Proceedings of the American Association of Equine Practitioners - Focus Meeting

Focus on Dentistry

Albuquerque, NM, USA – 2011

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Acquired Disorders of Equine Teeth

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Introduction

Equine dental disorders are of major importance, with a British survey showing that 10% of equine practice time is spent on dental-related work.1 Likewise, a U.S. survey showed dental disorders to be the third most common equine medical problem encountered by large animal practitioners.2 Despite its importance, equine dentistry has until recently been a neglected area, with postmortem studies indicating up to 80% prevalence of undiagnosed, clinically significant dental disorders in equids.4-8 For example Honma et al. found that all horses > 12 years of age had dental caries4; Baker9 found that 60% of horses >15 years old had periodontal disease and 79% had infundibular caries. More recently, a high prevalence of dental disorders, and in particular CT diastemata, was demonstrated in aged donkeys in a postmortem survey with some associated with fatal colics.7,10

Abnormalities of Wear

Normal tooth wear (attrition) occurs when the occlusal surfaces of opposing teeth come into occlusion and grind off each other. Any asymmetry in the position of the jaws (e.g. as occurs with developmental cranio-facial abnormalities) or of individual teeth (e.g. with hypodontia (too few), polydontia (too many) or displacements will result in uneven dental wear. The periodontal membranes adjacent to overgrown teeth can become diseased, due to abnormal drifting of overgrown teeth causing diastemata. Additionally, the pain and possibly mechanical obstruction caused by dental overgrowths can restrict masticatory movements that in turn will restrict intra-oral saliva and food movements. This can lead to peripheral caries, periodontal disease11 and occasionally, local calculus formation.

Overgrowths can also cause soft tissue trauma which also can lead to clinical signs such as biting abnormalities in ridden horses and less commonly quidding.

Cheek Teeth Overgrowths (“Enamel Points”)

The presence of anisognathia (jaws of unequal widths) and also having maxillary cheek teeth (CT) that are wider than their mandibular counterparts contributes to the development of enamel overgrowths on the buccal aspect of the maxillary and lingual aspect of the mandibular CT in equids. These overgrowths lead to buccal mucosal ulceration (rarely to tongue ulceration) and in severe cases may cause biting problems and quidding. The lateral aspect of maxillary cheek teeth of some horses have very exaggerated vertical ridges (cingulae) that predispose to the development of focally sharp areas. Horses on a forage diet have wide masticatory movements in contrast to a predominantly vertical crushing stroke, when high levels of concentrates are fed12.
that, along with the reduced amount of time spent masticating concentrates, promotes the development of CT enamel overgrowths and increased CT occlusal angles. However some horses that never receive concentrates will develop sharp maxillary cheek teeth overgrowths with buccal ulcers opposite the caudal maxillary teeth. Enamel overgrowths predominantly cause clinically significant disease in ridden horses, especially when associated with the use of tight nosebands. There is however still some debate on the need to remove tall and sharp overgrowths in horses without buccal ulceration and factual research is needed in this area.

Shear Mouth

If the above noted generalized CT overgrowths are not reduced by routine dental treatment, they may increase to such an extent that they interfere with the normal lateral masticatory action which further perpetuates overgrowths and may lead to a condition termed shear mouth in some horses. It was formerly believed that cheek teeth with occlusal angles of >15° could be termed shear mouth, but it is now accepted that the caudal mandibular teeth normally have angles of up to 30°13 and shear mouth represents much higher (>45°) occlusal angles.14,15 Horses affected with shear mouth have reduced effectiveness at grinding food on the affected side, especially when fed dried forage such as hay, and may later exhibit quidding, due to soft tissue injury and to the inevitable painful periodontal disease that accompanies this disorder.

Wave Mouth

Wave mouth is the presence of an undulating occlusal surface of the cheek teeth row in a rostro-caudal direction. Differential rate of CT eruption between opposing CT may be a cause of wave-mouth (that may even increase with time),15 as has the presence of large focal overgrowths (e.g. due to absence of or defective opposing teeth).15 Severe wave mouth can cause restricted mastication, and multiple concurrent dental (e.g. shear mouth or diastemata) and periodontal disorders will inevitably develop later.

Step Mouth

The loss of a cheek tooth can cause a rectangular shaped overgrowth, i.e. stepmouth due to increased eruption and absence of wear of the unopposed ipsilateral tooth. However, dental drift of the teeth adjacent to the extracted tooth may in time, cause the overgrowth to become more triangular in shape.16 Step mouth can also be caused by CT maleruptions, such as different rates of eruption of opposing CT, with the earliest erupted CT becoming and remaining overgrown (“dominant”). Less severe CT maleruptions may lead to wave mouth and there is often an overlap between these two disorders. These overgrowths can mechanically interfere with normal mastication leading to wavemouth or shear mouth.

The maxillary CT of older horses with worn infundibulae (especially the 09s that erupt first and also commonly have infundibular cemental hypoplasia), or maxillary CT with developmentally short infundibulae or with infundibular caries, have reduced enamel content and thus wear resistance that allows the opposite mandibular CT to overgrow. Likewise, older horses can have reduced peripheral enamel infolding of their mandibular CT and so will develop overgrowths of the opposite maxillary CT.

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Smooth Mouth

In older equids the reduction and eventual loss of peripheral and infundibular enamel is a normal end-stage of dental attrition. This leads to the presence of a smooth occlusal surface containing predominantly cementum (due to increased deposition around the residual roots) and dentine, with minimal areas of protruding enamel. This condition is commonly termed smooth mouth. The residual dentine and cementum are no longer protected from normal attrition by the presence of adjacent (harder) enamel and such teeth are ineffective at grinding, and rapidly wear further. Smooth mouth can develop in younger dysplastic teeth where there is insufficient or poorly distributed enamel peripheral or infundibular enamel or enamel dysplasia.

Diastemata

As noted in the developmental disorder section, diastemata (a detectable interdental space between adjacent teeth) can be termed primary (developmental – inadequate compression) or secondary. Secondary diastemata can develop beside developmental or acquired displacements (usually medial (lingual) displacements of lower 10s and 11s), supernumerary cheek teeth or dental loss. Because equine CT slightly taper towards their apices and the angulated 06s, 10s and 11s lose their angulation with age, senile diastemata commonly develop between many CT in aged horses. With marked food entrapment, the periodontal disease progresses to cause lysis and remodeling of alveolar bone and less commonly apical infection, osteomyelitis of the mandible or maxillae or oromaxillary fistula formation. Quidding is the most common clinical sign with CT diastemata, which is regarded as one of the most painful dental disorders of horses. Open mouth radiography can help assess the cause, severity and prognosis with CT diastemata. In younger horses with this disorder, further eruption of the CT and compression of the CT rows may even result in resolution of the diastemata, provided there is sufficient CT angulation.

Disorders of Pulp

Pulpitis

Because of the intimate relationship between dentine and pulp, these two tissues can be termed the dentino-pulp complex, which emphasises the fact that an insult to dentine can insult pulp and the reverse also holds true. In human teeth, pulpitis (inflammation of the pulp) occurs most commonly secondary to caries that has penetrated the enamel. Other potential causes of pulpitis in any species include pulpar exposure secondary to accidental or iatrogenic trauma (e.g. due to use of motorized dental equipment or dental shears).

The inflammatory response by pulp includes the development of pulpar oedema and the influx of inflammatory cells. Because pulp is encased in a rigid dentine chamber, such an inflammatory response increases pressure in the affected pulp that can collapse its vasculature. This can cause pulpar hypoxia that may lead to localised or generalized pulpar necrosis and death. However, equine teeth especially young teeth, have large apical foramina and a large blood supply to their pulp (which allow lifelong deposition of subocclusal dentine), and so equine teeth may resist a
degree of pulpar oedema and inflammation that would cause ischaemic pulpar death in brachydont teeth. If an exposed equine dental pulp survives the inevitable oedema, and also the invasion by oral bacteria, tertiary dentine formation will seal off the area of insulted pulp from the healthy underlying pulp and hopefully result in complete resolution of the pulpitis.\textsuperscript{22}

Alternatively, if the pulp cannot seal off the insulted area, it may undergo necrosis or severe bacterial infection and spread to involve the full pulp including the common pulp chamber, and then extend to involve the periapical tissues leading to apical infection. If the pulpar death can be localized by the body’s defenses, the occlusal aspects of the pulp may die but the underlying pulp remains viable and may seal off the pulp horn at a lower level – giving clinical occlusal pulpar exposure that does not extend very deeply. This finding in multiple pulps of some teeth can occasionally be found in older horses and the cause of the pulpar insult is unknown.

\textit{Pulp Stones}

Pulp stones, that more accurately should be termed \textit{false pulp stones}, because they have no internal tubular structure have been observed in grossly healthy equine teeth, both within viable pulp (free stones) and in areas replaced with secondary dentine indicating that they do not usually compromise their pulp vitality.\textsuperscript{23}

\textit{Occlusal Pulpar Exposure}

Odontoblasts that line the pulp cavity produce secondary dentine that gradually obliterates the pulp cavity circumferentially and subocclusally over the life of the tooth.\textsuperscript{24} In particular, subocclusal secondary dentine deposition (that is up to 100 times the rate of secondary dentine deposition on the pulp horn walls) prevents the pulp from becoming exposed on the occlusal surface in hypsodont teeth over the lifelong eruption of these teeth.\textsuperscript{23-25} This preferential subocclusal secondary dentine deposition is due to occlusal stimulation of the underlying odontoblasts. It was erroneously believed that an imbalance between occlusal wear and subocclusal secondary dentine deposition caused pulpar exposure on the occlusal surface, resulting in, \textit{descending infection} and ultimately apical infection of the cheek teeth. However, more recent studies\textsuperscript{26-28} indicate that occlusal pulpar exposure does not occur in healthy equine CT, but is caused by prior pulpar damage that caused cessation or reduced deposition of subocclusal secondary dentine that eventually leads to occlusal pulpar exposure.

Cheek teeth pulpar exposure can be recognised clinically. Dacre \textit{et al.}\textsuperscript{27,28} found occlusal pulpar exposure in 34\% of apically infected mandibular CT and in 23\% of apically infected maxillary CT, while van den Enden \textit{et al.} found occlusal pulpar exposure in 32\% of apically infected (maxillary and mandibular) cheek teeth, and also found occlusal pulpar exposure in 42\% of CT with idiopathic fractures.\textsuperscript{29} Ultrastructural examinations of equine teeth have shown that exposed dentinal tubules can be present on the occlusal surface that may provide a potential route of infection of the pulp from the occlusal surface.\textsuperscript{30}

Multiple pulpar exposure (in single or multiple teeth) is occasionally seen in equid teeth (mainly in older animals) that have \textit{no evidence} of apical infection\textsuperscript{28,31} and thus the presence of pulpar exposure does not necessarily indicate that pulpar or tooth death is present. Histological
examination of some equid CT with pulpar exposure has shown a layer of tertiary dentine protecting the pulp and sealing it off from an area of insulted pulp at the pulp horn occlusal tip that allowed (limited) pulpar exposure. However, especially in younger horses, the presence of multiple pulp horn exposure, of pulpar exposure with marked dentinal caries around the area of pulpar exposure, or of pulps that on probing are found to be deeply (>10mm) exposed, indicate the likelihood that the entire endodontic system has been severely damaged or is dead.

Apical Infections

Apical infection is a more accurate term to use in equids than “tooth root infection” because these infections often develop in young horses prior to any true root development, but they also occur in older CT with well developed roots. These infections are usually an extension of pulpar disease, as discussed earlier. Apical infections of incisor or canine teeth are rare in equids, but apical infections of CT are relatively common. CT apical infections are particularly significant in horses because of their great length, and consequently the infections usually spread to cause clinical changes in the alveoli and supporting bones. The clinical signs caused by CT apical infections depend on the site and age of the infected tooth, and the duration and the extent of the infection. When the rostral 2-3 maxillary CT are infected, rostral maxillary swellings and sinus tracts occur, with nasal discharge less common. Maxillary sinusitis is the main sequel when the caudal 3-4 maxillary cheek teeth are infected. Mandibular swellings and sinus tracts commonly occur with mandibular cheek teeth infections in younger horses. With older teeth, an apical infection may more readily drain via the periodontal membranes into the oral cavity and such case may just have halitosis as their only clinical sign.

As noted, occlusal pulpar exposure of CT is almost certainly a sequel to pulpar damage. The most commonly recognized cause of equine CT apical infection is anachoretic infection, i.e. blood or lymphatic borne bacterial infection (Figs. 1 & 2). Vertical impactions and hyperaemia of the developing apex can result in large eruption cysts (“3 and 4 year old bumps”) that in turn may predispose to anachoretic infections and this may explain the high prevalence of CT apical infections in younger horses.

Dental fractures are the second most common cause (20%) of apical cheek teeth infection (Figs. 1 & 2) and these fractures include gross, usually sagittal fractures and also hairline (fissure) fractures that communicate between the pulp and tooth periphery. Fissure fractures usually have dark staining (by bacterial or food pigments) on cut sections of extracted teeth. However, these fissures are not clinically obvious on the tooth surface unless the operator uses an oroscope or dental mirror and is aware of them. Not all idiopathic fractures (e.g. lateral slab fractures through the lateral pulp horns) cause apical infections as some such pulps can manage to seal off the exposed pulp laying down a layer of tertiary dentine to prevent infection spreading down the pulp horn.

Extension of infundibular caries to adjacent pulps is a specific disorder of maxillary CT that can cause apical infections (Fig. 2). Infundibular cemental hypoplasia (especially the 09s) with subsequent food impaction in the cemental defect predisposes to the development of infundibular cemental caries that may cause apical infections; either by weakening the tooth resulting in midline sagittal fractures; or by extension of the caries through the infundibular enamel into dentine and then pulp.
Figure 1. Routes of apical infection in equine mandibular cheek teeth.

Figure 2. Routes of apical infection in equine maxillary cheek teeth.
Apical infections can also occur secondary to developmental dental disorders (polyodontia, dental dysplasia, hypoplasia, diastemata and displacements), usually by an apical extension of periodontal disease (Figs. 1 & 2). As noted, periodontal disease can also be secondary to apical infections especially in older cheek teeth where exudate from the infected apex drains into the oral cavity via periodontal tracts – and thus avoids disease of the supporting bones (e.g. facial swellings or sinusitis).

Dental Caries

Caries is characterised by destruction of the calcified dental tissue with acid-producing bacteria the primary initiator of a chain of events. Bacterial fermentation of simple carbohydrates releases acids that decalcify the inorganic dental components (mainly calcium hydroxyapatite) at pH of 4 – 5.5.22 The most common type of dental caries identified in equine teeth is maxillary CT infundibular cemental caries.4,6,14,34,35 Honma et al.4 reported a prevalence of 100% in (maxillary) CT of horses over 12 years of age. Equine maxillary cheek teeth are predisposed to developing caries due to presence of developmental cemental hypoplasia of the infundibula, often near the apical aspect of the infudibulum, that only become occlusally exposed with age.36 A recent study found that only 11% of infundibulae were completely filled with grossly normal cementum, and areas of cemental hypoplasia and cemental discoloration, respectively, were observed in 22% and 72% of infundibulae.37

Cemental hypoplasia of the infundibulae of the rostral 3 cheek teeth can be due to premature destruction of the dental sac, such as by premature removal of overlying deciduous “caps”.18 However, recent examinations of CT of 1 – 3 years following eruption (dental age) showed a viable blood supply to the apex of infundibulae in many teeth. That would allow continued infundibular cemental deposition to occur for some years following eruption – but for some reason it is incomplete especially in the 09s. Marked infundibular cemental caries was found in 8% of infundibulae, with the Triadan 09 positions disproportionally accounting for 47% of these carious infundibulae.37

Infundibular caries has been classified by Honma4 according to the extent of its spread into the different dental tissues. A modified classification of infundibular caries has been proposed by Dacre,38 which is also applicable for grading peripheral caries (Table 1).

Caries of the peripheral aspect of the equine teeth is increasingly recognized,39 but has been poorly described in the literature. Because it most obviously involves the peripheral cementum, it was termed peripheral cemental caries, which does not describe the full extent of this disorder that can also involve the underlying enamel and dentine. Peripheral dental caries may affect infolded peripheral cementum, including cementum that lies on the occlusal surface, and therefore can predispose to increased rate of occlusal wear and fracture of unsupported (hard but brittle) enamel. By causing destruction of the normal peripheral cement-periodontal attachments, peripheral caries can also initiate local periodontal disease. Infundibular caries can also extend through the infundibular enamel – even causing coalescence of both infundibula.

Severe peripheral dental caries involving all classes of teeth (incisors, canine and CT) have been found in some groups of horses fed high concentrate, low roughage diets, where the reduced time
spent masticating, high levels of fermentable simple carbohydrates and decreased volume of buffering saliva may predispose to prolonged periods of low oral cavity pH that causes demineralization of calcified dental tissue. Individual horses may be susceptible to this disorder. Peripheral caries is also concurrently found with other dental abnormalities, where restricted food and saliva movement may predispose to its development. Extensive generalised dental erosions has also been recorded in horses fed diets with a low pH, where excessive acids were added to silage (haylage) and in equids fed diets consisting largely of simple carbohydrates, i.e. processed maize foodstuffs. Unfortunately, there is minimal information on the bacteriology of the equine mouth in health or disease and this area that needs investigation.

Table 1. Modified Grading System for Equine Dental Caries

<table>
<thead>
<tr>
<th>Degree</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0 degree: no macroscopic visible caries (can include infundibular hypoplasia)</td>
</tr>
<tr>
<td>1st</td>
<td>1st degree: caries only affecting the cementum</td>
</tr>
<tr>
<td></td>
<td>- from small pitting superficial spots (class 1)</td>
</tr>
<tr>
<td></td>
<td>- extensive destruction and loss of cementum (class 2)</td>
</tr>
<tr>
<td>2nd</td>
<td>2nd degree: caries affecting cementum and adjacent enamel</td>
</tr>
<tr>
<td>3rd</td>
<td>3rd degree: caries affecting cementum, enamel and dentine</td>
</tr>
<tr>
<td>4th</td>
<td>4th degree: caries now affects the integrity of the tooth i.e. development of an apical abscess or secondary tooth fracture.</td>
</tr>
</tbody>
</table>

Dental Fractures

Traumatic Dental Fractures

Although incisors contain predominantly equine type-2 enamel that is relatively fracture resistant, traumatic dental fractures, particularly of the incisors, are relatively common in horses due to external trauma. In contrast, equine cheek teeth are composed of high levels of hard but brittle equine type-1 enamel, with even higher proportions of type 1 enamel in equine maxillary than mandibular cheek teeth. Nevertheless, traumatic equine fractures of equine CT are less common than incisor fractures due to their anatomical protection, with mandibular CT fractures due to external trauma and iatrogenic fractures (from use of dental shears) being common causes of CT fracture.

Idiopathic Cheek Teeth Fractures

The majority of equine CT fractures have no known history of trauma and consequently are classified as idiopathic CT fractures. Because maxillary midline sagittal fractures have been shown to be associated with advanced infundibular caries – these could now be re-classified. A
A practice-based survey showed 0.4% of horses to be affected with idiopathic fractures.\textsuperscript{42} These fractures are often asymptomatic but can cause quidding and less often biting or behavioral problems, and halitosis.\textsuperscript{42,43} The upper 09s are most commonly involved and the predisposition of these teeth to infundibular caries is a likely factor for their midline sagittal fractures. The most common fracture pattern in idiopathic CT fractures are lateral “slab” fractures through the two lateral (buccal) pulp horns,\textsuperscript{41-43} possibly because the mineralised dental tissues are thinner at the sites of the pulp horns and therefore, the CT are weakest at this point\textsuperscript{27} (Figs. 3 & 4).

Examination of dentine showed thinner dentine in 25% of CT with idiopathic fractures, indicating prior pulpar disease, with the resultant thinner adjacent dentine mechanically predisposing to fracture. Dental pulps are inevitably involved in every (maxillary and mandibular) cheek teeth idiopathic fractures.\textsuperscript{41-43} Despite pulpar involvement, some idiopathic fractures (lateral slab fractures in particular), can clinically resolve without the development of clinical signs of apical infections, indicating that the resultant pulpitis has remained low grade or that the underlying pulp has become sealed off from the fracture site by the deposition of tertiary dentine. Many such fractured CT that survive following an idiopathic fracture have long-term radiographic apical changes, a subclinical endodontic and apical response to these CT fractures.

A proportion of fractured CT will develop pulpar infection that extends to clinically affect the apex, with the resultant clinical signs dependant on which tooth is involved. Clinical apical infection (including sinusitis) is common with maxillary midline sagittal fractures and with any type of mandibular CT fracture. Apically infected teeth always require extraction. In other affected teeth, mobile fracture fragments cause periodontal membrane stretching and pain during quidding until smaller dental fragments are spontaneously shed or extracted. Some fractured cheek teeth develop food impaction in the fracture site causing lateral or less commonly, medial
displacement of fracture fragments that causes soft tissue (usually buccal) ulceration and resultant quidding. Removal of the grossly displaced and/or all mobile fracture segments is indicated. Prevention of dental fractures secondary to infundibular caries has been attempted by removal of carious infundibular cementum and filling the infundibular defect with endodontic restorative materials, but there is no objective research on the value of this treatment.

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