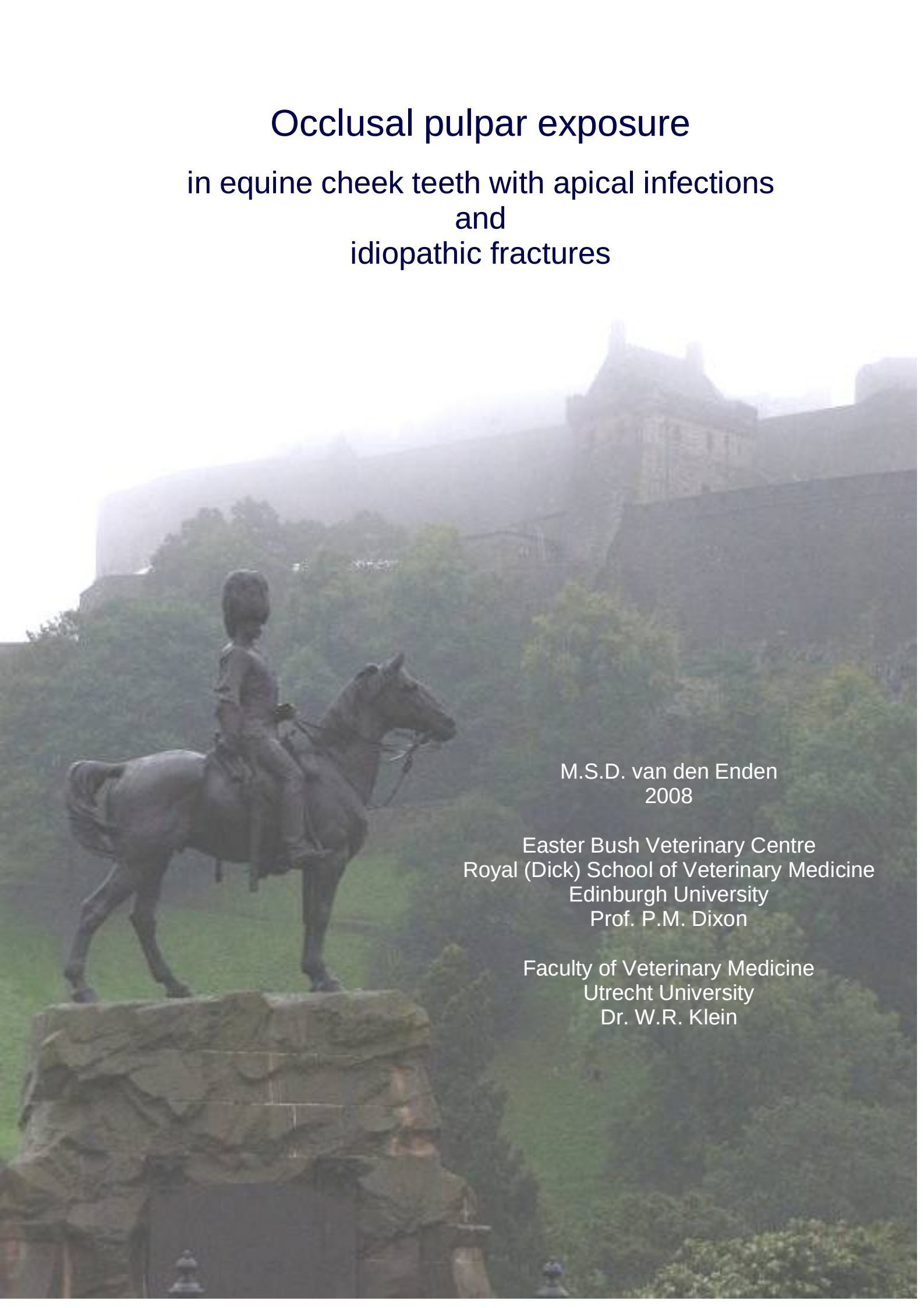
Occlusal pulpar exposure

in equine cheek teeth with apical infections and idiopathic fractures



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Summary

Hundred and ten cheek teeth that were extracted (1993 - 2008) because of apical infection (n=79; mean dental age 3.5 years) or idiopathic cheek teeth fractures (n= 31; median dental age 8.5 years) were examined, including analyses of transverse and longitudinal sections and CAT scan images of these teeth. Computerised axial tomography was useful to determine pulp chamber anatomy and could identify pulp horns suspicious of pulpitis and exposure. The apical infections were mainly (68%) due to anachoresis (blood or lymph born infection), with the residual cases caused by descending periodontal disease (23%), infundibular caries (4%), fissure fractures (3%) and dysplasia (3%). The idiopathic fracture patterns were similar to previously described patterns. Occlusal pulpar exposure was found in 32% of apically infected cheek teeth, including exposure of multiple pulps in 27% and a single pulp in 5%. This finding re-enforces the value of detailed intra-oral examination of suspect apical infection cases. However, 10% of apically infected cheek teeth had changes to the occlusal secondary dentine termed occlusal pitting, but did not have exposure of the underlying pulp. The term occlusal defect would therefore be more appropriate to use in live patients. Multiple pulpar exposures occurred in some cheek teeth with apical infections, and the pulp involvement reflects the anatomic relationships of these pulps. A higher proportion (42%) of cheek teeth extracted because idiopathic fractures had pulpar exposure (26% multiple, 16% single pulps), especially with midline sagittal maxillary and miscellaneous pattern mandibular cheek teeth fractures, but only 3% had occlusal pitting.

1. Introduction

Recent years, equine dentistry has become an important field in veterinary medicine. Scientific research and education in this area expanded, which has resulted in advanced therapeutic and prophylactic dental care (4). Increasing knowledge and experience in equine dentistry allows identification of disorders that were not fully appreciated before. Several authors have demonstrated a causal relation between apical infections and pulpar exposure in equine cheek teeth (3;5). Furthermore, recent research has suggested that some cheek teeth with idiopathic fractures develop apical infections (6-8).

These findings call for further investigation into the prevalence of occlusal pulpar exposure in cheek teeth with apical infections and idiopathic fractures. As an introduction to these research objectives, equine dental anatomy is discussed followed by the pathophysiologies of pulpar injury, pulpar exposure, apical infections and idiopathic fractures.

1.1 Morphology of equine dentition

Horses evolved from browsing herbivores in the Eocene era to a grazing species: ‘Equus caballus’ (9). The deciduous equine dentition nowadays consists of three incisors and four premolars in each quadrant. Permanent dentition counts three incisors, one canine, four premolars and three molars in every quadrant. According to the Modified Triadan Nomenclature, the teeth are indicated with three digits. The first digit represents the quadrant, the second and third indicate the position of the tooth. In a clockwise direction, the right maxillary quadrant is labelled quadrant 1, the left 2, the left hemimandible 3 and the right hemimandible 4. In deciduous dentition the quadrants are indicated with 5 to 8. The central incisor is labelled 1, and all dental elements are subsequently labelled in a caudal direction. The right maxillary permanent cheek teeth row for example, is indicated with 106 to 111. Supernumerary cheek teeth are labelled 12, 13 et cetera (10).

These modern-day horses normally grind abrasive foodstuffs for up to 18 hours per day (11) and as a consequence the cheek teeth have a high degree of wear at their occlusal surface. The prolonged eruption and the hypsodont (long crowned) nature of these teeth compensate for

this occlusal wear, in contrast to the brachydont (short crowned) teeth of their ancestors. Although prolonged, the eruption of equine teeth ceases after 20 to 25 years and tooth growth also has a limited timescale (anelodontia) (9;12;13).

Cheek teeth in particular have adapted to the horses' fibrous diet. The first premolar, the wolf tooth, is rudimentary if present. The second, third and fourth premolar are similar to the three molars in anatomy and function due to molarisation. These teeth are therefore collectively termed the cheek teeth, which sit in close approximation of each other in a long cheek teeth row. These six teeth act as a single battery in grinding fibrous foodstuffs, maximising the masticatory surface (13).

The gross and ultrastructural characteristics of dental tissues in equine cheek teeth represent another dietary adaptation. Dental tissues include enamel, cementum, dentine and pulp (Fig. 1). Pulp lies in the centre of the tooth and is surrounded by secondary and primary dentine. Enamel is essentially draped over these, with infoldings in a transverse plane. In the maxillary cheek teeth two additional longitudinal cup-like enamel invaginations comprise the rostral and caudal infundibula. Cementum covers the periphery of the tooth and also fills the infundibula (13;14).

Pulpar connective tissue provides sensory innervation to the teeth through a branch of the trigeminal nerve and autonomous fibres. It is rich in lymphatics and vasculature to provide for the metabolically active odontoblasts (13;15). The odontoblastic cell layer resides at the periphery of the pulp, embedded in its secretory product: the developing predentine. Dentine is later formed as the organic matrix of predentine becomes mineralised as a tubular structure (13). During this continuous process of dentine deposition, the odontoblasts gradually retract in dentinal tubules they created and migrate centrally in the narrowing pulp chamber (16).

Primary dentine is formed prior to tooth eruption and secondary dentine formation by odontoblasts follows after the tooth comes into occlusion and continues throughout the life of the tooth (17). While prolonged eruption of the tooth normally compensates for wear, the progressive occlusion of the pulp chambers with secondary dentine ensures the pulp remains protected from the oral environment at the occlusal surface (11;13;18).

Secondary dentine can be further subdivided into regular and irregular dentine. Regular secondary dentine is usually used to describe physiological secondary dentine. The terms irregular secondary dentine, tertiary dentine, reactive and reparative dentine have interchangeably been used as pathological types of dentine (15;19;20). General consensus is that tertiary dentine is formed as reaction to a dental insult (16;21;22). It can be further distinguished as reactive dentine produced by odontoblasts and reparative dentine synthesised by mesenchymal pulpar cells (6;17). Recently however, it has been proposed that irregular secondary dentine should be used to describe a physiological odontoblast deposition. Irregular secondary dentine is widespread in all equine teeth and thought to be synthesised by odontoblasts to occlude the central part of pulp horns. Both regular and irregular secondary dentine would contribute to the physiological dentine layer occluding the pulp chamber (23).

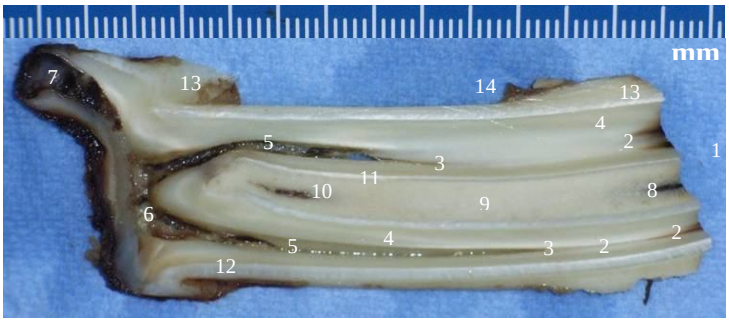


Figure 1: A transversely sectioned maxillary cheek tooth. Smallest bars on scale represent millimetres.

1. *The angulated and irregular occlusal surface. Notice the interchanging dental tissues.*
2. *Secondary dentine over a pulp horn. The thickness of this layer varies between the two visible pulp horns.*
3. *Pulp is therefore present at different levels from the occlusal surface.*
4. *Primary dentine*
5. *Individual pulp horns*
6. *Common pulp chamber*
7. *Apical foramen*
8. *Central vascular channel*
9. *Infundibular cementum*
10. *(Remnant of) vascular supply to infundibulum*
11. *Infundibular enamel*
12. *Peripheral enamel*
13. *Peripheral cementum*
14. *Periodontal membrane*

Enamel is most resilient to wear but brittle, since it is a highly mineralised acellular structure. Enamel folds are therefore supported by the somewhat elastic primary dentine and the more flexible cementum that act as shock absorbers (13). Secondary dentine is softest of the dental tissues because it largely consists of less mineralised intertubular dentine (24;25). It has therefore the highest degree of wear and greatly absorbs food pigments (26).

The combination of these mineralised structures makes the teeth hard enough to withstand high occlusal wear but elastic enough to take large masticatory forces without fracturing. It

also creates a perfect grinding surface. Cementum and enamel covering the occlusal part of cheek teeth is worn away when opposing teeth come into occlusion after eruption. This creates the functional, so called secondary occlusal surface with alternating dental tissues. Undulations and ridges develop due to different rates of wear between the dental tissues exposed at the occlusal surface, with the harder enamel ridges projecting above the softer and thus more worn cementum and dentine (Fig. 1 and 2) (13;27).

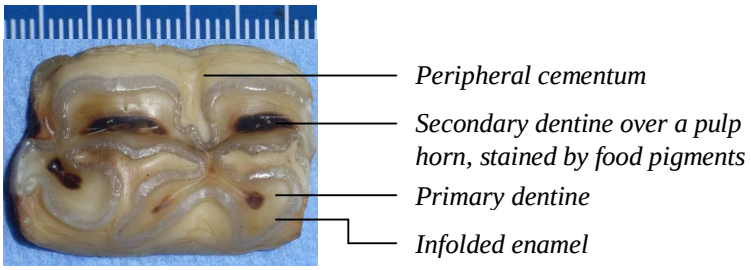


Figure 2.: Transverse occlusal section of a mandibular cheek tooth showing the occlusal surface.

1.2 Endodontic and apical anatomy

Young equine cheek teeth have large open apices without true roots (enamel-free areas). These apices continue to develop: one to two years after eruption true roots have formed due to apical cementum and dentine deposition (13); in older cheek teeth the apical foramina are small and roots have become constricted (28). At eruption, the cheek teeth have a common pulp chamber continuous with all pulp horns (also termed individual pulp chambers). Pulp chambers house the live, pulpar connective tissue which communicates with the periodontium through the apical foramina (13).

Five individual pulp horns are present in Triadan positions 07 to 10, whereas additional pulp chambers can be identified in the first and last cheek tooth of all quadrants (Fig. 3) (17;23). The pulp nomenclature system proposed by du Toit et al. (29) is used in this text to refer to pulp horns. Numbering begins at the rostro-buccal pulp horn in both maxillary and mandibular cheek teeth. Corresponding numbers in maxillary and mandibular cheek teeth represent similar anatomical positions at the occlusal surface and this allows comparison between pulp chambers in all quadrants.

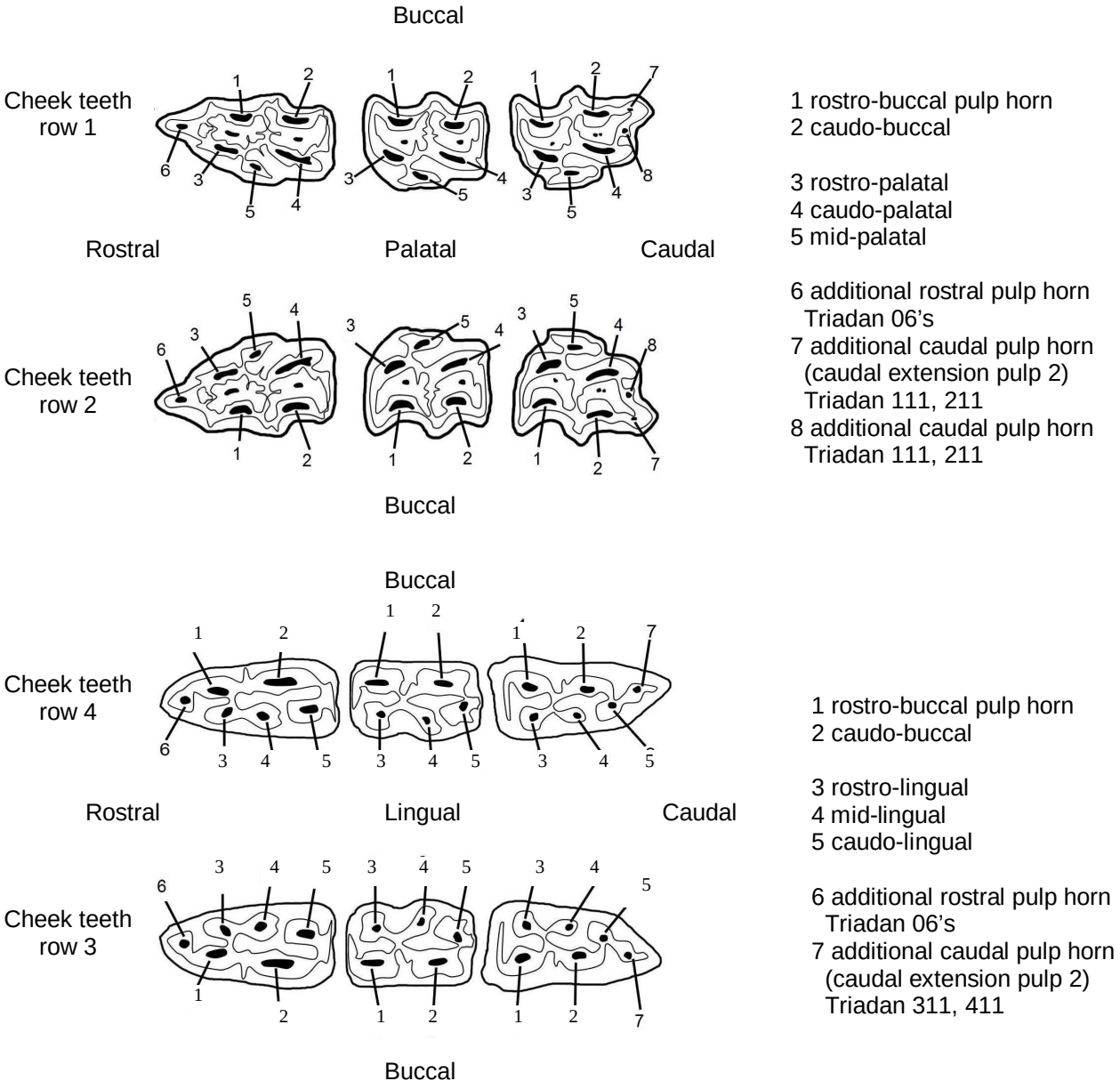


Figure 3. Pulp nomenclature system described by du Toit et al. (29) (illustration modified from Dacre et al. (23), with permission)

As a result of the ongoing circumferential replacement of vital pulp by secondary dentine, the common pulp chamber and individual pulp horns gradually become smaller and narrower as the tooth matures (30). The additional caudal pulp horns 7 and 8 can therefore only be identified on the occlusal surface of older Triadan 11's when progressive secondary dentine deposition separates pulp horns and the tooth is erupted and worn down to that level (23). Furthermore, communications between pulp horns are more frequently present in young lower cheek teeth than in young upper cheek teeth, which may indicate a more complex endodontic

structure in younger mandibular cheek teeth compared to maxillary ones. Maxillary pulps 3 and 5, and mandibular pulps 2 and 5 are reported to communicate most frequently (23).

1.3 Pulpar response to insults

Acute pulpar injury such as infiltration of molecules from the oral environment, pH changes and bacterial invasion, is followed by an inflammatory response. Compared to brachydont pulp cavities, the more spacious hypsodont pulp chambers and wider root canals can presumably better accommodate pulpar hyperemia and edema by allowing continued arterial and venous blood flow. Collateral circulation however is absent in pulp and inflamed pulp is still confined to a rigid chamber. As pulpar hydrostatic pressure rises, pulpar ischaemia can occur and lead to pulpar necrosis (Fig. 4). In the presence of (bacterial) pulpitis, odontoblasts are affected and secondary dentine deposition will decrease or in case the odontoblastic layer is no longer vital it will cease completely. The pulp chamber will not be occluded any further (15;31;32).

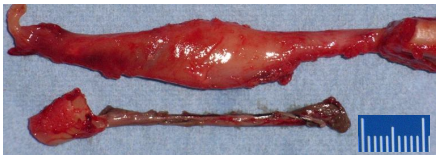


Figure 4: Grossly healthy (top) and diseased pulp (bottom), just after extraction of a fractured Triadan 308 (also pictured in Fig. 27). The diseased pulpar tissue has a dull, purple appearance with sparse acute hemorrhage. The pulp shows degeneration, compared to the well vasculated healthy pulp.

Surviving pulp however, is often capable of shielding itself from devitalised areas in several ways (Figs. 7, 14). Focal ischaemia of odontoblasts or a low grade pulpitis stimulate surviving odontoblasts to lay down reactive dentine and mesenchymal pulpar cells can differentiate into reparative dentine producing cells (6;17). Primary dentine can become sclerotic, which impedes the invasion of bacteria and molecules from the occlusal surface into dentinal tubules (13). Moreover, odontoblast processes and fluid movement in healthy dentinal tubules are thought to prevent the advance of bacteria in those tubules (16). Periapical tissues finally are able to develop fibrosis and granuloma to seal off the infected area (6).

Microorganisms are associated with pulpar and apical infection, but the primary causal agents are yet to be determined (33). Mixed cultures are often isolated and both aerobic and anaerobic bacteria are identified. Bienert (34) recorded dominance of *Fusobacterium spp*. and *Prevotella spp*. in infected pulp, periapical tissues and sinuses of twenty horses. Mueller and

Lowder (15) also suggested that gram negative aerobic and anaerobic bacteria are involved frequently, and *Bacteroides fragilis* in particular.

1.4 Definition of apical infection, occlusal pulpar exposure and occlusal pitting

The term *apical infection* is used to describe infection of tissues at the distal, enamel-free area of a tooth: the apex. In hypsodont dentition this term is more relevant than 'tooth root infection', because it can appropriately describe infection of young and sometimes even unerupted cheek teeth, where true roots have not developed yet. The infection can affect pulpar tissue, dentine, cementum, periodontal ligaments, alveolar bone and spread to adjacent bone and apices. It can be present as an abscess or drain via the periodontium, an oro-nasal fistula, the paranasal sinuses or the ventral mandibular cortex. Since this pathology can be present in various extent, it is not surprisingly referred to by many terms. Apical infection, apical abscess, peri-apical abscess, tooth root abscess, peri-radicular infection, dento-alveolar infection and dental sepsis all have been used (5;35;36).

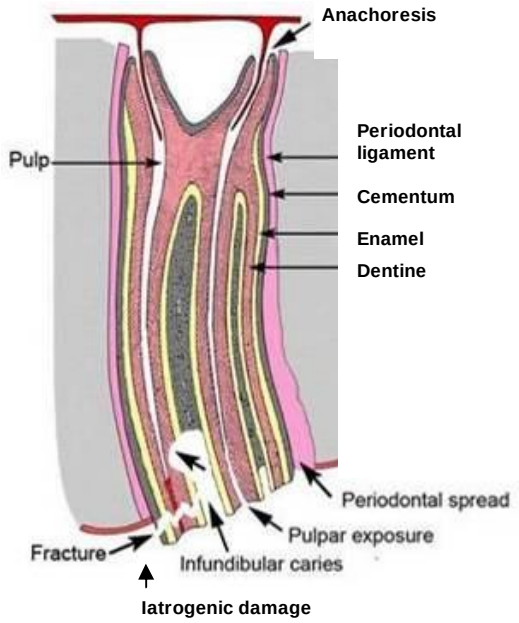
Occlusal pulpar exposure is defined as a defect in the secondary dentine over a pulp horn at the tooth's occlusal surface. Consequently, the pulp communicates with the oral environment through the un-occluded pulp horn (Fig. 6b). When the occlusal defect only extends a small distance into the tooth, the pulp is still sealed off from the oral environment. This is referred to as *occlusal pitting of secondary dentine* (Fig. 6a) (37;38).

1.5 Aetiopathogeneses of apical infection and occlusal pulpar exposure

As explained earlier, odontoblasts lay down secondary dentine over the occlusal aspects of the pulp horns. When the layer of secondary dentine on the occlusal surface is incomplete, molecules and bacteria from the oral environment can travel from this exposed pulp chamber down through vital pulp towards the apex of the cheek tooth. Thus pulpitis and apical infection is directly established through *primary occlusal pulpar exposure* (3;5).

Alternatively, any local or general insult to vital pulp and its odontoblasts could result in a decrease or cessation in secondary dentine formation. Subsequently, dentinal thickness in the

affected pulp chambers will be reduced (39). As the cheek tooth is progressively worn away at its occlusal surface, the pulp chamber containing devitalised or dead pulp is ultimately exposed. Hence, such occlusal pulpar exposures develop *secondary* (indirectly) to an apical infection (3;5;40).



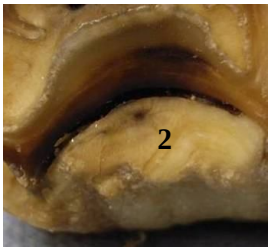
Several routes of apical infections in equine cheek teeth have been proposed that can lead to pulpar exposure (Fig. 5). These include anachoresis, periodontal disease, maxillary infundibular cemental caries, iatrogenic damage and fractures. It has also been proposed that dental dysplasia and an imbalance between attrition and dentine deposition predisposes cheek teeth to apical infections (1-3). These various infection routes will be reviewed in the next paragraphs.

Figure 5: Infection routes.

One pathophysiology where apical infection can result in secondary pulpar exposure is *anachoresis*. The term anachoresis is used to describe the haematogenous or lymphatic spread of bacteria to apical pulp (Fig. 5). If this infection progresses from the periapical tissues to the pulp, it will compromise the odontoblasts, which in time can lead to secondary occlusal pulpar exposure. Anachoretic pulpitis is known in other species as a frequent infection route and is regarded as an important aetiological factor of apical infections in horses (3;5;41). Becker (1962) recorded the presence of opened pulp chambers on the occlusal surface and termed this 'Porodontie.' He already proposed a haematogenous infection route as cause for cessation of secondary dentine deposition and pulp chambers to be opened 'spontaneously.' 'Spontaneous' in the sense that there is no physical access to the pulp chamber as is the case with fractures or caries. Recently, Dacre (33;37) reported anachoresis was the most likely cause of apical infection in 62% of examined maxillary and 59% of mandibular equine cheek teeth. This route of bacterial invasion has not been histologically proven thus far, but in cases

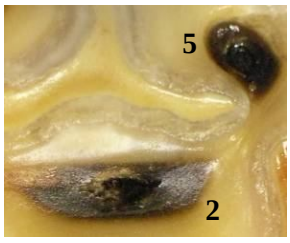
where physical access from the oral cavity to the pulp is absent, anachoresis seems the most likely aetiology (Figs. 12, 13, 15, 16, 22, 23).

Descending periodontal disease is another identified cause of apical infections in cheek teeth. Periodontal disease in horses develops secondary to diastemata, abnormal wear patterns and fractured, displaced or supernumerary cheek teeth (42). The condition is at the onset characterised by gingivitis, but with continued inflammation deeper periodontal structures are eroded. A periodontal pocket then forms in which feed is impacted and bacteria rapidly multiply, causing deeper inflammation in a self- propagating fashion. The inflammation progresses towards the tooth apex and in certain teeth, infection spreads via the periapical tissues to pulp. This pathophysiology can ultimately lead to occlusal pulpar exposure (11;15;43). Hypsodont periodontal ligaments are continuously reformed in the eruption process and so regeneration is possible. Periodontal disease (uncomplicated by apical abscessation) can therefore be resolved in horses, in shear contrast to the condition in brachydont species (44).



a

Figure 6: Occlusal defects in Triadan 107, dental age 4 y (Fig. a) and Triadan 307, 11 y (Fig. b). Occlusal pitting (a) could not be distinguished from occlusal pulpar exposure (b) through inspection of the occlusal surface; only after sectioning could the extent of the defects be assessed. Numbers indicate pulp horns.

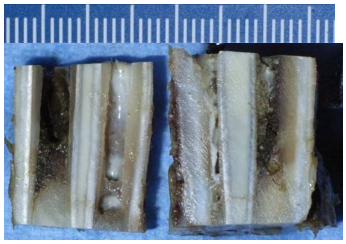


b

Apical infection can also be attributed to *dental caries* in some cases. Caries is a process of demineralisation of inorganic dental components and dissolution of organic dental components (17;45). Infundibular cemental hypoplasia (Fig. 16) predisposes to the development of infundibular cemental caries, because this is an ideal environment for feed to accumulate and subsequently for micro-organisms to multiply (46;47). Bacteria ferment carbohydrates to acids, which demineralise calcified dental tissues. Caries is not necessarily confined to the infundibular cementum: it can ultimately affect adjacent enamel and dentine and expose a pulp horn directly. The bacterial infection can then extend into pulp which can ultimately lead to apical infection and secondary exposure of other pulp horns (Fig 17). Such physical communication between infundibular cemental caries and a pulp chamber is seldom found, but more systematic studies may prove otherwise (5).

Various *developmental abnormalities (dysplasia)* are recognised and some may predispose to apical infection and occlusal pulpar exposure. Infundibular cemental hypoplasia is the incomplete filling of an infundibulum by cementum and as described above, this condition can predispose to infundibular cemental caries (48). Caries is also seen in dysplastic infundibula and exaggerated peripheral enamel infoldings, which can resemble infundibula (Fig. 19). Abnormal infolding of peripheral enamel has also been related to descending periodontal disease (33;37).

Figure 7: Longitudinal section of Triadan 307 which had subtle occlusal lesions of pulps 1 and 4 but deeper into the tooth un-occluded pulp horns grossly impacted with food (stars) were present. The pulp horns are sealed off (arrows) apically from this diseased area.



Pulpar exposure has also been proposed to develop from an *imbalance between attrition and secondary dentine deposition* (38;39;49). Non infectious factors that could negatively influence odontoblasts' production include general health status and nutritional deficiencies (3;11). Alternatively, increased wear at the occlusal surface could be caused by grazing abrasive grasses with high phytholith content or short, sand covered vegetation (33). Multiple cheek teeth throughout the arcades would then be expected to develop multiple occlusal pulpar exposures. While this seems plausible in theory, healthy odontoblasts should normally react to an increase in wear with increased secondary dentine deposition.

Such reactive odontoblast activity is also seen after tooth rasping, but *iatrogenic pulpar exposure* can occur with over-floating, diastema burring, bit-seating or cutting with dental shears (5;6;16). The distance between lively pulp and the occlusal surface of cheek teeth is small and varies with dental age, dental overgrowths (i.e. whether the tooth is adequately opposed), health status of the pulp and odontoblasts, individual horses and individual cheek teeth (Fig. 1). Becker (11) suggested an average of 10 millimetres and Dacre (3) reported measurements of two to six millimetres of subocclusal secondary dentine. Especially since the use of high power dental instruments care is taken not to damage one or multiple pulps. Direct (primary) exposure of a pulp horn is recognised by appearance of a pink translucency in the dentine, followed by haemorrhage in this area. Additionally, thermal damage to odontoblasts can indirectly lead to occlusal pulpar exposure considerable time after use of these

instruments (31). Furthermore, ultrastructural research shows that even when vital pulp is not directly exposed dentinal tubules with odontoblastic processes are greatly disrupted by use of both handfloats and motorised dental equipment. This could predispose to colonisation by microbes (16).

Fissure fractures, traumatic fractures and idiopathic fractures are direct ways of entry for bacteria into pulp chambers and deeper apical tissues involved in the fracture line (primary pulpar exposure). Occlusal pulpar exposure of unfractured pulp horns can subsequently develop after ischemic necrosis or spread of septic pulpitis (secondary pulpar exposure). Fissure fractures are hairline fractures that can extend a variable distance from the occlusal surface and can involve various dental tissues as well as the pulp cavities (Fig. 18). Traumatic fractures in cheek teeth occur after accidents (kicks, sport related falls and so forth) or occur iatrogenically during dental extraction, repulsion or, as previously was discussed, when cutting cheek teeth. Idiopathic fractures are fractures that occur without a known traumatic history and these will be discussed in more detail in the following paragraphs (6;11;50). Several different idiopathic fracture patterns are recognised in equine cheek teeth, with *maxillary lateral (buccal) slab fractures* being most encountered (Fig. 26). (6-8) The fracture plane in this pattern involves the two lateral pulp chambers, pulp number 1 and 2. It runs from the occlusal surface to the lateral clinical or coronal reserve crown, thus creating an unstable buccal tooth fragment that is usually displaced laterally (6).

Another frequently diagnosed longitudinal fracture pattern involves carious infundibula and is therefore termed *infundibular sagittal fracture* or *maxillary midline sagittal fracture* (Fig. 28) (6). These cheek teeth typically have progressive infundibular cemental caries which dissolved enamel and dentine surrounding the cemental lakes. This coalescence of the rostral and caudal infundibulum makes the tooth structurally very weak. It can no longer tolerate the extreme mechanical forces to which it is subjected during mastication and fractures through the carious midline of the tooth. Since the carious defect can stretch deep into the tooth, the fracture usually involves the whole length of clinical and reserve crown (6;15;51).

In mandibular cheek teeth, the *mandibular lateral (buccal) slab fracture* is found most commonly (Fig. 27). The fracture plane is directed longitudinally and runs through lateral pulp chambers 1 and 2 (6-8).

Various less typical fracture planes are recorded in both maxillary and mandibular cheek teeth: *miscellaneous fracture patterns* (Fig. 25, 29) (6-8). In maxillary cheek teeth the fracture line usually runs through medial (palatal) pulp horns (6). Although heterogeneous, it is important to recognise this group because of the involvement of pulp chambers in the fracture planes.

2. Research Objectives

Previous paragraphs have outlined scientific support for various aetiopathogeneses of apical infections and occlusal pulpar exposure, including idiopathic fracture. Occlusal pulpar exposure in equine cheek teeth was recorded both in earlier studies of general horse populations (all cited by 11;38;52;53;54) and in more recent studies of referred equine populations (5;33;37;55;56) and donkey cheek teeth (57). Little is known however about possible predilection of certain pulp chambers to exposure. This could reflect a relationship with infection routes and fracture patterns, or alternatively relate to endodontic anatomy. It would also be valuable to differentiate between occlusal pitting of secondary dentine and occlusal exposure of pulp, since they could reflect the pulp's ability to overcome injury.

The objectives of this study were *1.* to establish the prevalence of occlusal pulpar exposure in cheek teeth that were surgically extracted because of apical infection or idiopathic cheek teeth fractures; *2.* to assess which pulp horns, and which pulp horn combinations are preferentially exposed in these disorders; and *3.* to assess the aetiology of the apical infections, and establish the fracture patterns of the fractured cheek teeth to assess possible relationships between patterns of occlusal pulpar exposure and the type of apical infection or fracture patterns that necessitated cheek teeth extraction.

3. Materials and methods

A total of 110 cheek teeth extracted or repulsed at Easter Bush Veterinary Centre between 1993 and 2008 were used (98 of which were removed between 2004 and 2008). The cheek teeth were fixated in 10% buffered formalin and stored in a labelled, closed plastic container

since their removal (Fig. 8). Complete clinical and ancillary histories were available for 88 teeth. The specimens included 79 apically infected cheek teeth without any obvious gross cause of infection (e.g. no gross fracture or periodontal disease) of median *dental* age of 3.5 years (range -0.5 to 25 years) and 31 cheek teeth with idiopathic fractures of median dental age of 8.5 years (range 1 to 20 years). The dental age is the time since eruption and therefore reflects the functional age of the tooth. Adjacent teeth in a horse of a certain age have different maturity and root development, since eruption of permanent equine cheek teeth is staggered over a period of three years (13;14;39). These data are summarised in Table 1.

Figure 8. Some collected cheek tooth specimens.

Cheek teeth with idiopathic fractures that had concurrent apical infections were retained in the idiopathic fracture classification group. Apically infected cheek teeth which were later found to have fissure fractures on detailed examination, were retained in the apical infection group.

	Total	Apical infection		Idiopathic fracture	
Number of specimens	110	79		31	
Number specimens known history	88	64		24	
Number of specimens incomplete history	22	15		7	
Median dental age (years)		3,5		8,5	
		Number of CT	% of ap inf CT	Number of CT	% of fx CT
Dental age < 5 years	35	30	38%	5	16%
Dental age 5-10 years	26	17	22%	9	29%
Dental age 10-15 years	19	11	14%	8	26%
Dental age > 15	8	6	8%	2	6%
Dental age unknown	22	15	15%	7	23%
Maxillary cheek teeth	63	41	52%	21	68%
Mandibular cheek teeth	47	38	48%	10	32%

Table 1: Number and dental age of cheek teeth (CT) with apical infections (ap inf) and idiopathic fractures (fx).

The idiopathic cheek teeth fractures were categorised into five described fracture patterns (6) and all cheek teeth were visually examined. The pulp nomenclature of du Toit et al. (29) was used in this study (Fig. 3). To further assess changes, 65 apically infected and 11 fractured cheek teeth were transversely sectioned and 4 cheek teeth with apical infections were sectioned longitudinally. A water-cooled tile saw (Dimas TS 230F, Electrolux Construction

Products France) was used to transect the specimens. The following sections were made: an occlusal section circa 0.5 centimetres distal from the occlusal surface, a subocclusal section circa 0.5 centimetres distal from the previous section, a cut through the middle of the crown (which produced two midsections) and an apical section at the apex of the cheek tooth (Fig. 9).

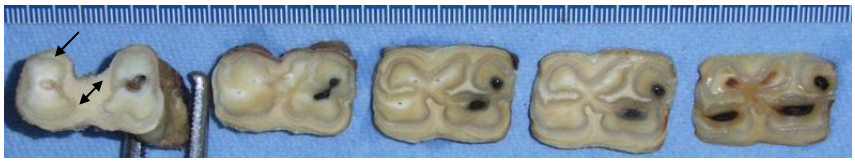


Figure 9: A sectioned mandibular cheek tooth (Triadan 307, dental age 11). From right to left; the occlusal section, the subocclusal section, the two midsections and the apical section. Enamel (arrows) is present at the apical slice and the tooth was therefore not truly sectioned through the apex.

Tooth structure and occlusal pulpar exposure was also visualised using Computerised Axial Tomography (CAT). Five apically infected cheek teeth with visible occlusal pulpar exposure and six without visible pulp exposure were imaged with a Somatom Esprit CAT scanner (Siemens AG) at the Scottish Agricultural College (SAC) and processed with OsiriX software.

Two-Sample T-Tests were carried out to determine if there were statistically significant differences in occlusal pulpar exposure between maxillary and mandibular cheek teeth (confidence interval 95%), using the statistical programme Minitab.

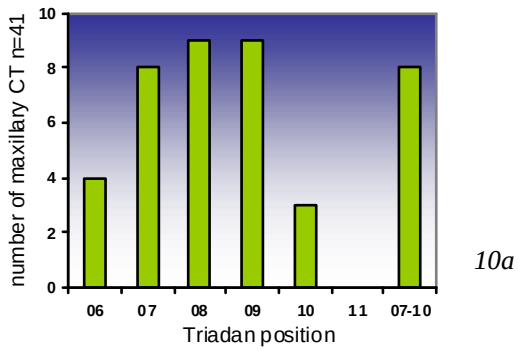
4. Results

The central Triadan positions 07, 08 and 09 were over-represented in both apically infected and fractured cheek teeth. Twenty-four percent of apically infected cheek teeth were Triadan 07s, 20% Triadan 08s and 19% Triadan 09s. Maxillary cheek teeth were more commonly fractured (65% of all fractured cheek teeth) than mandibular cheek teeth (32%), with maxillary 09s preferentially affected (Triadan 07s comprised 10%, Triadan 08s comprised 19% and Triadan 09s comprised 42% of all fractured maxillary cheek teeth). These findings are summarised in table 2 and figures 10 and 11.

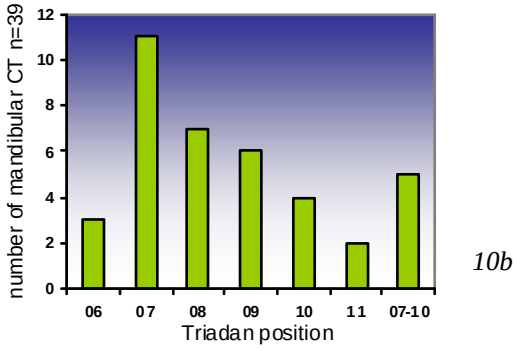
	Apical infection		Idiopathic fracture	
	Number of CT	Percentage of CT	Number of CT	Percentage of CT
Triadan number 06	7	9%	0	0%
Triadan number 07	19	24%	3	10%
Triadan number 08	16	20%	6	19%
Triadan number 09	15	19%	13	42%
Triadan number 10	7	9%	1	3%
Triadan number 11	2	3%	1	3%
Triadan number 07/08/09/10	13	16%	7	23%
Total	79		31	

Table 2: Triadan positions of CT (cheek teeth) with apical infections and idiopathic fractures.

Because of the similar morphology of Triadan positions 07 to 10, the correct Triadan numbers of 13 apically infected and 7 fractured cheek teeth with incomplete histories could not be determined with certainty. Because of the distinct morphology of 06s and 11s however, these could be recognised and recorded appropriately even if the case history was incomplete.



10a



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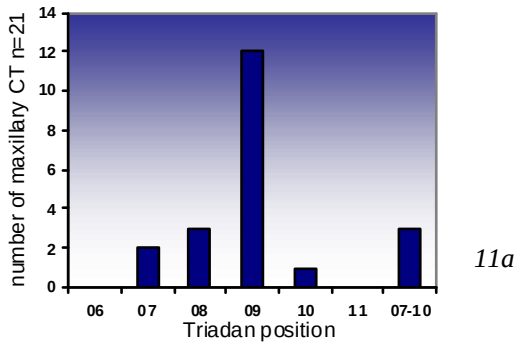
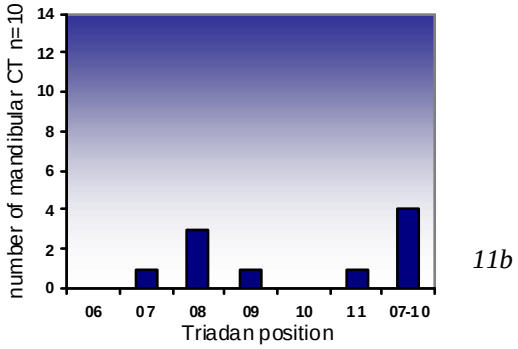


11a

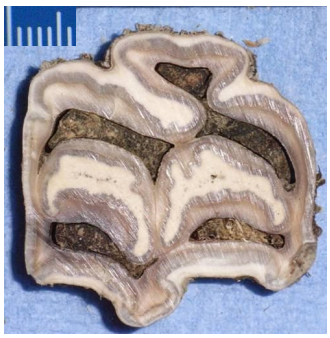


11b

Figure 10: Triadan positions of a. maxillary (41/79 CT; 52%) and b. mandibular CT (38/79; 48%) with apical infections.
Figure 11: Triadan positions of a. maxillary (21/31 CT; 65%) and b. mandibular CT (10/31 CT; 32%) with idiopathic fractures.

4.1 Cheek teeth with apical infections

12



13

Figure 13: Midsection of Triadan 208, dental age 2 y that had all pulps occlusally exposed. All pulps are impacted with food. The circumpulpar dentine is discolored, but no gross caries is present.

Occlusal pulpar exposure in these two teeth was deemed secondary to anachoretic apical infection.

Figure 12: Occlusal pulpar exposure of all pulp horns is present in this mandibular cheek tooth (Triadan 408, dental age 3,5 y). Secondary dentine is totally absent on the occlusal surface and food is packed into the exposed pulp horns (arrow). The surrounding primary dentine is darkly stained (star).

Identification of occlusal pulpar exposure

Occlusal pulpar exposure was characterised as a grossly visible defect in the secondary dentine of the occlusal surface (i.e. overlying a pulp horn). Some long-standing occlusal lesions had eroded all of the occlusal secondary dentine and the defects had the same diameter and outline of the underlying pulp cavity (Figs. 12, 13). However, the majority of occlusal defects had some secondary dentine remaining that was usually stained brown to black (Figs. 6b, 9, 14). The primary dentine lying adjacent to exposed pulps usually remained normal, retaining its cream-coloured translucent appearance, with staining of primary dentine rarely present.

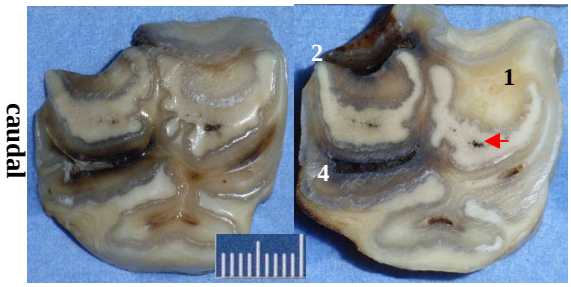


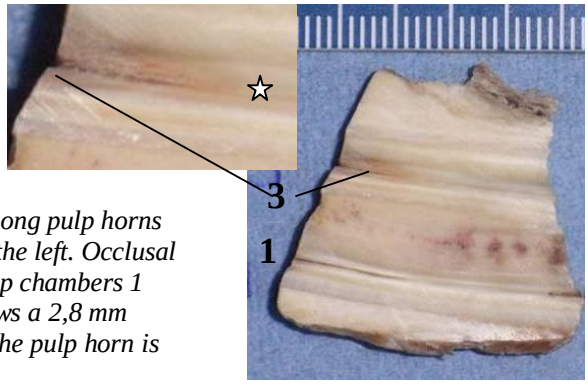
Figure 14: Occlusal and subocclusal section of Triadan 110, dental age 16 y. Descending periodontal disease was present at the caudal surface of the crown with loss of cementum. Adjacent pulps 2 and 4 were compromised, since occlusal exposure, food impaction and staining of secondary dentine can be identified there. Some extraction damage is visible over pulp 2. Pulp horn 1 is sealed off by tertiary dentine and is surrounded by sclerotic primary dentine, whereas pulps 3 and 5 appear healthy. Notice the central vascular channel in each infundibulum (arrow).

The larger occlusal defects were readily identified on visual examination and a two millimetre wide probe could easily be introduced into the patent pulp horn from the occlusal surface. In contrast, a probe could only be advanced a few millimetres into some shallow occlusal secondary dentine indentations or occlusal exposure lesions of a small diameter. Transverse or

longitudinal sections of eight cheek teeth (10% of apically infected cheek teeth) with shallow occlusal secondary dentine lesions showed that the underlying pulp remained protected by a deeper overlying layer of dentine and these superficial lesions were termed *occlusal pitting* of secondary dentine (Fig. 15). In other specimens, a discrete occlusal defect led to a grossly infected or even food-filled pulp horn more apically and these cheek teeth were defined as having occlusal pulpar exposure. Examination of transversely and longitudinally sectioned cheek teeth showed decreased translucency and discoloration of pulps with occlusal exposure and in more chronically infected cheek teeth, necrotic or absence of pulp, with the hollow cylindrical shaped pulp horn usually impacted with food material (Fig. 13).

a

Figure 15a: A maxillary cheek tooth (Triadan 109, dental age 4 y) with occlusal defects in the secondary dentine of pulp horns 1,2,3 and 4. Anachoresis was the most likely aetiology in this tooth.



b

Figure 15b shows the longitudinal section along pulp horns 1 and 3, with the occlusal surface placed to the left. Occlusal pitting of secondary dentine is present in pulp chambers 1 and 3. The magnification of pulp horn 3 shows a 2,8 mm deep indentation in the secondary dentine. The pulp horn is occluded (star) distally from this defect.
This indicates that secondary dentine production had decreased in the past and that the odontoblasts were able to fully occlude the pulp horn distally from this insult.

Aetiopathological findings in apically infected cheek teeth

No physical entry route for oral bacteria into the endodontic system could be identified and therefore a blood or lymph borne infection (anachoretic infection) was considered the most likely cause of infection in 54/79 (68%) of apically infected cheek teeth (Figs. 15, 16). The median dental age was 3.5 years (Table 3). Although 19 of these 54 cheek teeth (35%) had pulpar exposure (Figs. 12, 13), there was no evidence such as iatrogenic pulpar exposure or traumatic fracture to suggest that this pulpar exposure was the *cause* of infections. All occlusal exposure was therefore deemed to be secondary to pulpar death as also found by Dacre et al.(33;37).

Eighteen of the 79 apically infected cheek teeth (22%) with median dental age of 11 years, were believed to have been caused by descending periodontal disease. This was characterised by a continuous tract along the crown from the occlusal surface to the apex with loss of periodontal membranes and erosion of the underlying cementum. This was distinguished from ascending periodontal disease which was often characterised by reactive cementum deposition with chronic infection and largely intact periodontal ligaments at the gingival level in some cheek teeth.

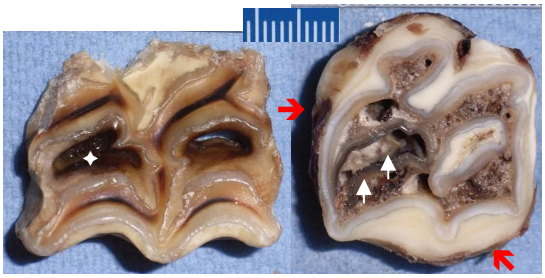


Figure 16: Occlusal and apical section of Triadan 207, dental age 3 y. The occlusal secondary dentine over all pulp horns is intact. The periodontal membranes were largely intact (red arrows). Both infundibula are incompletely filled with cementum (infundibular hypoplasia, star) and cemental caries is present in the rostral infundibulum (arrows). No communication between this carious process and pulp was found and in the absence of any physical entry route, apical infection was attributed to anachoresis.

Cemental caries of one or both infundibula was found in 18 of 79 (23%) apically infected maxillary cheek teeth but was usually aetiologically insignificant for the apical infections (Fig. 16). However, a gross communication was present between the carious infundibulum and a pulp chamber in one cheek teeth and between carious infundibula and the infected apices in two cheek teeth (Fig 17). Consequently, apical infections were attributed to extension of infundibular caries in these three cases (5%, median dental age 3.5 years).

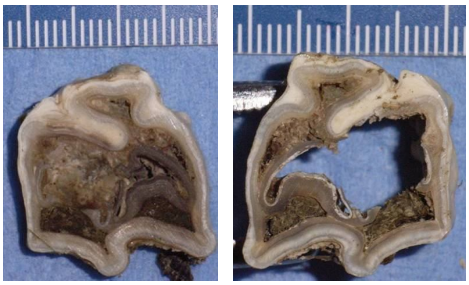


Figure 17: Mid- and apical transverse section of Triadan 110 (dental age 5 y). Cemental caries of the caudal infundibulum has extended into adjacent dental tissues. The caudal infundibulum communicated with the tooth apex, the rostral infundibulum, pulp chamber 2, 3, 4 and indirectly also with pulp chamber 5. Pulp chamber 1 and 2 contain food material, the other pulps are necrotic. This suggests that infundibular caries led to pulpitis and periapical abscessation.

Fissure fractures were found in three apically infected cheek teeth (all mandibular). These were subtle lesions, with staining along the fissure line only evident on examination of transversely sectioned teeth. In one cheek teeth, the fracture only involved peripheral cementum, but in the other two cheek teeth (3%) (aged dental age 4 and 6 years) the fracture

extended to involve the buccal pulp horns (pulp horns 1 and 2) that were necrotic, indicating that the fracture was the likely infection route (Fig. 18).

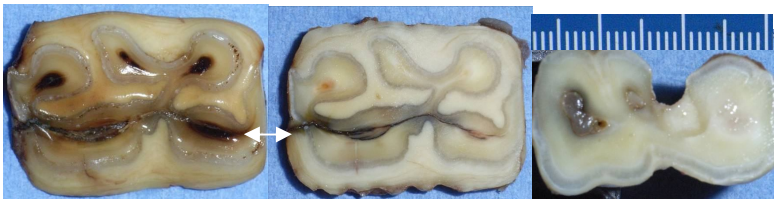


Figure 18: Occlusal, subocclusal and apical section of Triadan 407 (dental age 6 y) shows a fissure fracture through the lateral (buccal) pulp horns (arrow). Pulp in the caudal common pulp chamber has a dark and dull appearance.

Four (5%) apically infected cheek teeth were grossly dysplastic, with the infection directly attributed to dysplasia in two cheek teeth (3%, median dental age 2.5 years). In one tooth, peripheral cementum incompletely filled a peripheral enamel infolding, creating a full length longitudinal defect, that allowed descending periodontal disease to access the apex. Exaggerated peripheral enamel infoldings, resembling infundibula, were present in three mandibular cheek teeth and cemental caries of one such infolding provided a route of infection to the apex (Fig. 19).

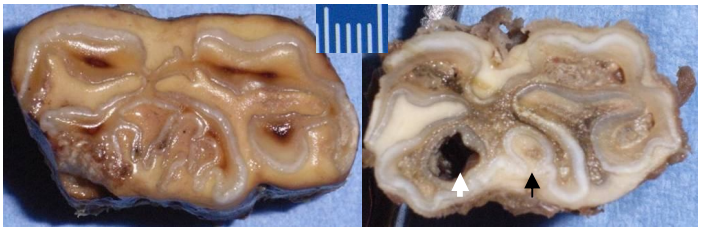


Figure 19: Exaggerated enamel infoldings in this mandibular cheek tooth (Triadan 407, dental age 1 y) resemble two infundibula (arrows) deeper into the tooth. Cemental caries is present in such infolding (white arrow) and all pulps have a discoloured, dull appearance.

Prevalence of occlusal pulpar exposure in cheek teeth with apical infections

Occlusal pulpar exposure was present in 25 (32%) cheek teeth with apical infections, with multiple pulps exposed in 21 (27%) cheek teeth and a single pulp exposed in four (5%) cheek teeth (Table 4). Occlusal pitting of secondary dentine occurred in eight cheek teeth (10%), with four (5%) cheek teeth showing pitting of multiple pulp horns and four (5%) cheek teeth showing dentinal pitting over a single pulp horn (5%). The prevalence of occlusal pulpar exposure for each proposed aetiology is summarised in Table 3.

Infection route	Number of CT (n=79)	Median dental age (years)	Occlusal pulpar exposure		
			Total	Multiple	Single
Anachoresis	54/79 (68%)	3,5	19/54 (35%)	16/54 (30%)	3/54 (6%)
Periodontal disease	18 /79 (23%)	11	3/18 (17%)	2/18 (11%)	1/18 (6%)
Infundibular caries	3/79 (4%)	3,5	1/3 (33%)	1/3 (33%)	0
Fissure fracture	2/79 (3%)	5	0	0	0
Dysplasia	2/79 (3%)	2,5	2/2 (100%)	2/2 (100%)	0

Table 3: Number, median dental ages and prevalence of occlusal pulpar exposure in CT with apical infections for each infection route.

Exposure of specific pulp horns with cheek teeth apical infections

No statistical significant difference (p = 0,285) between the prevalence of occlusal pulpar exposure in maxillary versus mandibular cheek teeth of all age groups was found (Fig 20).

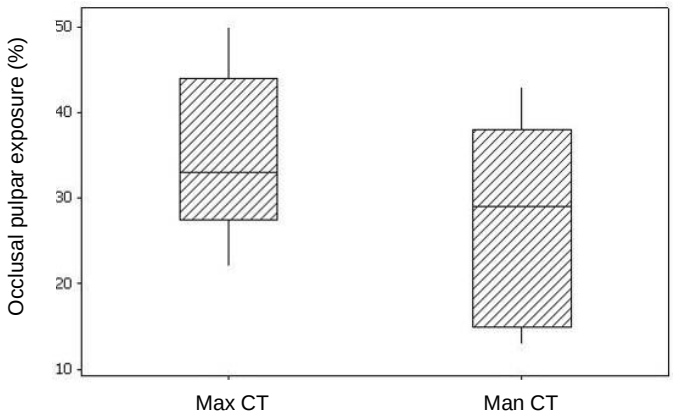


Figure 20: Prevalence of occlusal pulpar exposure in maxillary and mandibular CT (all age groups)

Because equine maxillary and mandibular cheek teeth have different endodontic anatomy (23;29) data on exposure of the individual pulp horns in maxillary and mandibular cheek teeth are presented separately (Table 4) with the pulp horn nomenclature of du Toit et al. (29) used. The caudo-medial pulp horn was most frequently exposed in both upper and lower cheek teeth, i.e. pulp 4 was affected in 10/14 (71%) occlusally exposed maxillary cheek teeth and pulp 5 in 9/11 (82%) occlusally exposed mandibular cheek teeth. No occlusal exposure of pulps 6, 7 or 8 was found in this study.

	Occlusal pulpar exposure			Pulp 1	Pulp 2	Pulp 3	Pulp 4	Pulp 5	Pulp 6	Pulp 7	Pulp 8
	Total	Multiple	Single								
Max CT	14	12	2	8 (57%)	7 (50%)	7 (50%)	10 (71%)	7 (50%)	0	0	0
Man CT	11	9	2	4 (36%)	5 (45%)	7 (64%)	7 (64%)	9 (82%)	0	0	-
Total	25 (32%)	21 (27%)	4 (5%)								

Table 4: Exposure of individual pulp horns, expressed as percentages of total occlusal pulpar exposure in apically infected maxillary (Max) and mandibular (Man) CT. Note that data in horizontal rows are not cumulative, since multiple pulps can be simultaneously exposed.

Furthermore, exposure of multiple pulps occurred in various combinations (Fig. 21), with simultaneous exposure of pulps 3 and 5 most commonly recorded in maxillary cheek teeth, i.e. in seven of 12 (58%) upper cheek teeth with multiple exposures, whereas pulps 2 and 5 were most frequently simultaneously exposed in mandibular cheek teeth (Figs. 6b, 9), i.e. in seven of 9 (78%) lower cheek teeth with multiple pulp exposures.

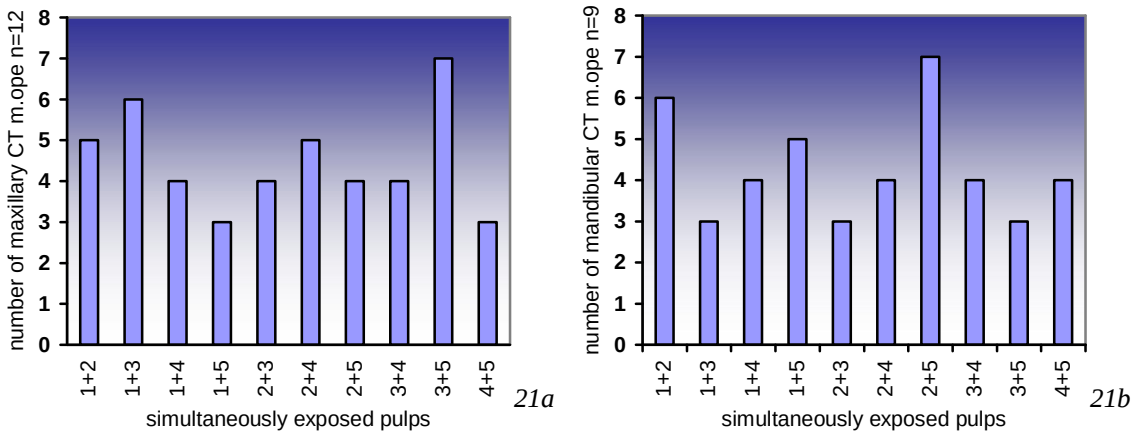


Figure 21: Histogram identifying the pulps that are simultaneously exposed in a. maxillary and b. mandibular apically infected cheek teeth (m.ope, multiple occlusal pulpar exposure).

4.2 Computerised Axial Tomography of apically infected cheek teeth

Different densities were selected to visualise different dental tissues. The enamel skeleton and the apical, enamel free area could be identified when cemental density was blocked out (Fig. 24). The infundibula surrounded by enamel and filled with cementum (or alternatively infundibular cemental hypoplasia) and reactive changes such as hypercementosis could also be identified through CAT scan imaging.

Moreover, computerised axial tomography was useful to visualise pulp chamber anatomy. Images at different transverse levels of the cheek teeth showed communication and separation of the different pulp horns (Fig. 23). Pulp chambers could be visualised from the tooth apex to the occlusal surface in longitudinal images.

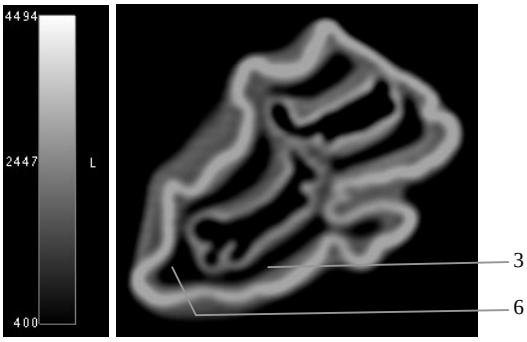


Figure 22: Transverse CAT scan image of Triadan 206, dental age 3,5 y. At this midlevel (Z axis 76,8 mm) communication between pulp chambers 3 and 6 is visible. All pulp chambers contain pulp at this level. The two infundibula appear hollow, but this is mainly the result of the chosen imaging settings. At other settings this cheek tooth had well filled infundibula with central vascular channels. No physical entry route was found with CAT scan imaging and macroscopic examination and apical infection of this cheek tooth was therefore attributed to anachoresis.

Un-occluded pulp chambers were visible as lucent areas due to their low tissue density and air filled spaces. Air in pulp horns indicate the presence of gas producing bacteria in vivo studies in other species. This observation however was considered an artefact in these dead equine cheek teeth, because of the inevitable loss of pulpar tissue and presence of air in normal pulp chambers after extraction and preservation (29). Infundibula of some relatively normal equine cheek teeth contained air which required careful inspection of CAT scan images to distinguish this from gas production in pulps. Furthermore, the changes in radiodensity of exposed pulp horns were often subtle and also depended in some degree on the direction of the image selected. The radiographic appearance of pulp horns also depends on the tooth's age. For these reasons, CAT scan images indicated suspicious areas to investigate further but could not identify occlusal pulpar exposure conclusively.

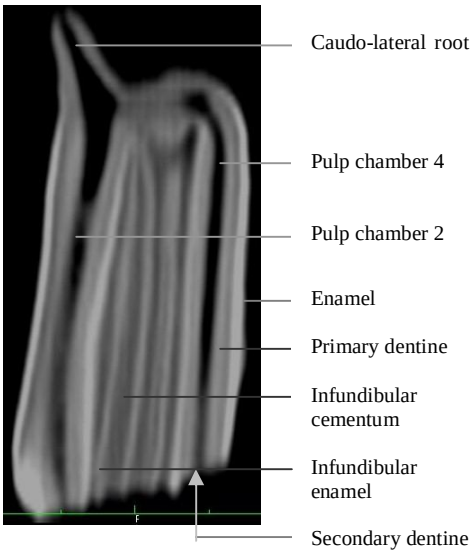


Figure 23: Longitudinal CAT scan image of Triadan 207, dental age 5 y. Pulp chamber 4 is fully visible from the tooth apex to the occlusal surface. The occlusal layer of secondary dentine can be recognised, being more radiodense than chamber content and more lucent than adjacent primary dentine and enamel. The image also shows the caudal infundibulum.

The secondary dentine layer is less prominent in pulp chamber 2, which appears highly suspicious of occlusal pulpar exposure. Visual examination of transverse sections proved however, that all pulp horns were occluded at occlusal and subocclusal levels. Anachoresis was considered to be the infection route into this tooth.

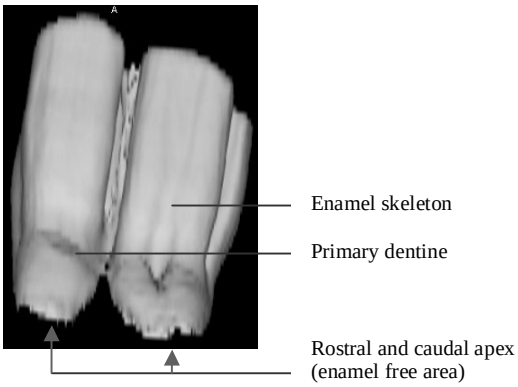


Figure 24: Triadan 308, dental age 10 y. The chosen density allows visualisation of enamel and primary dentine. The cementum, and therefore the shape of the apex, is not visible.

4.3 Cheek teeth with idiopathic fractures

Gross examination

Fracture planes that ran through pulp horns provided longitudinal views of the fractured pulp horns with most of these appearing to contain a normal amount of secondary dentine (Fig. 26). However, the fractured pulp horns of three cheek teeth in particular, contained no secondary dentine and were grossly discoloured, indicating that pulpar death and occlusal exposure were most likely present before the fracture occurred, as also recorded by Dacre et al. (6), although some such changes could represent carious changes that developed after fracture (Fig. 25).

Such interpretation of pulpar health prior to fracture could not be made about the pulps uninvolved in the fracture plane. Visual examination could not deduct if occlusal pulpar exposure was present prior to tooth fracture, or that pulpar death occurred as a result of the fracture.

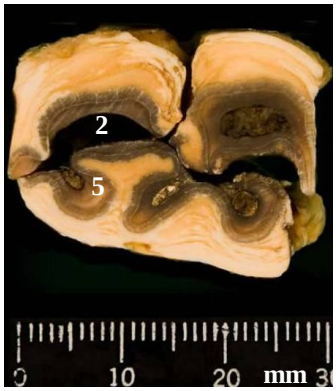


Figure 25: Mandibular miscellaneous fracture pattern (CT row 3) through pulp horns 2,5, primary dentine surrounding 3,4 and cementum between 1,2. The figure shows the subocclusal section.

The absence of secondary dentine in fractured pulp horns 2 and 5 reflect longstanding pulpar death before the tooth fractured.

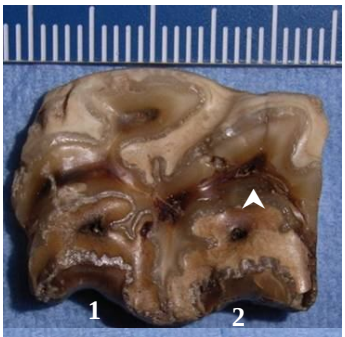
The un-occluded pulp horns show food impaction and some degree of dentinal caries.

Recorded fracture patterns

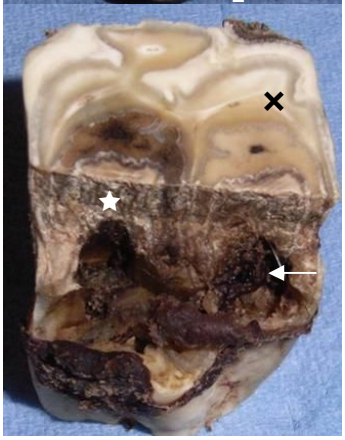
The fracture patterns recorded in the examined specimens (Figs. 25-29) corresponded with the five idiopathic fracture patterns discussed earlier. These fracture patterns were very consistent and always involved one or more pulp horns and/or infundibula. Table 5 presents the recorded prevalence of idiopathic fracture patterns. The midline sagittal fracture pattern was the most common pattern in maxillary cheek teeth (48% of maxillary cheek teeth fractures). Miscellaneous fracture was the most frequently recorded pattern in mandibular cheek teeth (60% of mandibular cheek teeth fractures).

	Maxillary cheek teeth		Mandibular cheek teeth	
	Number of CT	Percentage of max CT	Number of CT	Percentage of man CT
Max midline sagittal fracture	10	48%		
Max lateral slab fracture	4	19%		
Max miscellaneous fracture	7	33%		
Man lateral slab fracture			4	40%
Man miscellaneous fracture			6	60%
Total (n=31)	21		10	

Table 5: Prevalence of idiopathic cheek teeth fracture patterns in 31 cheek teeth.



26a

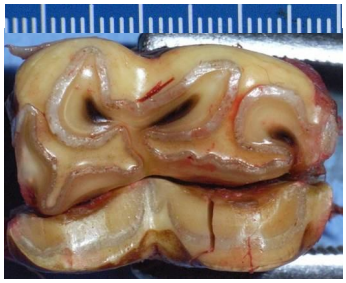


26b

Figure 26a: Maxillary lateral slab fracture (Triadan 109, dental age 10 y) through lateral pulp horns 1 and 2.
Notice the occlusal pitting of secondary dentine (arrowhead) over pulp horn 4, while this pulp horn is occluded (cross) on the subocclusal section pictured in Fig. 26b. Notice the central vascular channels of the infundibula and limited caries deeper into the rostral infundibulum.

26b: The occluded part (star) of the chamber and the apical part containing pulpar tissue (arrow) are visible in the fracture plane, indicating that pulps 1 and 2 were not compromised prior to fracturing.

Figure 27: Mandibular lateral slab fracture (Triadan 308, dental age 5 y) through lateral pulp chambers 1 and 2. This tooth was extracted soon after fracture, marked by the presence of secondary dentine over all pulps and the absence of carious changes.
The appearance of this tooth's pulp in the sub-acute stage of injury is shown in Fig. 4.



27

Prevalence of occlusal pulpar exposure in cheek teeth with idiopathic fractures

Occlusal pulpar exposure was present in 13/31 fractured cheek teeth (42%) involving multiple pulp chambers in 8 (26%) and a single pulp in 5 (16%) cheek teeth. Pitting of occlusal secondary dentine was recorded only over one pulp horn in a maxillary cheek tooth with a lateral slab fracture. These findings are summarised in tables 6 and 7. No statistical significant difference (p = 0,743) between the prevalence of occlusal pulpar exposure in maxillary versus mandibular cheek teeth was found.

	Single ope	Multiple ope	Single pitting	Prevalence ope per idiopathic fracture pattern
Max midline sagittal fracture	2 (20%)	2 (20%)	0	40%
Max lateral slab fracture	0	1 (25%)	1 (25%)	25%
Max miscellaneous fracture	2 (29%)	0	0	29%
Man lateral slab fracture	0	1 (25%)	0	25%
Man miscellaneous fracture	1 (17%)	4 (67%)	0	83%
Total (n=31)	5	8	1	

Table 6: Prevalence of occlusal pulpar exposure (ope) and pitting with five different idiopathic fracture patterns.

Occlusal pulpar exposure occurred in all fracture patterns (Table 6) with the highest prevalence in mandibular cheek teeth with miscellaneous fractures (5/6, 83%) and maxillary cheek teeth with midline sagittal fractures (4/10, 40%).

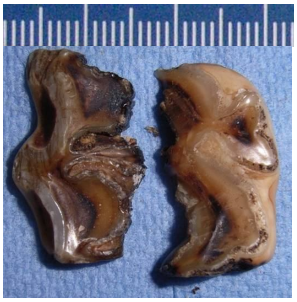


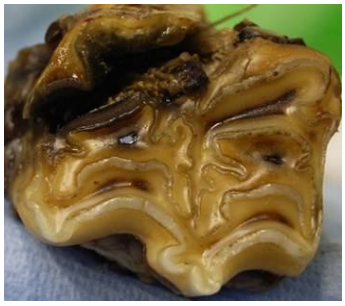
Figure 28: Maxillary midline sagittal fracture pattern (Triadan 109, dental age 9 y). All pulp horns are occluded at the occlusal surface. Extensive infundibular caries is present and the fracture line is directed through these coalesced infundibula.

Exposure of specific pulp horns in cheek teeth with idiopathic fractures

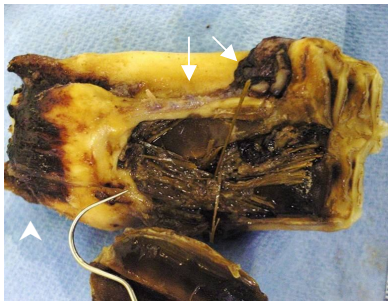
Maxillary pulps 1, 3 and 5 were exposed with the same frequency (43%). Mandibular pulp 5 was affected most frequently in lower cheek teeth (5/6 83%). Occlusal exposure of pulps 6, 7 and 8 was not found (Table 7).

	Occlusal pulpar exposure			Pulp 1	Pulp 2	Pulp 3	Pulp 4	Pulp 5	Pulp 6	Pulp 7	Pulp 8	Pulp un-known
	Total	Multiple	Single									
Max CT	7	3	4	3 (43%)	2 (29%)	3 (43%)	2 (29%)	3 (43%)	0	0	0	1 (14%)
Man CT	6	5	1	1 (17%)	2 (33%)	3 (50%)	3 (50%)	5 (83%)	0	0	-	0
Total	13 (42%)	8 (26%)	5 (16%)									

Table 7: Exposure of individual pulp horns, expressed as percentages of total occlusal pulpar exposure in maxillary (Max CT) and mandibular cheek teeth (Man CT) with idiopathic fractures.



a.



b.

Figure 29a. Maxillary miscellaneous fracture of Triadan 107 (dental age 3 y). The fracture plane runs through pulp horns 3 and 5, that are impacted with fibrous food material. The surrounding dental tissues are stained. The occlusal dentinal layers of all pulp horns are intact.

Figure 29b. Longitudinal view of 29a. The fragment is reflected to show food impaction. Reactive cemental deposition and iatrogenic damage during tooth repulsion have deformed the tooth apex (arrowhead). The periodontium is largely intact (arrows).

In cheek teeth with multiple pulpar exposure, the combinations of exposed pulp horns differed between the identified fracture patterns (Fig. 30). Palatal and buccal pulp chambers were concurrently exposed only in maxillary cheek teeth with midline sagittal fractures. In the maxillary and mandibular lateral slab fracture patterns, the medial pulp chambers (pulps 3, 4 and 5) were usually exposed simultaneously. Simultaneous pulpar exposure was less uniform in the mandibular miscellaneous fracture pattern.

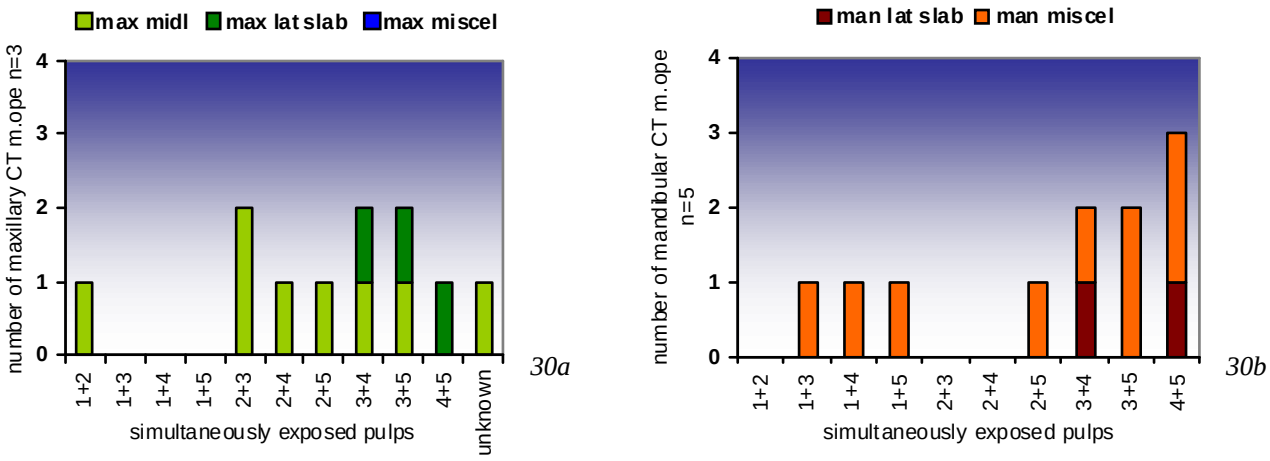


Figure 30: Histogram identifying the pulps that are simultaneously exposed in a. maxillary and b. mandibular cheek teeth with idiopathic fractures.
Note that multiple occlusal pulpar exposure (m.ope) in maxillary miscellaneous fracture patterns was not found.

5. Discussion

In maxillary cheek teeth, Triadan positions 07, 08 and 09 most frequently presented with apical infection as was also found by Wafa (38), Lane (58) and Dixon (5). Apical infection of mandibular cheek teeth was most commonly recorded in Triadan 07s and 08s, which corresponds with findings in other studies (5;45;58). This is possibly related to the predilection of centrally located cheek teeth to retention of deciduous teeth (caps) and vertical impaction. Pressure on the periapical bone during eruption can result in an eruption cyst around the developing, hyperaemic apex which predisposes it to infection (2;15;59). Apical infections of the first (06s) and last (11s) cheek teeth were relatively uncommon, but appear to be diagnosed more frequently since the widespread use of motorised dental equipment (60). In current study however, no macroscopic or radiographic (CAT scan) evidence of iatrogenic damage was found in apically infected 06s or 11s.

Idiopathic cheek teeth fractures were most commonly found in *maxillary* cheek teeth and Triadan 109 and 209 were affected in particular, as was also reported by others (6;7;11). Being the first permanent cheek tooth to erupt, significant infundibular caries and pre-senile excavation are more common in maxillary 09s and it is suspected that these teeth are therefore predisposed to idiopathic (midline sagittal) fractures (6;7;11). It also was proposed that maxillary 09s take the highest pressure during the mastication cycle, but it was recently suggested that in fact the caudal molars are subjected to most of this pressure (6).

Defects of the occlusal secondary dentine were often subtle and in some cases, occlusal pitting could only be distinguished from occlusal pulpar exposure after the affected teeth were sectioned (Fig. 6). Therefore, the term occlusal defect would be more appropriate to use to describe both occlusal exposure and occlusal pitting defects. Clinical diagnosis of these subtle lesions poses a challenge, because occlusal examination is compromised by the limited access to the equine mouth. Adequate sedation and use of a full mouth speculum, strong headlight, equine dental mirror and probe or alternatively a dental endoscope are required to accurately identify such occlusal lesions (61). Furthermore, CAT scan imaging was useful in current study to determine pulp chamber anatomy and could identify pulp horns suspicious of pulpitis and exposure. The use of computerised axial tomography in *live* patients however, is likely of greater value to assess endodontic changes and occlusal defects, since the radiographic appearance of the dental tissues is not compromised by (gas) artefacts.

Healthy pulp chambers should never become occlusally exposed (62) and therefore the presence of occlusal pulpar exposure is evidence that severe pulpar disease has occurred in the past (33;37). Under appropriate conditions, inflamed pulp is very capable of recovery and repair by depositing tertiary dentine formed by odontoblasts (reactive dentine) or undifferentiated mesenchymal cells (reparative dentin) (3) between vital pulp and the oral environment. In some maxillary cheek teeth with lateral slab fractures for example, the larger palatal fracture fragments can remain stable, without signs of clinical apical infections or pulpar exposure, indicating that the exposed pulps have become sealed off (6-8).

It would appear that three different scenarios can occur with pulpar insult. Firstly, with extensive pulpar damage (e.g. ischaemic or bacterial insult) no tertiary dentine is laid down in that horn and with further dental eruption, its occlusal aspect will be exposed allowing food and bacterial ingress into the already compromised pulp horn. Secondly, with less severe,

reversible insult of pulp horn tips, defective secondary dentine, grossly characterised by having an irregular, partial occlusal defect will develop in the occlusal secondary dentine, and this imperfect secondary dentine will clinically appear as occlusal pitting of secondary dentine. The pulp remains sealed off from the oral environment and remains viable below this level, and later produces normal secondary dentine. Thirdly, extensive and irreversible inflammation that is localised to the tip of a pulp horn may lead to local pulpar death and cessation of dentinal deposition at this site, and thus lead to pulpar exposure. However, the pulp remains healthy more apically to the pulp tip, laying down a layer of tertiary dentine that protects the pulp from the overlying occlusal exposure (37;57). This will grossly manifest itself as an apparent total occlusal defect – but on deeper probing – is found to be of limited depth and viable pulp remains deep to the tertiary dentine. A variation on the latter is when the whole endodontic system is occlusally exposed and becomes sealed off more apically by reactive calcified tissue.

Occlusal pulpar exposure is a common symptom in cheek teeth with apical infections and idiopathic fractures: a prevalence of respectively 32% and 42% was found in current study and similar findings in apically infected cheek teeth were reported by Dacre (17), Dacre et al. (33;37) and Casey and Tremaine (63). The study by Casey and Tremaine (64) also demonstrated that identification of occlusal defects is useful for detecting apical pulpitis (specificity of 98,9%, sensitivity of 54,5% in 44 apically infected cheek teeth and 90 controls). However, occlusal lesions confirm secondary dentine deposition has ceased in the *past* and lesions of occlusal secondary dentine including pulpar exposure are occasionally found in asymptomatic cheek teeth (false positive) (38;57;65). Furthermore, the absence of occlusal lesions does not rule out pulpitis and apical infection (false negative). Secondary dentine over pulp horns in some apically infected cheek teeth can be fully intact since pulpitis and apical infection only become apparent at the occlusal surface over time, when the tooth is worn down to the level of injury (Figs.15, 16 and 19). Consequently, the presence of occlusal defects does not necessarily give conclusive information about the *current* endodontic health status of the tooth, unless gross exposure of multiple pulps is present, such as the cheek teeth in Figs. 12 and 13. In these advanced cases however, the accompanying clinical signs and evidence from diagnostic imaging are so marked that the detected occlusal pulpar exposure is often not the decisive diagnostic factor.

The causes of apical infection in this study were similar to those described by Dacre et al. (33;37) with anachoretic infection the main cause, and periodontal disease, infundibular caries, fissure fractures and dysplasia all less common causes. In cheek teeth with apical infections exposure of multiple pulp horns was more common than exposure of a single pulp horn, regardless of aetiology of the apical infection. This indicates that when a clinical apical infection occurs, it usually causes (an infective or ischaemic) insult to multiple, rather than to single pulp horns (or the common pulp chamber) and this absent or defective secondary dentine deposition in multiple pulp horns leads to multiple occlusal pulpar exposure. Occlusal pulpar exposure was most frequently found in cheek teeth with anachoretic infection (median dental age 3.5 years) and multiple pulp horns were usually exposed. It has been suggested that because the apices of developing cheek teeth are often hyperaemic, these tissues may be particularly susceptible to blood borne bacterial invasion (5;66). Additionally, young cheek teeth may still a have a common pulp chamber or have large communications between pulp horns (30), which could facilitate spread of infection to multiple pulps.

Descending periodontal disease is usually present in older cheek teeth (median dental age 11 years in current study). It is reported to preferentially affect the lingual aspect of mandibular and the buccal aspect of maxillary cheek teeth interproximal spaces (15;45;67) and Becker (11) proposed that pulpar exposure in apically infected cheek teeth of periodontal disease origin would be limited to adjacent pulp horns. However, this was only observed in a minority of cases in the current study (Fig. 14) and usually multiple pulps were affected in teeth that became apically infected by the periodontal route. The pulps in chambers involved in a fracture plane or a carious process are directly exposed to the oral environment. However, multiple pulpar exposure found in these cheek teeth in this study indicates that the pulpitis also compromises the odontoblasts in other pulp horns, indirectly leading to secondary occlusal pulpar exposure of other horns.

Maxillary pulps 3 and 5, and mandibular pulps 2 and 5 were the pulp horn combinations that were most frequently concurrently exposed in apically infected cheek teeth. This is most likely due to their anatomical communications, which are most marked in young cheek teeth. Maxillary pulps 3 and 5 are reported to have anatomical communications most frequently in horses (3) and donkeys (29). In mandibular cheek teeth, most communications are between pulp 2 and 5, both in horses (3) and donkeys (29).

As a result of the ongoing circumferential replacement of pulp by secondary dentine, the common pulp chamber and individual pulp horns becomes gradually smaller as the tooth matures. It has been proposed that the common pulp chamber remains in equine mandibular cheek teeth for four to five years following eruption and that two separate pulp chambers with individual pulp horns can be identified after six years (30). The pulp communications present in younger equine cheek teeth as well as older donkeys' cheek teeth (3;29) are the same pulp combinations that are predisposed to become exposed simultaneously even in older cheek teeth.

Occlusal pulpar exposure was more frequently present with some idiopathic fracture patterns. Additionally, the combinations of pulps that became simultaneously exposed was usually related to the type of fracture pattern. Mandibular cheek teeth with miscellaneous fracture patterns had 83% of their pulp horns exposed at the occlusal surface. These particular fracture patterns often greatly disrupt the normal anatomy, and can involve both buccal and lingual pulp horns in the fracture plane (6). Maxillary cheek teeth with midline sagittal fractures area also predisposed to developing apical infections and thus multiple pulpar exposures (40% pulpar exposure in this study), because the fracture line through carious infundibula exposes deep endodontic structures and later there is invariably much movement of the two fragments and so many of these fractures teeth develop apical infection, including 5/5 (100%) recorded by Dacre et al (6) and 7/11 (64%) recorded by Dixon et al. (7).

In contrast, maxillary and mandibular lateral slab fractures and maxillary miscellaneous fractures are less prone to develop occlusal pulpar exposure (25%, 25% and 29% prevalence of occlusal exposure, respectively, were found in this study). The lateral slab fracture planes involved the two buccal pulp horns only and it is likely that reparative cells in the affected pulp horns have more opportunity to seal off this more localised inflammation. If infection of the exposed pulp horns persisted, it could spread to the contra-lateral pulp horns and consequently, the medial pulp horns were found to become exposed in maxillary and mandibular lateral slab fracture patterns (Fig. 30). The maxillary miscellaneous fracture pattern usually involved palatal pulp horns and exposure, if present, was limited to single pulp horns.

6. Conclusions

This study has confirmed the presence of occlusal pulpar exposure in 32 % of apically infected cheek teeth and further re-enforces the value of detailed intra-oral examination of such cases. Very significantly, 10% of apically infected cheek teeth had changes to the occlusal secondary dentine termed occlusal pitting, but did not have pulpar exposure. The patterns of multiple pulpar exposures with cheek teeth apical infections that reflect anatomic relationships are described. A higher proportion (42%) of cheek teeth extracted because idiopathic fractures had pulpar exposure but only (3%) had occlusal pitting. Apical infections were attributed to anachoresis in 68%. Other recorded infection routes included periodontal disease (23%), caries (4%), fissure fractures (3%) and dysplasia (3%). The recorded fracture patterns corresponded with previously described patterns and the type of pattern related to the extent of pulpar damage. Occlusal pulp exposure more commonly occurred with midline sagittal maxillary and miscellaneous pattern mandibular cheek teeth fractures.

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