Prevalence of occlusal pulpar exposure in 110 equine cheek teeth with apical infections and idiopathic fractures

M.S.D. van den Enden, P.M. Dixon *

Royal (Dick) School of Veterinary Studies, University of Edinburgh, Department of Clinical Science, University of Edinburgh, Midlothian EH25 9RG, UK

Abstract

Examination of 110 cheek teeth (CT) that were clinically extracted (between 2004 and 2008) because of apical infection ($n = 79$; mean dental age 3.5 years) or idiopathic CT fractures ($n = 31$; median dental age 8.5 years), including examinations of transverse and longitudinal sections, showed the apical infections to be mainly (68%) due to anachoresis, with the residual cases caused by periodontal spread, infundibular caries spread, fissure fractures and dysplasia.

The idiopathic fracture patterns were similar to previously described patterns. Occlusal pulpar exposure was found in 32% of apically infected CT, including multiple pulps in 27% and a single pulp in 5%. However, 10% of apically infected CT had changes to the occlusal secondary dentine, termed occlusal pitting, but did not have exposure of the underlying pulp. Multiple pulpar exposures occurred in some CT with apical infections, and the combination of pulp involvement reflects the anatomical relationships of these pulps. A higher proportion (42%) of CT extracted because idiopathic fractures had pulpar exposure (26% multiple, 16% single pulps), especially with midline sagittal maxillary and miscellaneous pattern mandibular CT fractures, but only (3%) had occlusal pitting.
© 2008 Elsevier Ltd. All rights reserved.

Keywords: Equine teeth; Occlusal pulpar exposure; Apical infection; Dental fractures

Introduction

Occlusal pulpar exposure in equine cheek teeth (CT) was recorded both in earlier studies of general horse populations (Rupprecht, 1936; Herzog, 1937; Gnädinger, 1947; Wafa, 1988) and in more recent studies of referred equine populations (Dixon et al., 2000; van Hoofd, 2005; Dacre et al., 2008a,b; du Toit et al., 2008a). Odontoblasts normally lay down secondary dentine (regular and irregular) that continues to seal off the occlusal aspect of the pulp horns throughout the prolonged eruption of equine CT (Dacre et al., 2008a,b). If this occlusal secondary dentine does not effectively seal off the pulp from the oral cavity, molecules, micro-organisms and pH changes from the oral environment cause pulpar inflammation.

In brachydont teeth, such inflammation within the rigid confines of the pulp chamber commonly compresses the fine apical and pulpar vasculature and causes ischaemic death of the pulp, however the wider apical foramina and larger vasculature of equine (hypsodont) teeth can help resist such ischaemia (Dixon and Dacre, 2005). Following exposure of pulp to the oral environment, bacterial infection may spread down through the pulp and extend out through the apical foramina causing apical (periapical) infection. Thus bacterial pulpitis and apical infection can be directly established through a primary pulpar exposure, such as following fracture of the clinical crown (Wafa, 1988; Mueller and Lowder, 1998; Crabill and Schumacher, 1998; Dixon and Dacre, 2005; Dacre et al., 2008c,d).

Alternatively, an insult to vital pulp (and consequently to its odontoblasts) within an anatomically intact pulp horn, such as by a blood-born bacterial infection (anachoresis), may result in a local or generalised decrease or cessation of secondary dentine formation in that pulp,

* Corresponding author. Tel.: +44 131 6506242; fax: +44 131 6506599.
  E-mail address: p.m.dixon@ed.ac.uk (P.M. Dixon).

depending on the size and nature of the pulpar lesion (Dacre et al., 2008b,c,d). As the remaining secondary dentine is progressively worn away on the occlusal surface, the underlying pulp chamber that contains devitalised or dead pulp is eventually exposed. Such an event can be termed a secondary occlusal pulpar exposure (Wafa, 1988; Dixon et al., 2000; Dixon and Dacre, 2005; Dacre, 2005). Recent studies (Dacre et al., 2008c,d; du Toit et al., 2008a) have shown most equine CT apical infections to be caused by anachorectic pulpitis rather than by primary pulpar exposure, and secondary occlusal pulpar exposure often occurs in such CT.

The objectives of this study were (1) to establish the prevalence of occlusal pulpar exposure in CT that were surgically extracted because of apical infection or idiopathic CT 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 CT to assess possible relationships between patterns of occlusal pulpar exposure and the type of apical infection or fracture patterns that necessitated CT extraction.

Materials and methods

A total of 110 CT extracted or repulsed at The University of Edinburgh Equine Hospital (98 CT extracted between 2004 and 2008) were used. Complete clinical and ancillary histories were available for 88 specimens. These teeth included 79 apically infected CT without any obvious gross cause of infection (e.g. no gross fracture; or marked periodontal disease such as due to diastema or displacements) of median *dental* age (i.e. time since eruption) of 3.5 years (range −0.5 to 25 years) and 31 CT with idiopathic fractures of median dental age of 8.5 years (range 1–20 years). Cheek teeth with idiopathic fractures that had concurrent apical infections were retained in the idiopathic fracture classification group.

The idiopathic CT fractures were categorised into five described fracture patterns (Dacre et al., 2007). To further assess changes, 65 apically infected and 11 fractured CT were transversely sectioned and four apically infected CT were sectioned longitudinally using a tile saw as previously described (Dacre et al., 2008a; du Toit et al., 2008b) and the dental

sections were visually examined. The pulp nomenclature system of du Toit et al. (2008b) was used in this study.

Results

Triadan positions of CT with apical infections and idiopathic fractures

In the 88 fully identified CT, the more central Triadan positions (07, 08 and 09) were over-represented. Thirty percent of apically infected CT were Triadan 07s, 25% Triadan 08s and 23% Triadan 09s. Maxillary CT were more commonly fractured (68% of all fractured CT) than mandibular CT (32%), with maxillary 09s preferentially affected (Triadan 07s comprised 13%, Triadan 08s comprised 25% and Triadan 09s comprised 57% of all fractured maxillary CT).

Cheek teeth with apical infections

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 as the underlying pulp cavity (Fig. 1). However, the majority of occlusal defects had some secondary dentine remaining that was usually stained brown to black (Fig. 2). The primary dentine lying adjacent to exposed pulps usually remained normal, retaining its cream-coloured translucent appearance with staining rarely present (Figs. 1 and 2). The larger occlusal defects were readily identified on visual examination and
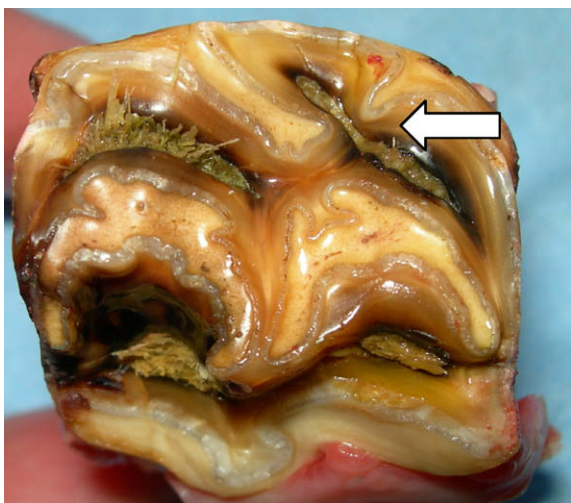

Fig. 1. Occlusal aspect of an apically infected maxillary CT with occlusal exposure of all five pulps that have food protruding from them. Note the coalescence of the 3rd and 5th pulp horns.

Fig. 2. Occlusal surface of an apically infected mandibular CT with occlusal exposure of the 1st and 2nd pulp horns (with a probe in 2nd pulp horn). The exposed pulps have a thin, shell-like covering of secondary dentine that has been perforated at their central aspects. The other three pulp horns are fully covered with normal appearing secondary dentine with scratch marks from a metal probe that was used to assess their integrity.

a 2 mm wide probe could easily be introduced into the patent pulp horn from the occlusal surface (Fig. 2). In contrast, a probe could only be advanced a few millimetres into some shallow occlusal secondary dentine indentations or occlusal pulpar exposure lesions of a small diameter.

Transverse or longitudinal sections of eight CT (10% of apically infected CT) with shallow occlusal secondary dentine lesions showed that the underlying pulp remained protected by a deeper overlying layer of pitted but intact dentine and these superficial lesions were termed *occlusal pitting* of secondary dentine. In other specimens, a discrete occlusal defect led to a grossly infected or even food-filled pulp horn more apically and these CT were defined as having occlusal pulpar exposure. Examination of transversely and longitudinally sectioned CT showed decreased translucency and discoloration of pulps with occlusal exposure and in more chronically infected CT, necrosis or absence of pulp, with the hollow cylindrical pulp horns impacted with food material in some (Fig. 3).

Aetiopathological findings in apically infected CT

No physical entry route for oral bacteria into the endodontic system could be identified and therefore a blood or lymph borne infection (anachorectic infection) was considered to be the most likely cause of infection in 54/79 (68%) of apically infected CT, that had a median dental age of 3.5 years. Although 19/54 CT (35%) had pulpar exposure, there was no evidence to suggest that this pulpar exposure was the *cause* of infections, and all occlusal exposure was deemed to be secondary to pulpar death as also assessed by Dacre et al. (2008c,d).

Eighteen (22%) of the 79 apical infections (median dental age 11 years), were believed to have been caused by descending periodontal disease, as characterised by a continuous tract from the occlusal surface to the apex on the tooth periphery, with localised loss of periodontal membranes and erosion of the underlying cementum. This was distinguished from ascending periodontal disease (for example, secondary to an anachoretic apical infection), which was often characterised by reactive cementum deposition with chronic infection, and largely intact periodontal ligaments at the gingival level in some CT (Dacre et al., 2008c,d).

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

Fissure fractures were found in three apically infected CT (all mandibular). These were subtle lesions, with staining along the fissure line only evident on examination of transversely sectioned teeth. In one CT, the fracture only involved peripheral cementum, but in the other two CT (3%) (dental ages 4 and 6 years) the fracture extended to involve the buccal pulp horns (numbers 1 and 2) that were necrotic, indicating that the fracture was the likely infection route.

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

Prevalence of occlusal pulpar exposure in cheek teeth with apical infections

Occlusal pulpar exposure was present 25 CT (32%) with apical infections, with multiple pulps exposed in 21 (27%)

Fig. 3. Mid-tooth transverse section of an apically infected maxillary CT that had all pulps occlusally exposed. All five pulp horns are filled with food and there is some discoloration, but no gross caries of the circumpulpar dentine. Some artefactual fractures are present in this section.

Table 1
Number, median dental ages and prevalence of occlusal pulpar exposure in 79 cheek teeth (CT) with apical infections as identified for each route of infection.

Infection route	Number of CT ($n = 79$)	Median dental age (years)	Occlusal pulpar exposure		
			Total	Multiple	Single
Anachoresis	54/79 (68%)	4	19/54 (35%)	16/54 (30%)	3/54 (6%)
Periodontal disease	18/79 (23%)	11	3/19 (16%)	2/19 (11%)	1/19 (5%)
Infundibular caries	3/79 (4%)	3.5	1/4 (25%)	1/4 (25%)	0
Fissure fracture	2/79 (3%)	5	0	0	0
Dysplasia	2/79 (3%)	2.5	2/2 (100%)	2/2 (100%)	0

CT and a single pulp exposed in four CT (5%). Three of these CT also had occlusal pitting of secondary dentine overlying other pulp horns. Occlusal pitting of secondary dentine, without pulpar exposure of any horns, occurred in eight CT (10%), with four (5%) CT showing pitting of multiple pulp horns and four (5%) CT having such changes over a single pulp horn (5%). The prevalence of occlusal pulpar exposure for each proposed cause of infection is summarised in Table 1.

Exposure of specific pulp horns with CT apical infections

Because equine maxillary and mandibular CT have different endodontic anatomy (du Toit et al., 2008b; Dacre et al., 2008a) data on exposure of the individual pulp horns in maxillary and mandibular CT are therefore presented separately (Table 2) using the pulp horn nomenclature of du Toit et al. (2008b). The caudo-medial pulp horn was most frequently exposed in both upper and lower CT, i.e. pulp 4 was affected in 10/14 (71%) occlusally exposed maxillary CT and pulp 5 in 9/11 (82%) occlusally exposed mandibular CT. No occlusal exposure of pulps 6, 7 or 8 was found in this study.

In some CT, exposure of multiple pulps occurred in various combinations (Fig. 4), with simultaneous exposure of pulps 3 and 5 (Fig. 1) the most commonly recorded pattern in maxillary CT, i.e. present in 7/12 (58%) upper CT with multiple exposures. In contrast, pulps 2 and 5 were most frequently simultaneously exposed in mandibular CT, i.e. in 7/9 (78%) lower CT with multiple pulp exposures.

Table 3
Prevalence of idiopathic cheek teeth (CT) fracture patterns in 31 fractured CT.

	Maxillary cheek teeth		Mandibular cheek teeth	
	Number of CT	Percentage maxillary CT	Number of CT	Percentage mandibular 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	

Max, maxillary; Man, mandibular.

Cheek teeth with idiopathic fractures

Exposed pulp horns/gross examination

Most fracture planes ran through pulp horns and provided longitudinal views of the fractured pulp horns with most of these appearing to contain a normal amount of secondary dentine. However, the fractured pulp horns of

Table 2
Exposure of individual pulp horns (expressed as percentages of total occlusal pulpar exposure) in apically infected maxillary (Max) and mandibular (Man) cheek teeth (CT). Note that data in horizontal rows are not cumulative, since multiple pulps can be simultaneously exposed.

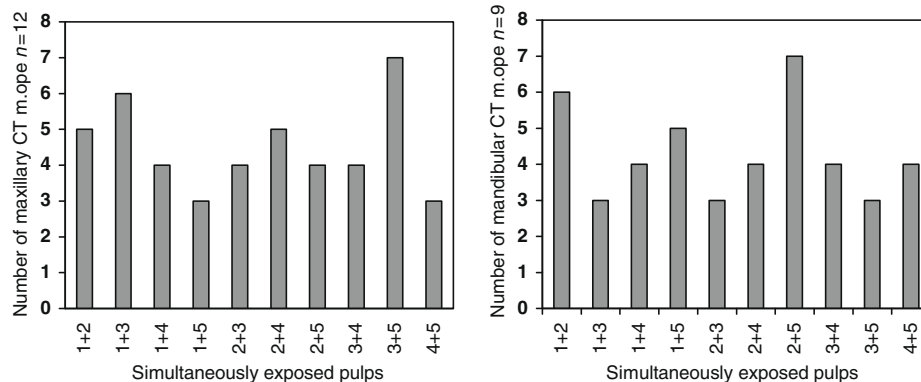|	Occlusal pulpar exposure			Pulp 1	Pulp 2	Pulp 3	Pulp 4	Pulp 5	Pulp 6	Pulp 7	Pulp 8
	Total	Multiple	Single								
Max CT	14	12	2	8 (57%)	7 (50%)	7 (50%)	10 (71%)	7 (50%)	0	0	0
Man CT	11	9	2	4 (36%)	5 (45%)	7 (64%)	7 (64%)	9 (82%)	0	0	–
Total	25	21	4								

Fig. 4. Histogram identifying the pulps that are simultaneously exposed in maxillary and mandibular apically infected CT (m.ope, multiple occlusal pulpar exposure).

three CT 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. (2007), although some such changes could represent carious changes that occurred following fracture.

Fracture patterns

The idiopathic fracture patterns present always involved one or more pulp horns and/or infundibula and corresponded with the five patterns described by Dacre et al. (2007) (Table 3). The midline sagittal fracture pattern (through both infundibula) was the most common pattern in maxillary CT (48% of maxillary CT fractures), whilst the miscellaneous fracture pattern was the most frequently recorded pattern in mandibular CT (60% of mandibular CT fractures).

Table 4
Prevalence of occlusal pulpar exposure and pitting with five different idiopathic fracture patterns.

	Single pulpar exposure	Multiple pulpar exposure	Single pitting	Prevalence pulpar exposure
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	

Max, maxillary; Man, mandibular.

Fig. 5. Fragments of a mandibular CT suffering from a miscellaneous fracture pattern showing a probe inserted into an occlusally exposed pulp horn that is not involved in fracture plane.

Fig. 6. Mandibular CT with lateral 'slab' fracture (running through 1st and 2nd pulp horn) showing gross occlusal exposure of the other three pulp horns (pulps 3–5).

Prevalence of occlusal pulpar exposure in cheek teeth with idiopathic fractures

Occlusal pulpar exposure was present in 13/31 fractured CT (42%), involving multiple pulps in eight (26%) and a single pulp in five CT (16%). Pitting of occlusal secondary dentine was found only over one pulp horn (3%) in a maxillary CT. Occlusal pulpar exposure occurred in all fracture patterns (Table 4, Figs. 5 and 6) with the highest prevalence in mandibular CT with miscellaneous fractures (5/6, 83%); and maxillary CT midline sagittal fractures (4/10, 40%).

Exposure of specific pulp horns in cheek teeth with idiopathic fractures

Maxillary pulps 1, 3 and 5 were all exposed with the same frequency (43%) in maxillary CT with occlusal pulpar exposure (Table 5), whilst mandibular pulp 5 was exposed most commonly (5/6; 83%) in mandibular CT with occlusal pulpar exposure. Occlusal exposure of pulps 6, 7 and 8 were not found.

In CT with multiple pulpar exposures, the combinations of exposed pulp horns differed between different fracture patterns (Fig. 7). Palatal and buccal pulp chambers were concurrently exposed only in maxillary CT 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 (Table 5, Fig. 7). Simultaneous pulpar exposure was less uniform in the mandibular miscellaneous fracture pattern.

Discussion

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. Therefore, the term occlusal defect would be more appropriate to use to describe both occlusal pulpar exposure and occlusal pitting defects. Clinical diagnosis of these subtle lesions poses a challenge, because occlusal examination is compromised by the limited access

Table 5
Exposure of individual pulp chambers, expressed as percentages of total number of pulps occlusally exposed in maxillary (Max) and mandibular (Man) cheek teeth (CT) with idiopathic fractures.

	Occlusal pulpar exposure			Pulp 1	Pulp 2	Pulp 3	Pulp 4	Pulp 5	Pulp 6	Pulp 7	Pulp 8	Pulp Unknown
	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	8	5									

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 (Simhofer et al., 2008), are required to accurately identify such occlusal lesions.

Healthy pulp chambers should never become occlusally exposed (Kilic et al., 1997) and therefore the presence of occlusal pulpar is evidence that severe pulpar disease has occurred in the past (Dacre et al., 2008c,d). 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) (Dacre, 2005) between vital pulp and the oral environment. In some maxillary CT with lateral slab fractures, the larger palatal fracture fragments remain stable, without signs of clinical apical infections or pulpar exposure, indicating that the two exposed pulps have become sealed off (Dacre et al., 2007; Taylor and Dixon, 2007; Dixon et al., 2007).

Lesions of occlusal secondary dentine, including pulpar exposure is occasionally found in asymptomatic equine CT (Wafa, 1988; du Toit et al., 2008a). Consequently, the presence of lesions in the occlusal secondary dentine, but this 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 CT in Figs. 1–3, 5 and 6. In these advanced cases however, the accompanying clinical signs and evidence from diagnostic imaging are so marked that the clinical detection of occlusal pulpar exposure is often not the decisive diagnostic factor.

It would appear that three different scenarios can occur with pulpar insult. Firstly, with extensive pulpar damage (e.g. ischaemic or bacterial insult) the pulp in one or more pulp horns dies, no secondary (or tertiary) dentine can be laid down and, with further dental eruption, occlusal aspects of dead pulp horns 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 dentine, grossly characterised by having an irregular, partial occlusal defect, develops on the occlusal aspect of the pulp horn, and this imperfect secondary dentine will clinically appear as occlusal pitting of secondary dentine. However, the pulp remains sealed off from the oral environment, remains viable below this level, and later produces normal secondary dentine. Thirdly, extensive and irreversible inflammation that is localised to the occlusal 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 (Dacre et al., 2008d; du Toit et al., 2008a). This will grossly manifest itself as an apparent total occlusal defect, that on deeper probing is found to be of limited depth and viable pulp remains deep to the tertiary dentine. A variation is when the whole endodontic system is occlusally exposed and becomes sealed off more apically by reactive calcified tissue as shown in an asymptomatic CT in Fig. 8. Histological examination of 'pitting' dentine should cast some further information on this disorder.

Occlusal pitting was observed in 10% of apically infected CT in this study. Likewise, Wafa (1988) identified defects in occlusal secondary dentine without communication to the underlying pulp chambers in 14 CT of 355 equine skulls (circa 0.16% of CT) that also showed no periapical changes, confirming the absence of apical infection.

The causes of apical infection in this study were similar to those described by Dacre et al. (2008c,d) with anachoretic
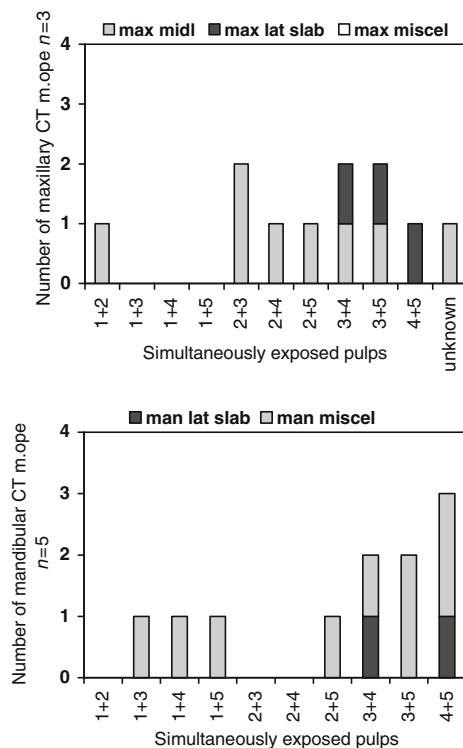

Fig. 7. Histogram identifying the pulps that are concurrent occlusally exposed in maxillary and mandibular CT with idiopathic fractures. No multiple occlusal exposures (m.ope) were present in maxillary CT with the miscellaneous fracture pattern.
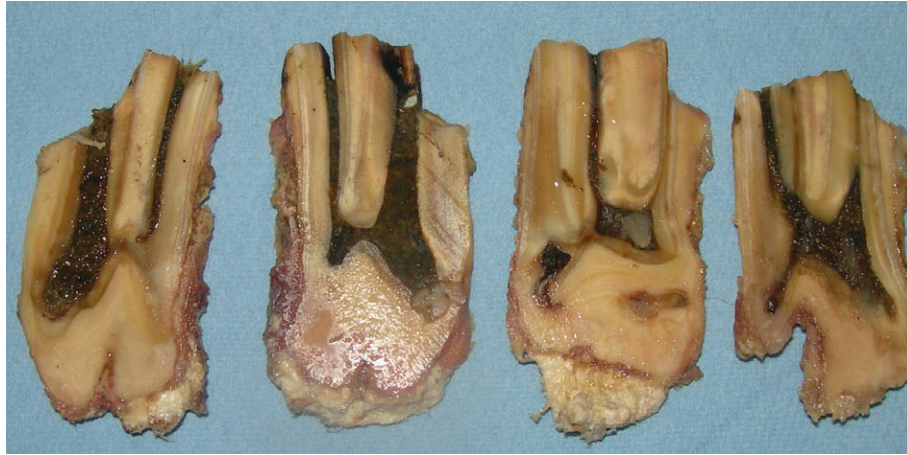
Fig. 8. Longitudinal sections of a 209 (not included in this study) that was extracted at post-mortem examination from a 9-year old horse (dental age 8 years) that showed no clinical or post-mortem evidence of apical infection. This tooth has multiple pulpar exposures. All pulp horns and a common pulp chamber are filled with food, yet the exposed endodontic system is fully sealed off from the apical area by reactive calcified tissue.

infection the main cause, and periodontal disease, infundibular caries, fissure fractures and dysplasia all less common causes. In CT 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 CT 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 CT are often hyperaemic, these tissues may be particularly susceptible to blood borne bacterial invasion (Crabill and Schumacher, 1998; Dixon et al., 2000). Additionally, young CT may still a have a common pulp chamber or have large communications between pulp horns (Kirkland et al., 1996), which could facilitate spread of infection to multiple pulps and this lead to multiple pulpar exposures.

Descending periodontal disease is reported to affect preferentially the lingual aspect of mandibular and the buccal aspect of maxillary CT interproximal spaces (Baker, 1970; Mueller and Lowder, 1998; Crabill and Schumacher, 1998). Becker (1962) proposed that pulpar exposure in apically infected CT of periodontal disease origin would be limited to adjacent pulp horns. However, in the current study, multiple pulps were usually 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, the multiple pulpar exposures found in these CT 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 CT. This is most likely due to their anatomical communications, which are more marked in young CT. Maxillary pulps 3 and 5 are reported to most frequently anatomically communicate in horses (Dacre, 2005) and donkeys (du Toit et al., 2008b). In mandibular CT, most pulp communications are between pulps 2 and 5, both in horses (Dacre, 2005) and donkeys (du Toit et al., 2008b).

As a result of the ongoing circumferential replacement of pulp by secondary dentine, the common pulp chamber and individual pulp horns become gradually smaller as the tooth matures. It has been proposed that the common pulp chamber remains in equine mandibular CT for 4–5 years following eruption and that two separate pulp chambers with individual pulp horns can be identified after 6 years. (Kirkland et al., 1996). The pulp communications present in younger equine CT as well as older donkeys' CT (Dacre et al., 2008a; du Toit et al., 2008b) are the same pulp combinations that are predisposed to become exposed simultaneously even in older CT.

Occlusal pulpar exposure was more frequently present with some idiopathic fracture patterns. Additionally, the combination of pulps that became simultaneously exposed was sometimes 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 (Dacre et al., 2007). Maxillary CT with midline sagittal fractures are 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 fractured teeth develop apical infection-including 5/5 (100%) recorded by Dacre et al. (2007) and 7/11 (64%) recorded by Dixon et al. (2007).

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 can 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 (Table 3, Fig. 6). The maxillary miscellaneous fracture pattern usually involved palatal pulp horns and exposure, if present, was limited to single pulp horns.

Conclusions

This study has confirmed the presence of occlusal pulpar exposure in 32% of apically infected CT and further re-enforces the value of detailed intra-oral examination of such cases. Very significantly, 10% of apically infected CT have changes to the occlusal secondary dentine termed *occlusal* pitting, but do not have pulpar exposure. The patterns of multiple pulpar exposures with CT apical infections that reflect anatomical relationships are described. A higher proportion (42%) of CT extracted because idiopathic fractures had pulpar exposure but only (3%) had occlusal pitting. Occlusal pulp exposure more commonly occurred with midline sagittal maxillary and miscellaneous pattern mandibular CT fractures.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

References

Baker, G.J., 1970. Some aspects of equine dental disease. Equine Veterinary Journal 2, 105–110.

Becker, E., 1962. Zahne. In: Dobberstein, J., Pallaske, G., Stunzi, H., Band, V. (Eds.), Handbuch der speziellen pathologischen Anatomie der Haustiere. Paul Parey, Berlin, pp. 246–292.

Crabill, M.R., Schumacher, J., 1998. Pathophysiology of acquired dental diseases of the horse. Veterinary Clinics of North America: Equine Practice 14, 291–307.

Dacre, I.T., 2005. A pathological, histological and ultrastructural study of diseased equine cheek teeth, Ph.D. Thesis. The University of Edinburgh, Edinburgh, pp. 1–324.

Dacre, I.T., Kempson, S.A., Dixon, P.M., 2007. Equine idiopathic cheek teeth fractures. Part 1: pathological studies on 35 fractured cheek teeth. Equine Veterinary Journal 39, 310–318.

Dacre, I.T., Kempson, S.A., Dixon, P.M., 2008a. Pathological studies of cheek teeth apical infections in the horse: 1. Normal endodontic anatomy and dentinal structure of cheek teeth. The Veterinary Journal 178 (3), 311–320.

Dacre, I.T., Shaw, D.S., Dixon, P.M., 2008b. Pathological studies of cheek teeth apical infections in the horse: 3. Quantitative measurements of dentine in apically infected cheek teeth. The Veterinary Journal 178 (3), 333–340.

Dacre, I.T., Kempson, S.A., Dixon, P.M., 2008c. Pathological studies of cheek teeth apical infections in the horse: 4. Aetiopathological findings in 41 apically infected mandibular cheek teeth. The Veterinary Journal 178 (3), 341–351.

Dacre, I.T., Kempson, S.A., Dixon, P.M., 2008d. Pathological studies of cheek teeth apical infections in the horse: 5. Aetiopathological findings in 57 apically infected maxillary cheek teeth and histological and ultrastructural findings in apically infected cheek teeth. The Veterinary Journal 178 (3), 352–363.

Dixon, P.M., Dacre, I.T., 2005. A review of equine dental disorders. The Veterinary Journal 169, 165–187.

Dixon, P.M., Barakzai, S., Collins, M., Yates, J., 2007. Equine idiopathic cheek teeth fractures. Part 3: A hospital-based survey of 68 referred horses (1999–2005). Equine Veterinary Journal 39, 327–332.

Dixon, P.M., Tremaine, W.H., Pickles, K., Kuhns, L., Hawe, C., McCann, J., McGorum, B.C., Railton, D.I., Brammer, S., 2000. Equine dental disease. Part 4: A long-term study of 400 cases: apical infections of cheek teeth. Equine Veterinary Journal 32, 182–194.

du Toit, N., Burden, F.A., Kempson, S.A., Dixon, P.M., 2008a. Pathological investigation of caries and occlusal pulpar exposure in donkey cheek teeth using computerised axial tomography with histological and ultrastructural examinations. The Veterinary Journal 178 (3), 387–395.

du Toit, N., Kempson, S.A., Dixon, P.M., 2008b. Donkey dental anatomy. Part 1: Gross and computed axial tomography examinations. The Veterinary Journal 176, 338–344.

Gnädinger, F., 1947. Erhebungen über die Häufigkeit von Zahnfehlern beim Pferd. Hannover. Cited by Becker, E., 1962. Zahne. In: Dobberstein, J., Pallaske, G., Stunzi, H., Band, V. (Eds.), Handbuch der speziellen pathologischen Anatomie der Haustiere, Paul Parey, Berlin.

Herzog, K., 1937. Statistische Erhebungen über Zahnerkrankungen beim Pferde. Leipzig. Cited by Becker, E., 1962. Zahne. In: Dobberstein, J., Pallaske, G., Stunzi, H., Band, V. (Eds.), Handbuch der speziellen pathologischen Anatomie der Haustiere, Paul Parey, Berlin.

Kilic, S., Dixon, P.M., Kempson, S.A., 1997. A light microscopic and ultrastructural examination of calcified dental tissues of horses: 3. Dentine. Equine Veterinary Journal 29, 206–212.

Kirkland, K.D., Baker, G.J., Marretta, S.M., Eurell, J.C., Losonsky, J.M., 1996. Effects of aging on the endodontic system, reserve crown, and roots of equine mandibular cheek teeth. American Journal of Veterinary Research 57, 31–38.

Mueller, P.O., Lowder, M.Q., 1998. Dental sepsis. Veterinary Clinics of North America: Equine Practice 14, 349–364.

Rupprecht, K., 1936. Statistische Erhebungen über die Häufigkeit von Gebißanomalien beim Pferde. Leipzig. Cited by Becker, E., 1962. Zahne. In: Dobberstein, J., Pallaske, G., Stunzi, H., Band, V. (Eds.), Handbuch der speziellen pathologischen Anatomie der Haustiere, Paul Parey, Berlin.

Simhofer, H., Griss, R., Zetner, K., 2008. The use of oral endoscopy for detection of cheek teeth abnormalities in 300 horses. The Veterinary Journal 178 (3), 396–404.

Taylor, L., Dixon, P.M., 2007. Equine idiopathic cheek teeth fractures. Part 2: A practice-based survey of 147 affected horses in Britain and Ireland. Equine Veterinary Journal 39, 322–326.

van Hoofd, M., 2005. No teeth no horse; Inventarisatie van macroscopische en microscopische afwijkingen aan elementen die verwijderd zijn, op basis van verdenking van een alveolitis, bij het paard. Dissertation. Utrecht University, Utrecht, pp. 17–23.

Wafa, N.S.Y., 1988. A study of dental disease in the horse, Ph.D. Thesis. National University of Ireland, University College Dublin, Dublin.