Endodontic Considerations of Equine Incisor and Canine Teeth

David O. Klugh DVM, FAVD/Equine

Author's address: Columbia Equine Hospital, 27841 SE Orient Drive, Gresham, OR 97080; E-mail: equinedental@earthlink.net.

Take Home Message

The complexities of the morphology of the pulp canal of equine incisors require sophisticated techniques, materials and instrumentation for their treatment. It is important to understand the age-related changes that occur in pulp tissue and in the dentin that fills the canal. The equine pulp is a dynamic, responsive tissue. The response to outside influence varies with age and severity of insult.

Introduction

Endodontics is the branch of dentistry that addresses the diseases and conditions of the dental pulp. Endodontic treatment of pulp disease is a standard treatment in man and small animals. Conventional endodontic treatment is performed via access from the occlusal portion of the crown of the tooth. Surgical endodontics is performed via bone flap created to allow access to the apical portion of the tooth root. In equine dentistry, only the incisors and canines are routinely amenable to conventional techniques, while premolar and molar pulp disease is treated by surgical access.

Indications commonly found in equine dentistry for endodontic treatment include non-vital pulp exposure, tooth discoloration, traumatic injury, problems secondary to periodontal disease, radiographic evidence of apical disease, and iatrogenic vital pulp exposure. Affected teeth may or may not be painful. Clinical signs of dental pain may be presented as dysmastication, bitting pain, or behavioral change. Fractured teeth are always painful until the pulp dies, or the tooth is repaired.

Consequences of failure to deliver treatment include secondary fractures, apical extension of disease to include periodontal structures, shifting of the tooth, and premature exfoliation. Though not proven, extension to distant tissues or organ systems as occurs in other species[1] may be a consequence of endodontic disease in equine patients. The diseased tooth can fracture because it is more friable than normal. Apical disease is diagnosed by radiographic examination, which can reveal lucent areas around affected apices. Shifting, or orthodontic movement of the tooth within the alveolus, occurs when the diseased tooth suffers loss of periodontal attachment as the inflammatory process extends from the pulp through the apex and involves the surrounding tissue. If attachment loss becomes severe enough, the tooth may exfoliate.

Development

Embryologic development of the pulp involves the condensation of ecto-mesenchymal tissue under the inner enamel epithelium of the bell stage. The multicellular character of the pulp develops thereafter. The function of the pulp begins early and includes formation of odontoblasts which produce dentin, and maintenance of the health of the tooth via the neurovascular bundle which is derived from embryologic pulp cells.[2]

Pulp chambers fill with dentin and elongate as the hypsodont root develops. Molars and premolars develop complex branching of pulp horns.[3] Teeth 7 through 10 (Triadan system) generally have 5 pulp horns. Teeth 6 and 11 have at least 6 and frequently more pulp horns. Maxillary molars and premolars usually have 3 roots, while mandibular molars and premolars usually have 2 roots. The newly erupted cheek tooth has a large common pulp chamber that eventually separates into a single chamber for each root. This separation takes 4 to 5 years to complete.[4] Until then, the pulp chambers and therefore the pulp horns can communicate. This anatomical characteristic is important when considering treatment options for endodontic disease.

Anatomy of Incisor Pulp

The embryologic development and tissue origins of incisors and canines, and the process of filling of the pulp with dentin are the same as for cheek teeth. Incisors and canines have been thought to have a single cone shaped pulp chamber.[5] However, in a study of the conformation of incisor pulp chambers and the changes that occur with age, several other characteristics were noted.[6]

In the newly developing and erupting equine incisor, the pulp chamber begins as a cone-shaped cavity surrounded by enamel and dentin. As the adult tooth begins to undergo attrition, the brown stained secondary dentin (dental star) evidences the labio-lingual flattening of the cavity. In some cases, the cavity is separated into two parts by the infundibulum. As the tooth erupts and the infundibulum disappears, the dental star becomes more rounded.

The radicular hypsodont tooth elongates by root development. The root is compressed laterally by the conformation of the bone in which it is anchored. More compression is seen in mandibular than maxillary incisors. This lateral compression creates two divergent pulp canals that are evident when the tooth is 1.5 to 2 years of age. The mandibular second incisors are the most commonly affected teeth. Divergence occurs in individual incisor teeth in all locations. The average length of diverging canals in this study was 21 mm for the labial canal and 13 mm for the lingual canal. The lingual canal fills with dentin at a faster rate than the labial, thus becoming obliterated before the labial canal.

Lateral compression causes the canal to assume shapes that are not amenable to standard endodontic instrumentation, such as a ribbon or figure “8” shape pulp canals. The need for chemical debridement and sterilization is thus emphasized.

Other anatomical variations encountered included:

1. Single apical communication with both canals.
2. Lateral accessory openings.
3. Significant variation in location of lateral apices.
4. Canals diverging and then coalescing.
5. Incomplete dentinal filling of pulp canals.

The morphology of the pulp canal of equine incisors is similar to the pulp canals of other species. The concepts of diverging canals and varying morphologies of the canal are important in canal preparation, sterilization and obturation, just as they are in other species. The difference is that the scale is much larger.

Dentinal Deposition

Dentinal deposition within the pulp by odontoblasts consists of various structural arrangements.[7] Primary dentin is generally considered to be that laid down until the external form of the tooth is completed. Secondary dentin is seen in two different conformations in equine teeth. Secondary dentin is divided into regular and irregular according to its histologic appearance.[3] Regular secondary dentin is produced by odontoblasts throughout the life of the tooth. The odontoblasts withdraw centrally as this tissue is produced. Irregular secondary dentin is deposited as the last stage of dentinal filling of the pulp. Tertiary or reparative dentin is deposited in areas where the pulp has been irritated.

Dentin appears to be deposited at a standard rate. This rate may be genetically predetermined. Outside forces, primarily those of mastication, can cause alteration in the rate of dentinal deposition. A decrease in masticatory force can result in a depressed rate of dentinal deposition. The subocclusal pulp in this case remains vital and can be inadvertently exposed during routine dental care. If masticatory forces increase beyond a threshold, or if the tooth is otherwise damaged by events such as traumatic fracture, the character of dentin deposited can change. More severe inflammation can result in a change in character of dentin to tertiary (reparative) dentin. This can be so significant that the canal becomes completely filled or obliterated.

If inflammation occurs to such a degree that the pulp undergoes necrosis, further eruption and attrition of the tooth will exposes the non-vital pulp. This can happen as a consequence of changes in masticatory force. The resulting non-vital pulp exposure is most commonly found in older patients, but the proportion of pulp suffering necrosis is highly variable. In some cases very little pulp is damaged, with much of the remaining pulp canal obliterated by tertiary dentin. Other cases have a greater proportion of pulp necrosis. Some have complete pulp necrosis with an apical lucency appearing on radiographic examination, but this is the exception.

Younger patients whose pulps are larger, more cellular and highly vascular are more able to withstand inflammatory pulpar insults. In these cases, when damage results in necrosis

of the coronal pulp, the remaining viable odontoblasts create a dentinal bridge that protects live pulp tissue. All these characteristics are visible only radiographically. It is imperative that the practitioner obtain quality radiographs when presented with a potential pulp disorder.

In summary, there are two pathways in the development of a non-vital pulp exposure. The first is death of the aging pulp from mastication forces, followed by attrition and eruption exposing the necrotic pulp. The second is direct exposure of a vital pulp and subsequent severe inflammation and infection that the pulp cannot withstand.

Age-related changes contribute to the pulp's loss of ability to respond to insult. Aging results in decreased cellularity and reduced vascular supply, along with overall reduced volume. The vascular supply is limited, so the ability of the pulp to withstand and respond to inflammation is reduced. Because the pulpal volume is reduced, it has less space in which to swell when irritated. These principles combine to make the aging pulp less able to withstand increased physiologic insult. This leads to necrosis and eventual pulp exposure. The same forces that are easily managed in the young pulp can potentially cause pulp necrosis in older horses.

The consequence of pulp exposure is significant. The tooth becomes infected and painful. The tertiary dentin of a dentinal bridge or in pulp canal obliteration may not completely protect the viable pulp. Endodontic treatment further protects the viable pulp, while debriding infected structures to prevent further bacterial spread. Relief of pain results from removal of infection and necrotic tissue and complete obturation, or filling, of the canal.

Diagnosis

Diagnosis of pulp disease requires thorough clinical and radiographic examination. Clinical examination is performed with the use of a speculum in a sedated patient, a good light source, a dental mirror and a dental explorer.

The explorer is drawn across the tooth surface. The tip of the explorer tissue will "catch", causing it to "stick" to the decayed dental tissue because the curvature of the tip forces the point into the softer decayed tooth structure. If decayed dentin is found, the examiner can attempt to insert the explorer tip into the pulp horn. Insertion in necrotic pulps is easy. If a pulp exposure is found, an endodontic file can be inserted to measure the depth of the open pulp.

Radiographic diagnosis of pulp disease of cheek teeth is challenging. Routine radiographs, including intraoral views yield good information regarding apical disease. Due to the complexity of the folding pattern of equine cheek teeth, pulp disease in the region of the reserve crown does not lend itself to easy diagnosis. Interpretation of incisor radiographs is more straightforward. Apical lucencies are easy to visualize as are changes in pulp density.

Contrast radiography of any pulp exposure using gutta percha points or an endodontic file can assist in determining whether the pulp is open or obliterated.

Treatment

Horses with pulp disease are treated by endodontic (root canal) therapy. Conventional root canal therapy (RCT) in incisors and canines can be employed. In cheek teeth, conventional RCT can be used in selected cases, but in general, the treatment of choice is surgical apicoectomy. The technique for RCT is thoroughly described elsewhere. (See further reading list.) Techniques are adapted for use in equine teeth.

Follow up radiographic examination should be used to determine repair of apical lucencies at 6 and 12 months after treatment. Thereafter annual radiographic examination is recommended.

Potential complications of treatment include failure of apical seal with extension of infection to involve periodontal structures resulting in tooth shifting or exfoliation, spread of infection to distant tissues and organ systems, and tooth fracture due to increased friability of the diseased tooth.

References

1. Wiggs RB, Lobprise HB. Basic endodontic therapy. In Wiggs RB and Lobprise HB (eds): *Veterinary Dentistry: Principles and Practice*. Philadelphia: Lippincott-Raven, 1997:280 – 324.
2. Emily, P. Endodontic diagnosis in dogs. In Holmstron, S (ed). *Veterinary Clinics of North America.* Philadelphia. WB Saunders. 28(5) 1998:1189- 1202.
3. Dacre I. A pathological study of equine dental disorders. PhD thesis. The University of Edinburgh.
4. Kirkland KD, Baker, GJ, Marretta, SM, Eurell, JC, Losonsky JM. Effests of aging on the endodontic system, reserve crown, and roots of equine mandibular cheek teeth. AJVR 57(1). 31-38.
5. St. Clair, LE. Teeth. In *Sisson and Grossman's: The Anatomy of the Domestic Animals*. Volume 1. 5th edition. Getty, R ,editor. WB Saunders, Philadelphia:460.
6. Klugh, DO. Equine incisor pulp canals: a closer look. In *Conference Proceedings.* 17th Annual Veterinary Dental Forum. 2003:200-202.
7. Muylle S, Simeons P, Lauwers H. A study of the ultrastructure and staining characteristics of the 'dental star' of equine incisors. Equine Vet. J. (2002) 34(3):230-234.

Further reading

Pathways of the Pulp by Stephen Cohen and Richard C. Burns
Veterinary Dentistry: Principles and Practice by Robert B. Wiggs and Heidi B. Lobprise
Small Animal Dentistry by Colin E. Harvey and Peter P. Emily

Continuing education available on this subject

AAEP Advanced Dental Wetlab, Lexington, Ky. Sept. 6 and 7, 2006. www.AAEP.org.
Minnesota Equine Dental Symposium (MEDS 2), University of Minnesota College of Veterinary Medicine, St. Paul, Mn. March 2007 www.cvm.umn.edu/outreach/events/meds.html.
Annual Veterinary Dental Forum. Portland, Or. Sept. 21 – 24, 2006. www.veterinarydentalforum.com