



# Improving Your Equine Dentistry Mini Series

Session Three: Dealing with Common  
Problems in Equine Dentistry - to  
Refer or Not to Refer?

**Chris Pearce CertEM(IntMed)CertES(SoftTissue)  
BAEDT MRCVS  
ECVS Diploma in Equine Dentistry**



### **Problems in Equine Dentistry – to refer or not to refer?**

- Dental overgrowths
- Dental apical abscesses / pulpitis
- Fractured teeth
- Cavities and caries
- Diastemata and periodontal disease

### **Dental overgrowths: overgrowth and odontoplasty**

Horses have developed over 500 million years as browser / grazers and in natural circumstances would graze for approximately 16 hours per day on fibrous forage utilizing the full range of excursion of their mandibles to masticate the food. The main function of the teeth is to soften and crush the forage, and cut it into lengths of 2-3mm for presentation to the intestinal tract. Inadequate dentition results in longer fibre lengths reaching the intestine and poor fermentation by the resident flora and fauna. Domestication of horses, alteration in feeding practices and diet results in a reduction in the daily mastication time and the range of motion of the mandibles due to feeding of processed food and soft plentiful forage (e.g. in UK). It has been demonstrated that when horses masticate softer, processed food the lateral excursion of the mandible is reduced. This combined with less mastication time results in uneven wear patterns of the cheek teeth, and possibly the incisors. Sharp enamel points on maxillary teeth and lingual points on mandibular teeth no longer receive even wear and the result is a variety of overgrown, sharp buccal and lingual points that may traumatise the adjacent soft tissues.

Even attritional wear of equine teeth not only requires adequate lateral mandibular excursions movements but also even rostro-caudal and bucco-lingual contact of the dental arcades, and even masticatory patterns. Alterations of this even masticatory contact will result in altered eating patterns and/or focal overgrowths of unopposed dental tissue. This may be due to:

- Conformational / congenital abnormalities e.g. malocclusions, prognathia, oligodontia
- Developmental malocclusions e.g. displaced or rotated teeth
- Pathological malocclusions e.g. fractured teeth, lost teeth, diastema, large cavities
- Pathological dysmastication e.g. painful pulp disease
- Neurological dysmastication e.g. facial nerve paralysis, muscle disease

Overgrowths of teeth have previously been referred by a number of titles – hooks, ramps, beaks, points etc. however the author prefers to use the term ‘focal overgrowth’ to describe any protruberance from the normal dental arcade. It should also be remembered that ‘overgrowth’ of a tooth is actually normal eruption and reduced attritional wear, consequently the cause of this focal area of reduced wear should be investigated.

Equine cheek teeth naturally have a series of transverse ridges which interdigitate occlusally to increase the surface area for mastication. These have been identified in many studies of feral horses, are present in fossil records of horses going back millions of years and can be considered a normal feature of an equine mouth. Whilst it is fair to assume that all transverse ridges should be approximately the same height, it is highly improbable and totally unscientific to follow the view of some lay-persons that all transverse ridges should be considered abnormal focal overgrowths.

Modern maintenance dentistry can be considered to be:

- Examination and assessment of the head and dental structures
- Identification of abnormalities including occlusal pathology
- Careful odontoplasty to restore and maintain normal masticatory function
- Additional odontoplasty to ensure comfort with tack (see below)
- Prophylactic treatment of developing pathology e.g. periodontal disease
- Advice on diet, further treatment and re-examination

## **Odontoplasty of focal overgrowths**

The goals of routine dental treatment are not just to use the latest, fastest, diamond coated powered instrument to sculpt the clinical crowns into an aesthetically pleasing, but functionally questionable form, based on unsupported pseudoscientific opinion on what constitutes a 'nice mouth'. Appropriate dentistry should comprise investigation followed by precise, considered, conservative adjustment or treatment to assist occlusion and mastication and is not merely to perform empirical tooth sculpture. As equine dental tools develop in efficiency it becomes increasingly easy to perform excessive odontoplasty in the pursuit of unscientific principles of floating and reduction.

It is important for the clinician to balance the need for physiological efficient mastication system with a desire to make a horse comfortable including when ridden with a bit and bridle. It is this balance that is impossible to quantify, but a good clinician will strive to perfect.

It is also essential when reducing overgrowths to assess the potential for causing iatrogenic damage to the pulp either through thermal trauma or by exposing sensitive sub-occlusal dentine or pulp.

### *Sub-occlusal secondary dentine*

Recent research has shown that the depth of sub-occlusal secondary dentine does not increase with age as has been traditionally thought. In fact, the pulp may be closer to the occlusal surface in older horses than young horses, with pulp as close as 3mm from the surface in some cases. Similarly, it had been thought that the depth of sub-occlusal secondary dentine was greater in unopposed teeth with marked overgrowth however this has also been shown not consistently to be the case. Therefore when reducing large overgrowths in young or older patients it is important to constantly observe the occlusal surface for changes in discoloration demonstrating penetration of the sensitive dentino-pulp complex.

### *Removal of dental tissue – odontoplasty*

Tooth rasping or "floating" has been performed since the 1840's to assist mastication of domesticated horses. In the last thirty years there have been advances to facilitate it comprising

- Better understanding of dental physiology and the consequences of iatrogenic damage
- Better more efficient instrumentation.
- Use of illumination and mirrors to enable precise application of the above
- Widespread use of sedation to facilitate precise treatment in a safely restrained compliant horse.

Modern odontoplasty may be performed with hand instruments ('rasps') or power instruments but either way careful consideration should be given to the effects of excessive odontoplasty

- Excessive reduction reducing crown length and longevity
- Thermal damage to pulp using power equipment
- Exposure of pulp either directly or indirectly

The teeth should be profiled to create edges that will not traumatise soft tissues, but does not undermine the grinding function of the occlusal surface. Peripheral cementum on the buccal aspects should not be removed as high as the gingival margin and it should always be remembered that dentine contains sensory nerves, so it should not be assumed that rasping is a painless procedure without potential consequence. During routine rasping the occlusal angles should be maintained within the 10-30° physiological normal range and not reversed.



Fig 1. Sharp buccal point and associated buccal mucosal trauma.



Fig 2. Appropriately profiled buccal point following odontoplasty.



Fig 3. Mandibular lingual enamel points without trauma; minimal odontoplasty required.

#### *Rostral profiling of the 06's*

The practice of rounding the rostral and buccal aspects of the 06's is popular, especially in horses used for technical disciplines such as dressage and polo, although currently there is no evidence to justify this in all cases. This practice has inaccurately and misleadingly been termed "bit seating" historically. The rationale for rostral profiling is that this prevents entrapment of the mucosal fold of the buccal commissure against a sharp dental periphery, which could result in trauma, and, in horses subjected to a lot of bit strain, or when wearing double bridles, there is some logic to this practice. However, the original explanation for creating a notch in the 06's for the bit, which should in fact sit rostral to the 06's and in contact with the mandibular bars of the mouth, appears to be total fallacy based on recent studies. The practice of performing this to excess with reduction to the level of the gingiva is likely to be harmful and can result in exposure of the rostral pulp horn.

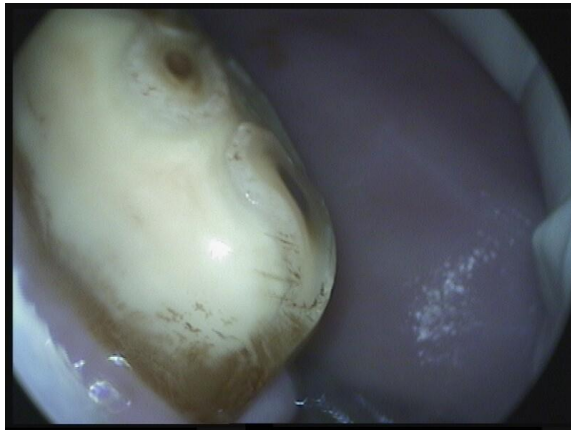


Fig 4. Conservative rostral profile of 306 avoiding pulpar exposure.

#### *Motorised instruments*

Modern powered instruments make the practice of routine dentistry less strenuous, and proper use of the instruments lead to a controlled and careful odontoplasty. The instrument shaft should be held with 2 hands and a 3 –point contact to stabilize the instrument whilst being constantly visualized to observe the effect on the teeth. Water irrigation will improve the efficiency of the head, and protect against thermal pulp damage. A recent study has shown that pulp may become heated to a critical level without water irrigation if a power instrument is placed for 20 seconds at maximum speed; therefore it is logical to use the instrument at the lowest effective speed and for short periods only. Without water irrigation, the head of the instrument should be cleaned in water every 10 seconds.



Fig 5. Modern disc instrument with additional 'apple core' attachment and diastema burrs.



Safety measures when using powered instruments include use of a residual current device if using mains power, a step down transformer to 110V, or a battery powered unit. Keeping the working area free of water and mouthwash is also essential. A clutch will also prevent iatrogenic damage to the tooth, and protect the instrument.



Fig 6,7. Correct use of a motorized instrument: comfort, control, visualization.

#### *Rostral and caudal hooks.*

Severe rostral and caudal hooks resulting from malocclusion between rostral and caudal arcades require reduction to the level of the rest of the arcade to facilitate normal chewing. Historically they were reduced using molar shears or guillotine devices which can be considered obsolete since the use of such instruments frequently results in pulp exposure. Reduction by using a cooled motorised burr is more precise and efficient, with careful observation to detect colour changes in secondary dentine overlying pulp horns to avoid inadvertent pulp exposure. It may be necessary to perform major reductions in stages separated by several weeks to avoid pulp exposure (although the precise time interval to allow production of secondary dentine over a pulp horn is not known).



Fig 8. Examples of tall overgrowths on Triadan 106 and 107 due to loss of (the opposing) 407 and subsequent caudal (distal) drifting of 406. In addition to reduced wear of 106 and 107, an increased rate of eruption has allowed these overgrowths to become taller. It is unclear at present how much of these overgrowths can be therapeutically reduced at the initial treatment without risking pulpar exposure. (reproduced with permission from Marshall, Shaw and Dixon 2012).

### *Transverse ridges*

Transverse ridges are a normal feature of the occlusal surface of cheek teeth. The development of these interdigitating features is a consequence of normal equine mastication. It is therefore contra-indicated to smooth off the transverse ridges completely along an arcade although evidence of this bizarre practice is commonly encountered in the wake of some modern powered tools.

*Excessive transverse ridges* (ETR's) can be identified as more prominent ridges on the arcade when compared to the other teeth in that arcade. Contrary to popular disinformation these do not cause 'locking' of the temporomandibular joint and other problems. However, such ETRs can limit normal chewing movement and they can impact food into the interproximal space opposite. It is appropriate to reduce such prominent ridges to the level of the other ridges in the arcade or to a greater degree if taking the individual tooth out of occlusion is desired.

Following any odontoplasty a careful visual and manual palpation should be performed to assess the result.

### **Removal of wolf teeth (1<sup>st</sup> premolar- 05's)**

This has been traditionally practiced and is widely popular despite little evidence for its necessity. Non-erupted or displaced wolf-teeth can be sensitive when in contact with the bit, but there is little evidence that normally sited normally placed wolf teeth cause any discomfort, or prevent routine rasping of the more caudal teeth. However it is acknowledged that many horse-owners expect wolf-tooth removal to be performed and it is acknowledged that fundamentally extreme opinions exist as to the benefits of wolf tooth removal within the profession around the world.

Wolf-teeth ideally should be removed in sedated patients with the administration of local anaesthetic to the palatal mucosa (or mandible for lower wolf teeth). A human dental syringe and 30G needle are ideal for local infiltration of the wolf tooth alveolus. Careful separation of the periodontium with an elevator or luxator is indicated until the tooth is sufficiently loose to be extracted with minimal force using rongeurs or small extraction forceps. The traditional Burgess type wolf-tooth kits, remain popular but are rather obsolete and ineffective compared to the elevators now available in the authors' opinion. Inadequate restraint and poor direction of the elevator can result in fractured roots or palatine arterial puncture with extremely colourful consequences.

### **Dental apical abscesses and pulpitis**

The aetiopathogenesis of dental apical abscesses and septic pulpitis was reviewed in the previous lecture and notes; a summary is below

- Most septic pulpitis is a result of anachoresis – inoculation of blood-borne bacteria within a region of pulpar inflammation.
- Bacteria become partially isolated from the immune system within the pulp canal and multiply.
- Immune response and inflammatory changes result in apical granuloma formation at the dental apex; for the rostral maxillary teeth and most mandibular teeth this results in focal and sometimes large bony swelling over the affected tooth apex, often described as an apical abscess, most common in young horses with large vascular pulp system, wide apical foramina and minimal root formation.
- Pulpitis and local immune responses (and possibly antibiotics) may control the infection and the pulp may survive; odontoblasts within inflamed regions of the pulp may produce tertiary / reparative dentine, e.g. pulp stones and 'osteodentine' – this may 'seal' the apex from the septic focus.
- Reparative dentine may occlude the entire pulp at any point from occlusal to apical, isolating the necrotic pulp distally from the apex.
- With extensive septic pulpitis apical sclerosis and cementosis may isolate the entire (now necrotic and septic) pulp system fully or partially controlling the apical reaction – these cases may appear 'resolved' in the short term as apical swelling reduces.

- Pulpal separation may result in only partial pulp system death.
- Pain from diseased teeth results in alteration of mastication patterns, often unnoticed by the owner.
- Continued eruption and occlusal attrition of such diseased teeth results in gradual appearance of a defect of secondary dentine as necrotic diseased pulp becomes exposed (secondary dentine production has ceased).
- Defects of secondary dentine (even minute) become impacted with food material, which tracks up the necrotic pulp canal with resultant secondary pulp caries; if no tertiary dentine bridge is present this will track apically to and sometimes even beyond the dental apex.
- Pulp caries deteriorates progressively as lesions enlarge and more food impacts and ferments releasing more acid.
- Eventually teeth may become unstable and fracture – i.e. a pathological fracture.

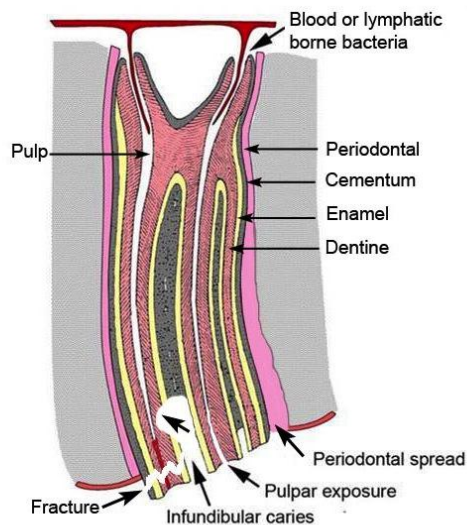


Fig 9. Drawing showing potential routes for infection of the pulp in a maxillary tooth – most cases are blood-borne (anachoresis) (image courtesy of P.Dixon)

*[NB The term 'caries' refers to the disease process whereby dental tissues become progressively damaged and decayed by acid produced by bacterial fermentation of entrapped carbohydrate; as a clinical term of disease it does not change if referring to singular or multiple lesions – they are all 'caries' (i.e. there is no singular term)]*

Occlusal dentine fissures / non-vital pulp exposures (NVPE, see image below) have been shown to be highly significant findings and in studies strong links have been demonstrated between such lesions and apical abscessation.

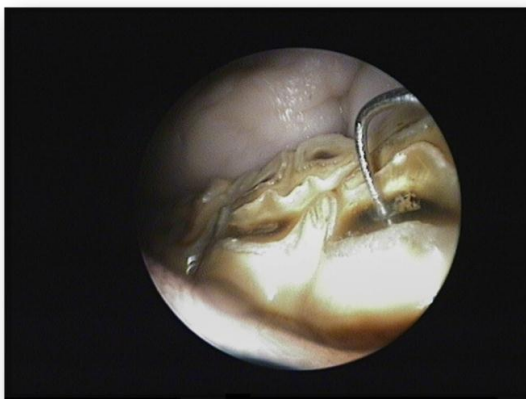


Fig 10. Orosopic image showing a defect of the secondary dentine with secondary food impaction and early caries often associated with apical abscessation. However despite the endodontic system being compromised, these findings may be incidental, and further information regarding any sub-occlusal reparative dentine formation is unknown from this image alone; radiographs recommended.



Initial treatment for apical abscessation / granuloma formation e.g. in a young horse when first presented may be as below:

- Mandibular / maxillary swelling / unilateral nasal discharge
- Radiography → apical abscessation / granuloma / periodontitis (→ possibly sinusitis)
- If **< 4 weeks duration** → AB therapy 2 weeks, repeat +4 weeks (+ check occlusal surface for pulp exposure)
- If **> 4 weeks duration** or if recurs after AB tx --> likely intractable pulpar sepsis → Extraction

Endodontic therapy

Extraction / extra-corporal root canal therapy / re-plantation

If antibiotic therapy appears to have been effective, the three previously described categories may be used to direct further treatment / management:

Category of cheek tooth pathology / endodontic status	Description	Action
Category 1	The pulp has survived the insult, tertiary dentine may be present, no apical infection is evident, no occlusal dentine defects are visible oroscopically (some fractures may be within this category)	No immediate action required; monitor closely (up to 3 years); if pulpar death has occurred, occlusal dentine defects may not appear for months or years
Category 2	No evidence apical disease, tertiary dentine bridges have formed within the pulp, but occlusal pulp / dentine fissures are present and caries is present (or Grade 3+ infundibular caries)	Treatment using endodontic / restorative techniques is appropriate
Category 3	The apex and some or all of the pulp system is septic / compromised (includes some fractures including <i>all</i> maxillary sagittal fractures); signs of apical abscessation are present radiographically, external discharging sinus tracts and/or sinusitis may be present	Extraction of tooth required



Fig.11 Orosopic images showing small (left) and larger (right) occlusal pulpar exposures, both in horses with apparently no clinical signs of dental disease; such findings are not uncommon and careful assessment of mastication patterns combined with diagnostic imaging is required to demonstrate the potential seriousness of the developing problem to the owner.

### Treatment: Dental Extraction

With appropriate equipment, training, planning and patient preparation dental extraction can be performed successfully by many experienced clinicians. However, consideration should be given to the age of the horse, any pathology present, the radiographic appearance and the experience of the practitioner. These considerations should guide the appropriate action – oral extraction in-house or referral to a specialist. The table below gives some guidelines regarding considerations

Table 1. Considerations for practitioners considering dental extractions in cases with septic pulpitis.

Duration	Age	Dentine defects	Decision
Short <2m	Young or geriatric	None	Attempt extraction
Short – mid (2-5m)	Not geriatric	Small fissures, pits	Consider if experienced
Mid – long (5-12m)	Not geriatric	Moderate / multiple	Must have plan B
Long (>12m)	Any	Multiple or fractured	Consider referral – plan B and C required
Long (>12m)	Any	09 tooth, dentine defects, sinusitis, +/- fracture	??Refer!

### Dental fractures

Fractures of teeth have been traditionally described as 'idiopathic' fractures as little explanation has been offered for the aetiopathogenesis of such fractures. However when considering the pathogenesis of dental caries it appears likely that most fractures are the result of long term chronic caries and as such are often end-stages of decay, probably from inflammatory events and ongoing caries possibly for many years (also from infundibular caries as below). Therefore the term 'pathological' is more appropriate than 'idiopathic' when describing caries-related fractures. Therefore a new categorisation of dental fractures could be:

- Traumatic fractures e.g. incisor teeth from falls, kicks
- Pathological fractures
  - Dentine caries related secondary to advanced septic pulpitis and occlusal pulpar exposure
  - Infundibular caries-related from advanced infundibular caries
  - Peripheral caries related from advanced peripheral caries
- Dentine fissure-related fractures (see below)
- Idiopathic fractures
  - No identifiable cause of fracture is present

As can be seen from the above images of pulpar exposure and secondary caries, chronic lesions result in larger pulpar defects as caries progresses, weakening the structure of the teeth and leading to eventual fracture (see image below).



Fig. 12. Oroscopic image of dental caries-related fracture (secondary to advanced secondary pulpar and dentinal caries)

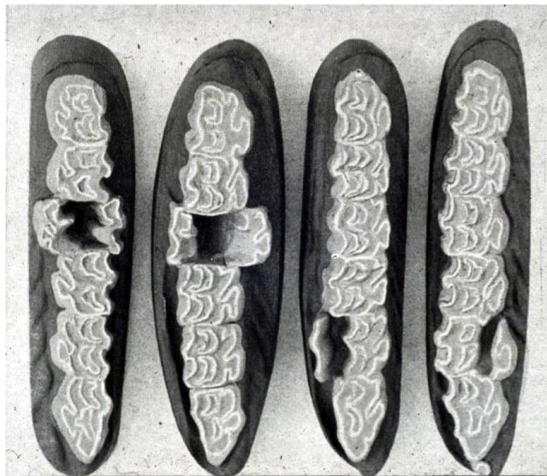


Fig. 13. Drawing by Becker, 1940 of commonly identified fractures

#### Dental cracks, dentine fissures and fissure fractures

Oroscopic examinations are able to detail dentine fissures and small defects more accurately than with direct mirror examination. Narrow cracks and dentine fissures are not infrequently observed and anecdotally in the author's experience horses with multiple teeth affected by dentine fissures or cracks are more prone to certain types of dental fracture. Interestingly these fissures and cracks appear to follow exactly the same patterns of fracture as described by Dacre *et al* 2007 (see image ).

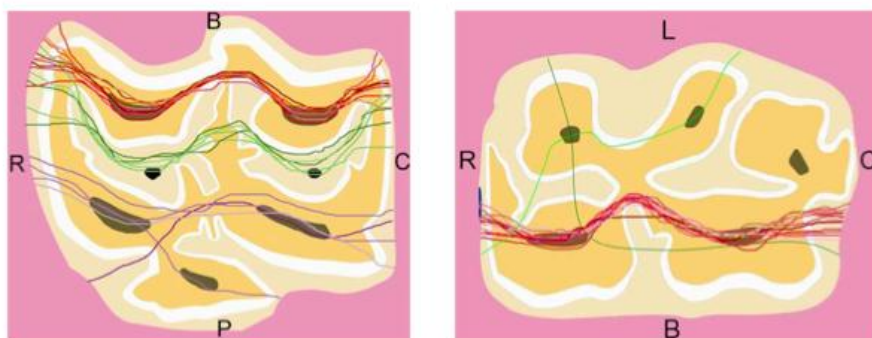


Fig. 14. Fracture planes recorded by Dacre *et al* 2007 – dentine fissures or cracks have been anecdotally found by the author to follow these same planes.



Fig. 15. Orosopic images of dentine fissures / cracks present in similar planes to those seen in dental fracture studies.

At present there is no method of treatment of these fissures. Occasionally food may be seen impacting into such fissures with secondary caries – such lesions may be suitable for restorative therapy.

### **Infundibular disease and dental restorations**

The anatomy of the equine infundibulum (plural: infundibula) has been previously described. All equine incisor teeth have a single, shallow infundibulum (commonly referred to as the 'cup'). With occlusal wear, this shallow infundibulum disappears relatively early in the life of an incisor tooth, and due to the differential eruption of incisor teeth, the progressive disappearance of the infundibulum from occlusal attrition gives an indication of age. For cheek teeth, there are two infundibula per tooth (rostral and caudal), however these are unique to maxillary teeth – i.e. there are no infundibula of mandibular cheek teeth. Compared to incisors, cheek teeth infundibula are considerably deeper structures, parallel sided for much of their length, and extending almost 80-90% of the distance to the root apex.

Abnormalities of the infundibula of the maxillary cheek teeth (CT) have been described as infundibular cemental hypoplasia, and infundibular caries.

#### *Infundibular cemental hypoplasia (IH)*

This developmental abnormality most commonly affects the more apical aspect of infundibula although rarely there may be more complete or even total absence of cementum within infundibula which may be described as complete cemental hypoplasia, aplasia or a patent infundibulum.

Recent studies have shown that the Triadan (maxillary) position 11 tooth is most commonly affected by apical cemental hypoplasia, however the largest defects and complete hypoplasia most commonly affect the 09 teeth, with 8.2% of maxillary Triadan 09 teeth having completely aplastic infundibula, and only 3% having normally filled infundibula. Many such lesions are bilaterally symmetrical.

#### *Infundibular Caries (IC)*

Equine teeth continue to erupt throughout life, with continual attrition at the occlusal surface. This results in progressive shortening of the reserve crown, and subsequent exposure of lesions within the reserve crown on the occlusal surface, at a time dependant on the depth of the lesion. Apical cemental infundibular defects will be exposed occlusally towards the end of the life of the tooth, i.e. as the apical aspect of the reserve crown becomes the clinical crown. More extensive infundibular hypoplasia defects extending further towards the occlusal surface will be exposed at a younger age, producing a risk of food impaction deep into the hypoplastic infundibulum. The small number of horses with complete cemental hypoplasia will have food packed into these empty infundibula from a very young age – as soon as the tooth erupts.

Dental caries has been defined as a progressive acidic demineralisation of the inorganic matrix of dental tissues secondary to bacterial fermentation of impacted carbohydrate substrate and subsequent organic matrix loss. It seems most likely that once a hypoplastic defect within an infundibulum is exposed to the occlusal surface, significant amounts of food become impacted resulting in progression of caries, as above. An abattoir study of CT pathology in Baker's PhD thesis of 1970 sums up well the situation regarding caries of the infundibula: *"...areas of cement necrosis were centred on the infundibulum and expanded centrifugally within the enamel invaginations. In advanced cases this expansion resulted in the coalescence of the area of the rostral and caudal cement lake necrosis"*.

The age at which this occurs depends on the extent of the hypoplastic defect. Small apical cemental defects with normal cementum occlusally are logically less likely to develop significant caries – bacteria may penetrate to the apex however significant food impaction and subsequent caries only develops once a large defect is exposed occlusally.

In many papers and studies, the Triadan (maxillary) 09 teeth are described as significantly over-represented for infundibular hypoplasia and caries lesions. In one study only 10% of teeth examined by computed tomography had normal infundibular cementum formation at the 09 position. The reason for this over-representation is not clear. One suggestion is that the premature removal of deciduous 'caps' results in the premature loss of the occlusal vascular supply to the infundibulum. However, the Triadan 09 tooth having no deciduous precursor invalidates this theory. It has been documented that cement deposition continues until the tooth erupts onto the occlusal surface, in which case it could be the case that the 09 being the first tooth to erupt would lose the vascular supply first possibly before full cement deposition apically has occurred.

In Sweden infundibular caries of the Triadan 106/206 positions were found to be specifically related to presence of a novel bacteria (*Streptococcus devreisei*), which may also be a significant factor in the more rapid progression of caries once large hypoplastic defects are exposed occlusally. Clinically, once cemental hypoplasia lesions are large enough to be identified occlusally, either or both of the above mechanisms is likely to accelerate the process of caries and the expansion of the lesion. Again, this was summarized well by Baker (1970) *"caries of cementum was only found in maxillary cheek teeth and a study of cementogenesis in Thoroughbred foetuses suggested that a primary hypoplasia was a major aetiological factor in the development of this condition, while lysis of cementum by the acid products of fermented foodstuffs was of secondary importance"*.

#### *Dental fracture*

Large caries lesions provide a central plane of structural weakness in the tooth predisposing it to pathological fracture when exposed to the normal forces of mastication. Advanced lesions of both rostral and caudal infundibulae, especially those that coalesce, are likely to result in significant structural weakness of the CT.

A recent study showed caries affecting the full length of the infundibulum in 8.2% of infundibula studied, most commonly in the 12-20 year age group, and concluded that this would be likely to predispose the tooth to pathological fracture. Many studies have shown significant over-representation of the maxillary 09 for sagittal fractures involving infundibula, with such fractures having apical infection 100% of the time, necessitating extraction. Also, extension of caries from the infundibulum has been shown to cause apical sepsis in 16% of maxillary CT apical infections.





Fig. 16. Pathological fracture of 107 secondary to advanced infundibular caries.

Infundibular caries based on occlusal appearance has been classified and graded as follows using a modified Honma classification system:

- Grade 1 – Caries of infundibular cementum
- Grade 2 – Caries of infundibular cementum and enamel
- Grade 3 – Caries of infundibular cementum, enamel and dentine
- Grade 4 – Advanced caries resulting in apical abscessation, fracture or tooth loss

The author uses a slight modification of this grading system described by Johnson and Porter 2004:

- Grade 4 - Advanced caries Grade 3 rostral and caudal infundibulum, coalescing lesion
- Grade 5 – Apical abscessation, fracture or tooth loss

The age at which fractures are most common has been documented in various studies, most common for sagittal fractures being the 12-20yrs age group, and it seems plausible that these teeth are most at risk when large infundibular defects become exposed occlusally and develop deep caries from food impaction, although this is not validated by research.

#### *Restoration of infundibular cavities*

The principles of restoration are to protect the pulp, arrest decay, restore the tooth to function, and prevent further disease. Techniques to obturate infundibular cavities (hypoplasia, caries or both) that could predispose to fractures have been described. Restoration procedures for apical hypoplastic lesions in young horses has been described as 'difficult or impossible' following anatomic studies showing that these lesions are often situated towards the apex, and may be up to 80mm from the occlusal surface, and follow a curved path from the occlusal aspect. These lesions are so numerous that they have also been described as 'virtually a normal feature'. As caries is only likely to progress at a rapid rate once these lesions reach the occlusal surface by attrition, it would be rare to be required to restore such a deep cavity as most are covered occlusally by normal cementum. Also, as these lesions are so widespread there has to be some protocol for selecting appropriate cases for restoration. Individual cases with large, grade 3 or more lesions identified at the occlusal surface would seem reasonable, especially if on a Triadan 09 tooth. In the 8% of infundibular caries lesions that are fully aplastic, techniques for deep cavity restoration are required.

Little has been published on the efficacy of infundibular restorations although the author has a case series analysed statistically of 188 cases with 6 year follow up, and results show that that with careful case selection, meticulous cavity preparation and good technique results can be excellent but further critical review is essential to assess against negative controls. In summary restorations are most likely to be recommended for:

- Grade 3/4 (or 3-4/5) lesions with probing depth  $\geq 10\text{mm}$
- (Maxillary) 09 teeth or less frequently 06 teeth
- Age group 10-16 most common

It would be most unusual to find IHC representing significant risk in e.g. 07 teeth in young horses (not impossible, just much less likely than older ages and 09 teeth).

Restorations of infundibula would generally be classified as intra-coronal and Class I (GV Black classification, 1908). To avoid over-treatment of lesions unlikely ever to become clinically problematic, the author has advocated selection of Grade 3 lesions, with depth of cavity at least 10mm, or those identified as having deep or aplastic infundibula on CT or intra-oral radiography. These will be commonly 09 infundibular lesions or rostral 06 lesions. Imaging prior to any restorative procedure is important to identify potential pulp involvement and make a decision on root canal therapy, restoration or extraction. Orosopic imaging before and during the procedure for assessment of cavity preparation and guidance is considered essential by the author.

A pre-requisite for any restoration procedure is a properly prepared cavity, free from loose debris or necrotic dental tissue. The technique of 'air abrasion' alone to remove all necrotic debris from infundibular cavities has been shown to be ineffective. However, when used in combination with a water-cooled high speed hand-piece it can be effective in aiding removal of debris (150-200psi, using prophy powder or 25-30micron aluminium oxide powder). Following debridement, flexible nickel-titanium H-files are used in combination with commercial root canal file lubricants, deep cavities can be well prepared removing all necrotic debris. Repeated and diligent observation of the cavity with a mirror or preferably endoscope, combined with repeated debridement, instrumentation with files and flushing will result in a well-prepared cavity. Repeated used of H-files, canal lubricant (e.g. EDTA/carbamide peroxide<sup>b</sup>) and NaOCl irrigation is a very effective means of cleaning and disinfecting stubborn necrotic debris from deep cavities. Following complete debridement and disinfection, the cavity is flushed with water or saline, and air-dried. Restoration is performed under oroscopic guidance using a flowable dual-cured microhybrid resin composite using 19G needle extensions of the standard mixing tips and a layered technique to build up the restoration from the apical aspect of the cavity.

Occasionally it will be evident that there is an apical breach of enamel, and the instrumentation process will result in a file extending beyond the apical enamel, possibly to the apex of the tooth. Positional radiographs with the file in situ are then required, and an alteration of technique to a root canal procedure is required, placing a temporary cement restoration over a cavity liner, such as calcium hydroxide paste. The client should be warned of the implications of this, and extraction considered. Cases can however be treated successfully and the author has published 6 such cases in young horses with signs of apical abscessation, treated conservatively by infundibular disinfection and restoration.

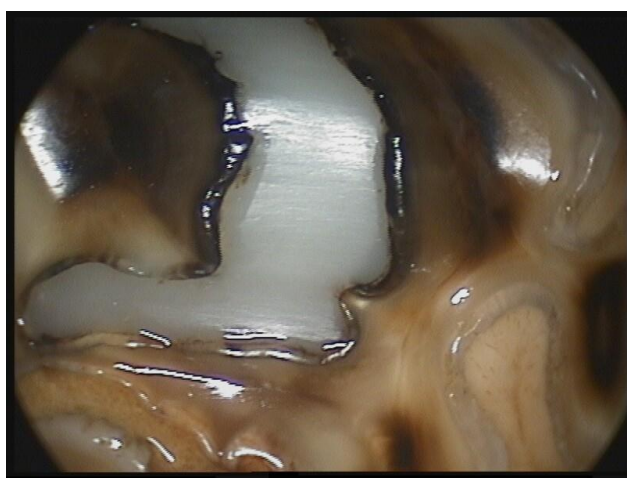


Fig. 17. Orosopic image of infundibular restoration (rostral infundibulum 109) 15 months post placement – note the fine wear lines from normal attrition across the surface of the restorative, and slightly protruding enamel wearing more slowly than the surrounding dentine and restoration, as in a normal tooth.



Fig. 18. Oroscopic image of restored 209 tooth 6.5 years post restoration – some material has been lost but there is minimal caries , normal attritional wear and no apical disease was evident.

### Periodontal Disease

Periodontal disease is a major cause of tooth loss in small animals, humans and equines. Equine periodontal disease has been documented in several studies and reports since the early 1900's and in 1905 was described as 'the scourge of the horse'. Baker (1970) recorded the prevalence of periodontal disease in the over 15 yr age group to be 60%, and Waffa (1988) found it to be 37% overall amongst all age groups with again 60% this time in horses over 20yrs of age. Ireland *et al* (2011) found a 45% prevalence in horses over 15 years of age but suspected that periodontal disease was probably under-reported in this study. Similar figures have been found in donkeys with duToit *et al* recording an overall prevalence of 50% suffering from periodontal disease.

Personal observations by the author and anecdotal evidence from other equine practitioners are that increasing numbers of periodontal disease cases are being identified and presented for treatment. This is likely to be due to an ageing population of domesticated horses and better identification of dental diseases generally.

### Anatomical and Physiological Considerations

The horse has a radicular hypsodont dentition with permanent eruption of a finite reserve crown, and root formation that occurs as the horse ages. The periodontium comprises four structures – the gingiva, periodontal ligament (PDL), Sharpey's fibres, the peripheral cementum and the dental alveolar bone. The PDL is a fibrous connective tissue interposed between the dental cementum and the alveolar bone. It provides fixation of the tooth and withstands masticatory forces at the same time. These functional requirements are met by a unique architecture of a collagen fiber bundle system in combination with an ample blood vascular system. Due to the lifelong eruption of the equine tooth the equine PDL has to provide mechanisms for continuous tissue remodeling and tissue repair.

There is constant remodelling of the periodontium resulting in a living vascular structure, which constantly detaches and reattaches to the tooth to 'lift' it towards the occlusal surface. Once the cementum of the periodontium becomes supra-gingival, it loses it's ability to remodel and therefore at this stage can be considered virtually inert. There is however a marked increase in the amount of cementum produced by the periodontium at the alveolar crest resulting in a 'thickening' of cementum here that results in natural 'bridging' of the interproximal spaces.

## Pathogenesis of periodontal disease

There has been little published data regarding the aetiology and pathogenesis of equine periodontal disease. It is however widely accepted that, as in other species, untreated periodontal disease is in time likely to progress to periodontal ligament disruption and eventual tooth loss. The prevalence of periodontal disease has been quoted to be over 60% in horses aged 15 years or over; empirical observations by the author show this to be conservative. Periodontal disease can develop in horses of any age mainly as a result of developmental malocclusions and domestication and may be severe in horses as young as 4 years. Horses are skilled eaters and are able to selectively avoid masticating in painful areas of the mouth. Development then occurs as a vicious circle of focal overgrowth, pain, feed stasis and inflammation resulting in deterioration of overgrowths due to lack of mastication at the affected site. Eventually horses will become symptomatic as they decompensate, but as with most modern interventionist dentistry it is the job of the modern dental practitioner to identify the changes well in advance of this stage.

Most periodontal disease occurs secondary to other developmental or pathological conditions: diastema, dental displacements and rotations, fractures or functional abnormalities. Primary periodontal disease is rare. Horses have a pellicle layer which in areas of stasis may develop to a thick plaque like substance which in turn appears to occasionally cause marked peripheral caries with destructive lytic lesions however these have yet to be linked definitely with periodontal disease (see image). Despite the current lack of evidence, it seems logical that loss of interproximal cementum and dentine through such lytic caries would result in the stasis of food in the resultant space.



*Fig. 19. Severe lytic peripheral caries on lingual aspect of Triadan 310*

Feed types play an important role. The softer the feed, the larger the range of motion and the less crushing motion used. With soft feedstuffs, such as green grass pasture, a wider range of mandibular motion is used. Feed material is ground in a circular pattern, rather than crushed. This wide range of motion creates a large amount of soft tissue contact, gingival crevicular fluid (GCF), and saliva flow. The soft tissue contact together with saliva mechanically cleanses the teeth, thus preventing feed stasis. Horses secrete 50 ml/min of saliva from the parotid salivary gland. Salivary flow is stimulated by mastication. Without mastication, salivary flow is limited to that amount needed to maintain a moist intraoral environment. Horses in free-range situations feed for approximately 14 hours per day. By calculation only, not by direct measurement, that would mean horses create over 40 litres of saliva per day when feeding on grass from one gland alone. The previously mentioned benefits of saliva, mechanical cleansing, acid buffering, and antibody production are very important in prevention of gingivitis and periodontal disease when its flow is both voluminous and continuous. GCF fluid flow and its advantageous components, leukocytes, and antibodies are important also. Together, these two host defense mechanisms provide a substantial barrier to infection.

When horses consume harder feeds such as hay and grain, all the above parameters change in favor of the development of periodontal disease. Range of motion is reduced; teeth and parts thereof become protuberant; feed stasis occurs; and decay and the cascade is set in motion. The saliva that is produced is absorbed by the dry feed to some degree, thus reducing its effectiveness. Because the time of feeding is reduced, the total daily saliva and GCF production is dramatically reduced. The net result is a much greater time for static feed material to decay. Reduced soft tissue contact and reduced range of motion leads to decay of feed material, thus creating the environment for periodontal disease to flourish.

#### Examination and Assessment

Oral examination is carried out as previously described paying particular attention to interproximal spaces, and the buccal and lingual aspects of cheek teeth for gingival recession, impacted food material with or without occlusal diastemas (Fig 2).

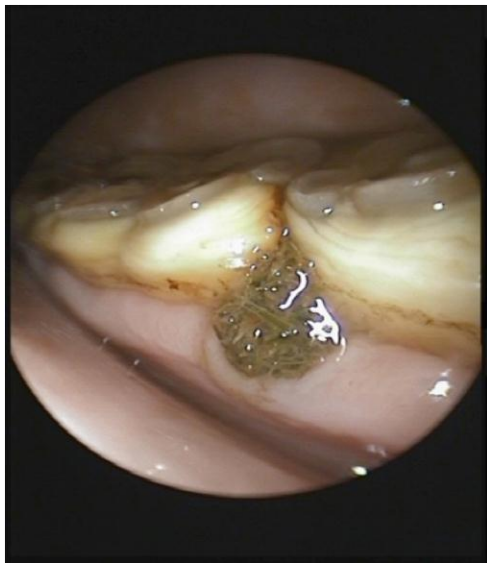


Fig 20. Oroscopic image of early periodontal disease; food impaction with no occlusal diastema.

Following this, meticulous cleaning of diastemas is required using picks, crocodile forceps, flushing units and scalers. Peripheral caries lesions should be debrided. This process can be painstakingly slow, and may require significant levels of sedation and analgesia including regional nerve blocks on occasion.

The condition of the tooth and periodontium is examined for gingival inflammation and erosion, condition of sulcular epithelium, pocket depth and mesial/distal length, condition of the supragingival and subgingival cementum, attachment loss, and tooth mobility. The interproximal spaces should be examined particularly carefully to assess the type of junctional contact between the teeth i.e. parallel or otherwise (see Fig 3), and for displacements, rotations and fractures.



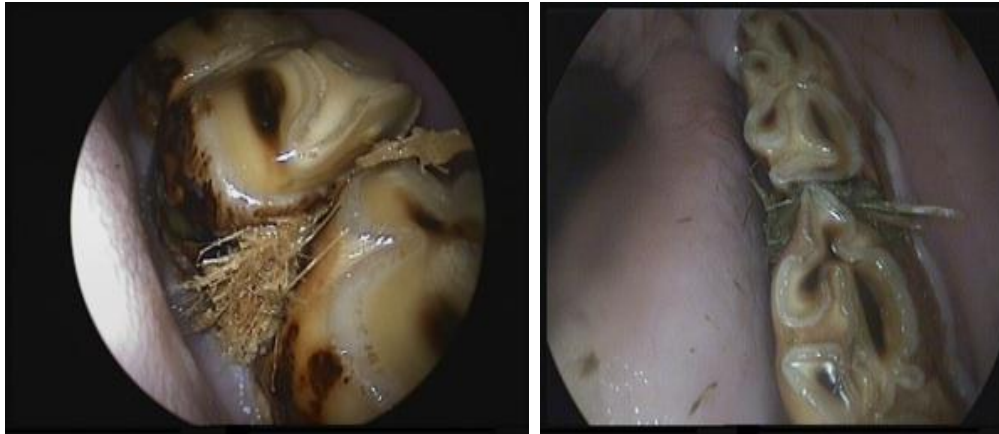


Fig 21. Orosopic images showing variations of the anatomy of the interproximal space leading to food impaction

Periodontal disease assessment should be performed using a combination of examination using a mirror, oroscopy and radiography to give a tooth mobility / periodontal disease index (Wiggs & Lobprise) as follows:

- Grade 0 no PDL loss
- Grade 1 Gingivitis, no attachment loss
- Grade 2 <25% attachment loss
- Grade 3 <50% attachment loss
- Grade 4 >50% attachment loss

#### Treatment and Prevention

In recent years there have been a number of treatments advocated for the management and treatment of equine periodontal disease. These include corrective dental floating, removal of necrotic food material impacted in interproximal spaces, use of abrasive prophylaxis powders mixed with water and propelled by pressurised gas, widening of interproximal spaces using motorised burs, application of temporary 'patches' of dental impression material, application of semi-permanent interproximal 'bridges', the use of perioceutic agents and exodontia.

The treatment may be broken down into simple steps which will cover all types of periodontal disease:

1. Meticulous cleaning of interproximal spaces and periodontal pockets
2. Short term measures to improve periodontal health
  - a. Corrective floating, equilibration
  - b. Perioceutic agents e.g. doxycycline gel
  - c. Temporary 'patches' to prevent immediate influx of food material
3. Long term measures to prevent further periodontal disease
  - a. Diastema widening
  - b. Bonded hard 'bridges' to occlude diastema more permanently

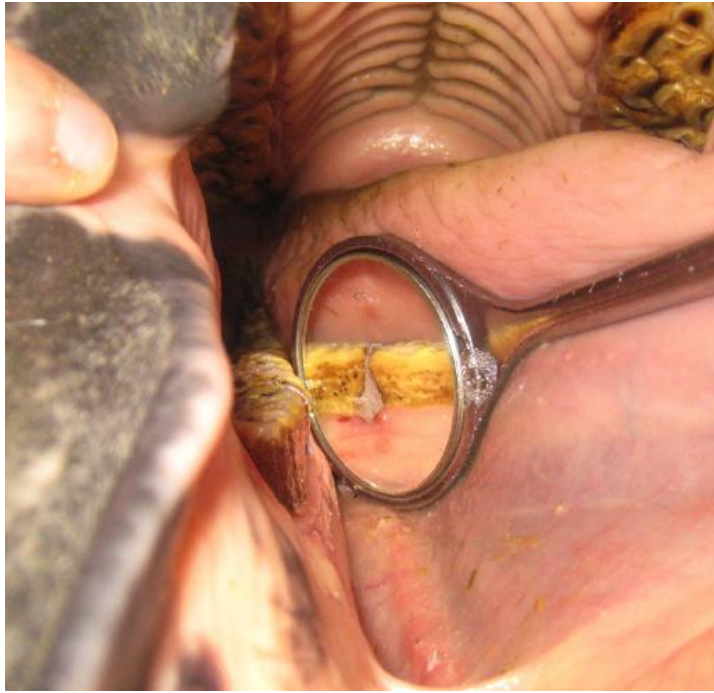


Fig 22. Cleaned diastema and periodontal pocket with temporary vinyl polysiloxane 'patch'

#### Diastema widening

Mechanical widening of diastemata can be a very successful procedure in the short-medium term however consideration needs to be given to the anatomy of the interproximal space and the potential proximity of the pulp horns, and the risk of thermal trauma to pulpar tissue by using a mechanical rotating burr in such close proximity to sensitive pulp. Water cooling should be mandatory for diastema widening, and if tools do not incorporate a water irrigation channel, water may be flushed using a variety of other more crude methods e.g. fluid pump, syringe and extension set or diastema irrigation pump.

An interproximal space of approximately 6mm or more in width is unlikely to trap food material resulting in peripheral caries, and periodontitis. Therefore, logically narrow diastemata will require more dental tissue to be removed in order to achieve this width than wide diastemata.

Bettiol and Dixon (2011) showed that:

Pulp horns (PH) are usually 5.75mm from IPS

PH may be 1.3mm from IPS

'Valve diastemas' should be safer (2.5x wider at base than occlusally)

*Higher risk of pulp exposure caudally* (caudal aspect CT, PH 2,4)

→ remove more from mesial/rostral aspect of CT behind IPS

#### The author recommends

- Polyfloat, Powerfloat, – water cooling essential / highly recommended
- 4-6mm burr – diamond, carbide
- Start with narrow burr (3-4mm)
- Keep checking alignment, oroscope preferred
- Start occlusally under visual control
- Or gingivally with conical burr

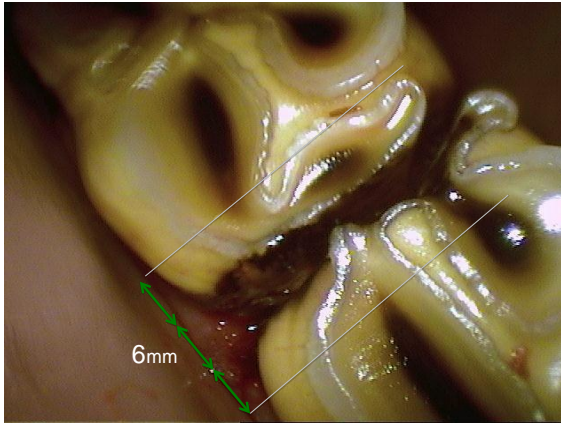


Fig. 23. Oroscopic image demonstrating how narrow diastemata may require potentially damaging amounts of dental tissue to be removed to allow a therapeutic interproximal space



Fig. 24. Image showing successfully widened diastema

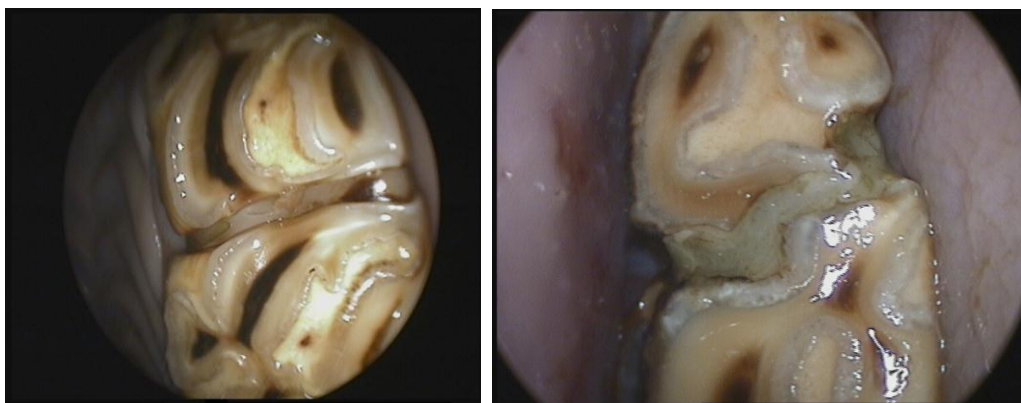


Fig. 25. Oroscopic images of interproximal 'bridges' using bonded resin polymer to occlude diastema

Further research and long-term studies are required in this area to ascertain the effectiveness and safety of these treatments. Personal experience has shown that after careful examination and assessment of cases a combination of the above techniques, initially utilising minimally invasive procedures can produce excellent results. For more severe and chronic cases, the use of interproximal 'bridging' or widening of diastema can also be very effective. Bridges may be:

- Polysiloxane applied only as temporary 'patches'
- PMMA (bone cement) (Klugh 2006)
- Plaster of Paris + gentamicin /
- Temporary crown/bridge material (Pearce)

'VOCO Structur 2' – Cold polymerising paste-paste resin polymer

Excellent handling and bonding to teeth

Chemically bonded to debrided teeth using total etch technique

Prevent food accumulation in IPS

- Long term success 55% mandibular, 86% maxillary (Pearce 2013, unpublished data)

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