Neaera Fletcher
BSc(Hons) BVMS CertAVP(EM) MRCVS

Neaera has worked as an equine veterinary surgeon since graduating from Glasgow University in 2009. She achieved advanced practitioner status in equine medicine in 2014, followed by equine dentistry in 2018. She works at Clyde Veterinary Group in Scotland – a member practice of XLVets – where she is responsible for advanced and referral dentistry.*

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**Suggested Personal & Professional Development (PPD)*

DENTISTRY

Equine dentistry – common problems and solutions

Oral pathology can go undetected because of the subtlety or absence of clinical signs in horses. Use of oral endoscopy has highlighted that oral pathology exists at a much higher prevalence than previously reported, and potentially painful oral pathology is frequently missed during dental examinations.

Detection of dental disease requires detailed and thorough examination at the time of routine dental examinations. A study examining 300 horses using oral endoscopy identified the most common cheek teeth abnormalities to include sharp enamel edges (96.3 per cent), focal overgrowths (64.3 per cent), diastemata (24.3 per cent) and infundibular caries (48.3 per cent) (Simhofer et al, 2008).

Focal overgrowths

Hypsodont teeth erupt continuously throughout the life of the horse. New clinical crown is erupting at the same time as the occlusal surfaces of the erupted clinical crown are worn down, thus there is a continual relationship between eruption and attrition of the equine dentition.

Focal overgrowths represent parts of the clinical crown that lack normal attrition rather than where there has been super eruption occurring (**Figure 1**). If, during mastication, the opposing occlusal surfaces do not meet each other, then attrition of the surfaces does not occur. Overgrowths subsequently develop in these areas.

Domestication of horses has meant reduced time grazing and increased access to concentrates which alters the chewing cycle to a more vertical than lateral motion (Dixon, 2000). There is also a disparity in width between the maxillary arcades (greater distance) and the mandibular arcades (anisognathia). The more vertical chewing pattern in combination with this anisognathia can contribute to sharp overgrowths on the buccal aspect of the maxillary arcades and lingual aspect of the mandibular arcades.

Overgrowths can also occur as a result of developmental disorders. Focal overgrowths are common on the rostral and caudal aspects of the cheek teeth arcades. Overgrowths of the maxillary triadan 06s and mandibular triadan 11s can arise secondary to disparities in the length of the maxillary and mandibular bones. These differences are developmental in nature and can be responsible for 'parrot mouth' and overbite conditions (**Figure 2**).

Other focal overgrowths can arise from differences in eruption times of cheek teeth, differences in timing of cap shedding and also from displaced teeth and supernumerary teeth. Overgrowths are also associated with acquired dental disorders such as loss of a cheek tooth. Where a cheek tooth has been extracted, or is missing, six-monthly dental treatment is paramount to prevent the development of overgrowths and to assess for other pathology.

Treatment of cheek tooth overgrowths

When reducing overgrowths, the structural anatomy of the equine cheek teeth arcades should be considered – including the natural rostral to caudal curvature of the maxillary arcades, the upper curvature of the occlusal surfaces of the caudal mandibular cheek teeth ('curve of spee') and the occlusal surface angles. When reducing overgrowths, in order to avoid inadvertent damage or exposure to the pulp horns, carefully staged reductions – ideally three months apart – will allow time for further secondary dentine protecting the pulp to be produced (**Figure 3**).

Subocclusal secondary dentine overlying the pulp horns varies significantly in thickness – even within the same cheek tooth – and can be

Figure 1. *Excessive transverse ridge on mesial 108.*

"A lack of clinical signs does not mean that there is an absence of oral pathology"

as low as 2mm thickness over some pulp horns (White and Dixon, 2010). The subocclusal secondary dentine thickness also does not increase with age, so care still needs to be taken when reducing overgrowths in a geriatric horse's teeth. Overgrowths of incisors should also, if required, be reduced very judiciously, given that subocclusal dentine thickness can occasionally be as low as 1mm (Englisch et al, 2018).

Thermal effect on teeth during reductions of overgrowths using motorised dental equipment can cause damage to pulp which can lead to pulp necrosis in some cases. Minimising time on the overgrown tooth during the reduction, alongside use of watercooling, reduces the risk of thermal damage to the pulp underlying the secondary dentine (O'Leary et al, 2012).

Diastemata

Equine diastemata are abnormal spaces between adjacent teeth which allow entrapment of feed material and subsequent periodontal disease (**Figure 4**). Diastemata are responsible for what is thought to be the most painful oral condition in horses owing to the associated significant periodontal disease. The presence of at least one cheek tooth diastema was found in 49.9 per cent (235) of 471 horses examined in a large study (Walker et al, 2012) indicating the high prevalence of the disease. The most common clinical sign of oral pain resulting from diastemata is quidding. Halitosis and buccal food packing can also occur (Collins and Dixon, 2005).

Diastemata are classified as primary, secondary or senile – depending on the cause. Primary diastemata are caused by the cheek teeth rows having insufficient caudal angulation of the first cheek teeth and insufficient rostral angulation of the caudal cheek teeth. Primary diastemata can also arise when the tooth buds develop too far apart (Dixon, 2010). Developmental cheek teeth diastemata carry a better prognosis than acquired diastemata as the spaces can close with ongoing dental eruption (Collins and Dixon, 2005). Secondary diastemata are acquired and develop when there are displacements of cheek teeth within the arcades, or due to overgrowths, supernumerary teeth or dental drift following extraction or loss of a cheek tooth (**Figure 5**). Prevalence of senile diastemata significantly increases with age. Due to the continuous eruption of hypsodont teeth, the narrower apices eventually erupt and the clinical crown becomes smaller in size resulting in senile diastemata.

Treatment of diastemata

There are many described treatments for diastemata, despite this there is a lack of defined guidelines on when and how to treat the condition. The first stage of treatment should be identifying the cause. Corrective equilibration of the cheek teeth is important and addressing any excessive transverse ridges opposite the diastemata, sharp enamel points or focal overgrowths can provide resolution in many cases (**Figure 6**). Reducing the occlusal surface of the crown immediately adjacent to the diastemata to remove it from occlusion will reduce the compression of food into the space (Collins, 2005).

Cleaning and lavaging the periodontal pockets is essential to allow healing of the gingiva. Placement of temporary packing such as polyvinyl

Figure 2. *Rostral focal overgrowth of the 106 and 206 maxillary cheek teeth of a Shetland pony. This pony has a mild 'parrot mouth'.*

Figure 3. *Shetland pony from Figure 2, taken one year later following staged reduction of the rostral focal overgrowths of the 106 and 206 maxillary cheek teeth. Note the brown staining of the secondary dentine of the mesial pulp is still present.*

Figure 4. *Valve diastema found between 408 and 409 mandibular cheek teeth.*

Figure 5. *The excessive transverse ridge of mesial 108 has cause a diastema between 407 and 408 mandibular cheek teeth.*

Figure 6. *Initial treatment of excessive transverse ridge (see Figures 1 and 5) to address the cause of the diastema found between the mandibular cheek teeth 407 and 408. Note the occlusal angles are maintained and this will be a staged reduction.*

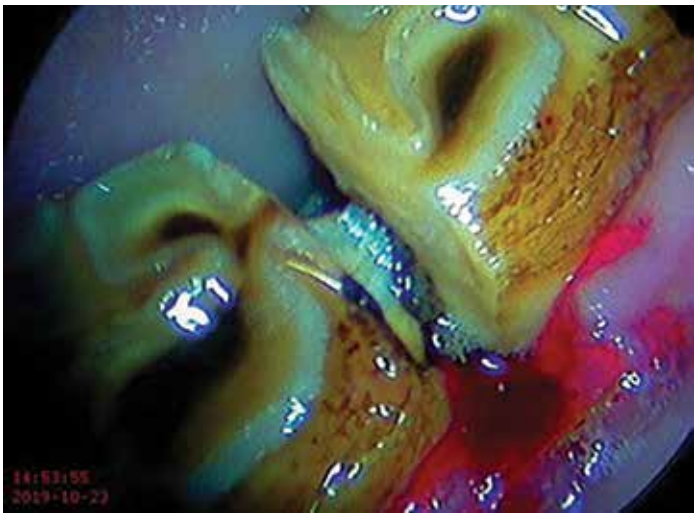

Figure 7. *Diastema which has been partially widened.*

siloxane is also commonly used. Polyvinyl siloxane should only be used temporarily and filling the periodontal pocket deep to the clinical crown will prevent reduction in size of the pocket. A chlorhexidine-based mouth wash can be useful for cleaning the cleared periodontal pockets; however, it will not be useful for treating periodontal disease caused by diastemata without addressing the food packing itself.

A recent study looking into a range of commonly used treatments (cleaning, packing and widening) found no significance in outcome between the different treatments, but an overall improvement in reducing the periodontal pocket depth from each of these treatments (Jackson, 2016). Widening of the interdental space to open diastemata can be considered for select cases of valve diastemata (**Figure 7**). Awareness of the proximity of pulp to the interdental space is essential. The proximity of pulp to the interproximal spaces can be as low as 1.3mm and the risk of exposure increased when dental tissue is removed from the caudal aspect of cheek teeth (Bettiol and Dixon, 2011). Thermal damage and pulp exposure are both risks of this procedure and careful visualisation during widening of the diastemata is important with use of irrigation.

Dietary modification, substituting long fibre with short fibre and grass can also be reasonably successful, although feeding short fibres will increase vertical chewing actions and increase the development of sharp enamel points (Collins and Dixon, 2005).

Where displacements of teeth are severe, extraction of the tooth may be required. Awareness of subsequent drift and development of new diastemata needs to be considered in these cases. Owners should be aware that repeat treatments are often required.

Infundibular hypoplasia and caries

Infundibular caries (IC) is often an insidious dental disorder of maxillary cheek teeth that can lead to pathological fractures and apical infection of the affected teeth. A cadaver study of

"Overgrowths can also occur as a result of developmental disorders"

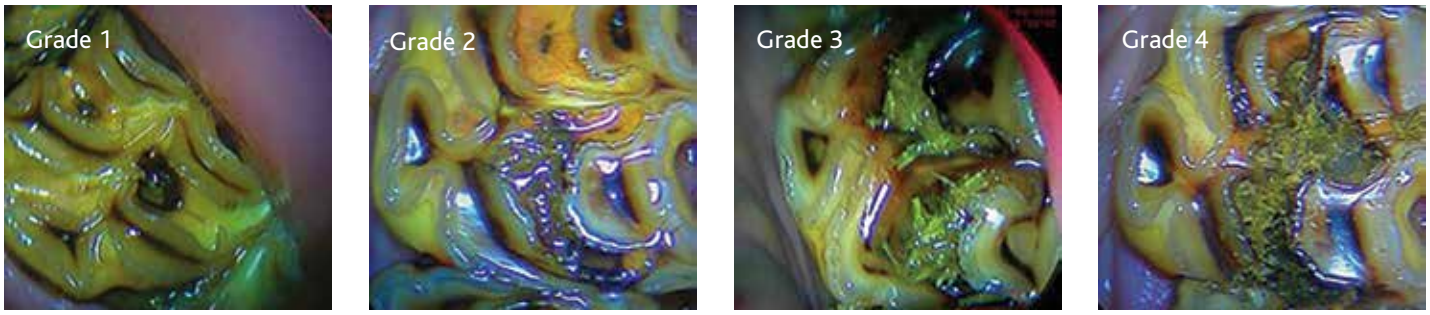

Figure 8. *Grades 1- 4. Infundibular caries are graded by the appearance of the occlusal lesions using the modified Honma classification system.*

706 UK horses showed 45.5 per cent had infundibular caries – most commonly present and most severe in the triadan 09s – and prevalence was significantly associated with increasing age (Borkent et al, 2017). Infundibular caries arise from infundibular cemental hypoplasia (ICH) – a developmental disorder where there is incomplete filling of the infundibula with cementum. Once the defects in the infundibula become exposed at the occlusal surface of the teeth, food material packs into the defects and bacterial fermentation ensues resulting in progressive infundibular caries. Sixteen per cent of cases of apical infection of maxillary cheek teeth are attributable to extension of infundibular caries (Dacre et al, 2007) and sagittal fractures involving infundibula, which invariably involve apical infection, are also caused by infundibular caries (Dixon et al, 2000).

Infundibular hypoplasia is extremely common with almost all horses having some degree present in the maxillary cheek teeth (Fitzgibbon et al, 2010). Infundibular caries is also very common, however not all affected teeth will develop fractures or apical infection. Infundibular caries are graded by the appearance of the occlusal lesions using the modified Honma classification system (**Figure 8**) (Dixon and Dacre, 2005):

- grade 1: caries of the cementum only
- grade 2: caries of the cementum and surrounding enamel
- grade 3: caries of the cementum, enamel and dentine
- grade 4: coalesced caries of the rostral and caudal infundibulum
- grade 5 (not pictured): advanced caries resulting in apical infection/ tooth fracture.

Treatment of Infundibular caries

Treatment involving restoration of infundibular caries is only necessary in select cases. For example, where the triadan 09s are more likely to be affected and result in progression to apical infection and fractures, these teeth are also more likely to reach the selection criteria. Chris Pearce (2015) suggested a selection criteria for those cases where restorations could be beneficial in preventing progression of lesions to fracture or apical infections. The suggested criteria included:

- IC of grade 3 or above identified at the occlusal surface, with at least 10mm probing depth
- IC of grade 2 or above if contralateral to a pathological saggital fracture (IC grade 5)
- deep cavities/aplastic infundibula identified on CT examination.

Restoration involves meticulous cleaning of the cavities, preparation of the cavity and use of restorative materials. Oroscopic imaging during this advanced procedure is essential.

Conclusion

The identification of oral pathology at the time of a dental examination is paramount in the provision of dental care in the horse. A routine dental examination is where the vast majority of pathology will be identified. A lack of clinical signs does not mean that there is an absence of oral pathology. In recent years, increased awareness of dental pathology alongside development of modern techniques for treating dental conditions – including advanced minimally-invasive procedures – has meant oral pain in horses can be treated effectively and, where identified in the early stages, progression of pathology can be prevented. ■

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PPD Questions

1. Which oral pathology is thought to cause the most pain and discomfort to horses?
2. What is the minimum thickness of subocclusal dentine found to be in incisors and cheek teeth?
3. What are the three techniques applied that can reduce the chances of thermal damage occurring when reducing dental overgrowths?
4. Which dental tissues are involved in infundibular caries of grade 3 based on the modified Honma system?
5. What two pathologies can progression of infundibular caries result in?

Answers

1. Periodontal disease caused by diastemata.
2. 2mm in cheek teeth, 1mm in incisors.
3. Minimising time of the motorised instrument on the tooth and using irrigation/ water cooling and staging reductions.
4. Cementum, enamel and dentine.
5. Fracture of teeth and apical infection.