In some periapical reactions cementogenesis was stimulated, resulting in the formation of nodules of cementum. In continuously erupting teeth the cementoblast layer of the root and reserve crown remains active to facilitate the rearrangement attachment of the fibres of the periodontal ligament. A modification of this process was responsible for the formation of the nodules described above and is assumed to be a protective mechanism, as is the formation of secondary dentine in cases of pulpitis.

Continuous eruption of the reserve crown balances the loss of arcade as a result of the forces of wear. The reserve crown is tapered to the root and eruption is facilitated by the deposition of new cementum and the incorporation within it of periodontal fibres linking the alveolar bone and cementum.

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