

# **Histological and Ultrastructural Anatomy of Equine Dentition**

Ian T. Dacre, BVSc, PhD, MRCVS

Author's address: IVABS, Massey University, Private Bag 11222, Palmerston North, New Zealand.

## **Endodontic Anatomy**

Knowledge of endodontic status (notably whether pulp has been replaced by dentine) is important in equine dentistry as it may give an indication of how much crown may be removed during clinical crown reduction before encountering vital pulp. This is important in conditions such as step-mouth where large (>5mm) crown reductions may be necessary. Knowledge of endodontic status (notably whether pulp has been replaced by dentine) is important in equine dentistry as it may give an indication of how much crown may be removed during clinical crown reduction before encountering vital pulp. This is important in conditions such as step-mouth where large (>5mm) crown reductions may be necessary (ref Dacre AAEP 2006 'How to' paper). Additionally, when performing clinical endodontic (i.e. pulp and pulp chamber) procedures, knowledge of the anatomy of the pulp chamber or horn must be known to improve the likely success of any intervention. "The hard tissue repository of the human dental pulp takes on many configurations that must be understood before treatment can begin" (Burns & Herbranson 2002). This quote by Richard Burns, editor and author in 'Pathways of the Pulp', a text detailing the 'art and science' of human endodontics, may indicate why equine endodontic therapy currently yields poor results - with one study reporting 58% (11/19) of CT endodontic procedures to have short-term success (i.e. 12 months post procedure) and only 40% (6/15) having long-term success (i.e. >24 months post-surgery) (Schramme et al. 1999). Knowing the complete endodontic anatomy of equine dentition, which has only recently been fully determined, may help improve the likely success of equine endodontic interventions (Dacre 2004b).

Pulpal insults can be caused by heat, pressure, vibration, desiccation, chemical exposure, and bacterial infection (Shafer et al. 1983). Heating of the pulp can cause various histopathological changes, such as burn reactions at the periphery of the pulp, including formation of 'blisters', protoplasm coagulation, and expansion of liquid contained in the pulp and dentinal tubules with resultant increased outward liquid flow from tubules (Castelnuovo & Tjan 1997). These reactions can lead to vascular pulpar injuries with subsequent tissue necrosis (Raab 1992). The potential for thermal pulpar insult to be caused by modern motorised equine dental equipment has been studied *in vitro*, with the investigators concluding that if motorised dental equipment is used incorrectly, equine dental pulp may suffer thermal trauma (Baker & Allen 2002).

Aside from the potential risks associated with thermal pulpar insult, aggressive dental reductions that do not expose a pulp cavity, such as full 'bit-seating' (i.e. reducing and rounding off the rostral aspects of the O6s for allegedly better bit comfort), have the

potential to expose sensitive dentine to the oral cavity (Kempson et al. 2003). In normal circumstances, dentine in contact with the oral cavity is sclerotic (i.e. the dentinal tubules have been occluded either by calcification of the odontoblast process, or by the tubule being filled by a 'smear' layer [ground dental dust]). Kempson et al. showed that this sclerotic layer could be removed completely with modern dental equipment, exposing sensitive odontoblast processes. To fully appreciate implications of dental interventions, a sound knowledge of ultrastructural dental anatomy is essential.

### **Dentino-Pulpal Complex**

The dentino-pulpal complex is so termed because of the intricate association between the dental pulp and surrounding dentine (Jones 1990; Torneck 1998). The dental pulp consists of an intricate network of loose connective tissue supported on a fibrous skeleton containing blood vessels, lymphatics and nerves that enter and exit at the dental apex. The outermost layer of pulp consists of odontoblast cells, responsible for continuous dentine production, which begins immediately before the formation of the enamel 'scaffolding' within the dental sac, and continues throughout the life of the equine tooth (Ferguson 1990).

Odontoblast cells leave long tubular odontoblast processes within the dentine (dentinal tubules) as they retreat centrally into the pulp, with progressive deposition of dentine by these structures (Kilic et al. 1997b; Lowder & Mueller 1998; Muylle et al. 2001). Equine odontoblast processes extend horizontally to the amelodentinal junction and vertically to the occlusal surface of equine teeth (Kilic et al. 1997a; Kilic et al. 1997b). It is believed that odontoblast processes may transmit normal (physiological) stimuli from the occlusal or interproximal surfaces via changes in fluid pressure within the dentinal tubule and that this can regulate the rate of dentine deposition in the underlying pulp or transmit noxious stimuli (e.g. from physical, chemical or thermal trauma or caries) to initiate tertiary dentine production (Kempson et al. 2003).

The progressive attrition of equine teeth at a rate of *circa* 2-3 mm/year necessitates the continued deposition of secondary dentine to prevent pulpal exposure. This metabolically demanding process means that a significant blood supply must be prolonged to the dentinogenic zone of the pulp cavity well into a horse's life (Dixon & Copeland 1993). In contrast, the apical foramina that carry the pulpar blood supply to brachydont teeth are narrowed by dentine deposition within the pulp canal, restricting maximal blood vessel diameter at a relatively early stage of the tooth's life (Berkovitz & Moxham 1981). If appropriately stimulated, odontoblasts may produce dentine throughout their lifespan (Fawcett 1987; Ten Cate 1998a).

### **Classification of Equine Dentine**

There is confusion with the classification of human dentine as being either primary, secondary or tertiary. Ten Cate (1998a) describes primary dentine as the dentine laid

down until the external form of the tooth is completed, with secondary dentine being laid down thereafter. Torneck (1998) defines secondary dentine as that which develops after root formation has been completed. Neither of these definitions are applicable to equine (hypsodont) teeth, with their prolonged deposition of peripheral cementum on the reserve crown and roots (Kirkland et al. 1996), thereby changing the external shape of the tooth (especially its apical aspect) throughout the tooth's life, possibly for up to 40 years (Dixon 1999). With equine dentition, it is more accurate to use a third definition of secondary dentine: *the dentine that is deposited once a tooth is in full occlusal contact* (Kierdorf & Kierdorf 1992).

In equine primary dentine, the odontoblast processes are surrounded by dentinal tubules, which are filled with intratubular dentine, which is present only in primary dentine (Kilic et al. 1997b; Muylle et al. 2001). A thin layer of intertubular dentine, in turn, surrounds the intratubular dentine.

Secondary dentine is classified as being either regular or irregular, with irregular secondary dentine also being referred to as reparative, reactive or tertiary dentine (Kilic et al. 1997b). Following dental eruption, regular secondary dentine is laid down by odontoblasts throughout most of the life of the tooth, with the odontoblast cells withdrawing centripetally (towards the centre of the pulp) from the previously laid down primary dentine. Secondary dentine is continuous with primary dentine, sharing the same odontoblast process within a continuation of the same dentinal tubule. Under normal circumstances, both in brachydont and hypsodont teeth, 'regular' secondary dentine is laid down within the pulp chamber by the odontoblasts until the pulp chamber is almost completely occluded.

Irregular secondary equine dentine (also referred to as reparative, reactive or tertiary dentine) has previously been classified as dentine that is laid down in response to noxious stimuli (Muylle et al. 2002). However, recent work has shown irregular secondary dentine to be present in all (n= 100) grossly normal equine teeth examined, with no evidence of prior exposure to noxious stimuli (Dacre 2004a). This secondary irregular dentine is laid down in the most central part of the pulp horn when this region is undergoing its final physiological stage of pulp replacement by dentine. The continued formation of both regular and irregular secondary dentine prevents pulpar exposure on the occlusal surface.

Tertiary dentine is laid down focally, in response to specific local noxious stimuli (Ten Cate 1998a). It may have some tubules that are continuous with those of secondary dentine; tubules that are sparse or distorted; or no tubules at all (Torneck 1998). Cells may be trapped within this rapidly laid down tissue, and this type of dentine is termed (tertiary) osteodentine. Tertiary dentine may be further subdivided into reactionary and reparative dentine, with the former being laid down by pre-existing odontoblasts and the latter by newly differentiated odontoblast-like cells from within the pulp.

Where areas of dentine have failed to mineralise sufficiently, interglobular dentine occurs. Being a failure of mineralisation rather than of matrix formation, the normal

architectural pattern of the dentinal tubules is present, running through zones where the globular calcospherites have failed to fuse into a homogenous mass within the maturing dentine. Interglobular dentine in human teeth is thought to arise due to vitamin D deficiency or following exposure to high fluoride concentrations at time of dentine formation (Torneck 1998).

Sclerotic dentine is formed when dentinal tubules become occluded with calcified material. The degree of dentinal sclerosis increases with age in human teeth that have no clearly identifiable external stimuli for this process, and this occlusion occurs by several different mechanisms. In horses, where dentine is exposed on the occlusal surface, we would expect the presence of a high degree of dentinal sclerosis to prevent bacterial invasion down such exposed dentinal tubules. Kilic (1995) and Kempson et al. (2003) demonstrated the presence of large numbers of odontoblast processes on the occlusal surface of equine dentine. It was suggested by Kilic that these processes were calcified (i.e. sclerotic remnants of odontoblast processes), however more recent work by Kempson et al. suggested there were viable odontoblast processes present at this site following recent dental rasping.

The presence of open dentinal tubules and odontoblast processes on the occlusal surface has been proposed to be part of a sensory mechanism to limit the stress applied to teeth (with fluid movement within dentine during tooth compression stimulating pulpar baroreceptors) and thus helping to facilitate discrimination of particle hardness during mastication (Paphangkorakit & Osborn 2000). This process was previously believed to be principally regulated via mechanoreceptors within the periodontal ligament.

Dentine is a sensitive tissue, and in a comparison of 40 contra-lateral pairs of human teeth, wet dentine exposed through grinding was found to be much less sensitive than recently exposed fractured dentine, or dentine that became dehydrated following exposure to air (Johnson & Brannstrom 1974). This, in part, may explain the absence of signs of obvious pain during mastication in horses that have large areas of exposed dentine on their teeth.

## **Enamel**

Enamel is the hardest substance in the body but is also very brittle (Kilic et al. 1997a). In both brachydont and hypsodont teeth it consists of 95-98% mineral by weight, being mainly composed of calcium hydroxyapatite [ $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH}_2)$ ] crystals, although minute quantities of other (inorganic) salts are found within its matrix.

## **Classification of Equine Enamel**

Kilic et al. (1997b) defined three types of equine enamel according to the transverse appearances of their enamel prisms and the presence, amount and appearance of their interprismatic enamel. Equine Type-1 enamel contains alternating rows of oval-shaped prisms and thick interprismatic enamel plates, and is found adjacent to the amelodentinal junction. Equine Type-2 enamel consists of 'keyhole' to 'horseshoe' shaped prisms, with

little or no interprismatic enamel, and is located adjacent to the amelocemental junction. Three-dimensional prism decussation (interweaving) is present in equine Type-2 enamel, which is the main type of enamel in equine incisors. Decussation makes equine Type-2 enamel much more resistant to cracking and fracture than equine Type-1 enamel. A greater amount of equine Type-1 enamel is present in maxillary CT than in mandibular CT, with equine Type-2 enamel being the dominant form of enamel in the mandibular CT. Equine Type-3 enamel is inconsistently present as a thin layer at both the amelodentinal and amelocemental junctions, where its interprismatic enamel forms a honeycomb-like structure, with each 'cell' occupied by an oval shaped prism. It is less highly evolved than equine Type-1 and 2 enamel (Kilic et al. 1997a).

## **Cementum**

Cementum is the softest of the three calcified dental tissues, being approximately 65% mineral, 35% organic, with calcium hydroxyapatite crystals being the principle mineral present (Kilic et al. 1997c; Ten Cate 1998b). Peripheral cement is deposited both directly and indirectly onto resorbed and unresorbed enamel, with a thin calcified layer interposed in areas of indirect mineralisation.

Cementum is a specialised calcified connective tissue that provides anchorage for the fibres of the periodontal ligament and also a means for dental eruption in both brachydont and hypsodont teeth. Its higher organic and water content confers some flexibility (like dentine), allowing it to give necessary support to the brittle adjacent enamel. In all horses (particularly as they become older), cementum also significantly structurally contributes to the size and strength of the crown and roots (Kilic et al. 1997c; Mitchell 2004).

Subgingival cementum is nourished by the periodontium, however, this vascular supply is lost following eruption (as part of the clinical crown) by more than a few millimetres, after which cementum may be considered an inert tissue (Mitchell 2004). Within the dental alveolus, and more specifically in the sub-gingival region immediately above the alveolar crest, cementum is deposited throughout the life of the tooth (Jones 1981; Mitchell 2004). Cementoblasts can also respond quickly to harmful stimuli by further rapid deposition of cementum.

## **Classification of Equine Cementum**

Once the thin cemental covering is worn from the occlusal surface of hypsodont teeth, exposing the secondary occlusal surface, equine cementum may be grossly classified as being either peripheral or infundibular (Kilic 1995).

Cementum can be classified on three levels namely:

1. Time of formation, being either primary or secondary (c.f. dentine formation)
2. Presence or absence of cells within its matrix (i.e. being either acellular or cellular)

3. Whether fibres within the matrix are intrinsic (i.e. resulting from cementoblast deposition) or extrinsic (i.e. Sharpey's fibres incorporated from the PDL)

This results in five categories of cementum (Freeman 1998):

1. Primary acellular intrinsic fibre cementum
2. Primary cellular extrinsic fibre cementum
3. Secondary cellular intrinsic fibre cementum
4. Secondary cellular mixed fibre cementum
5. Acellular afibrillar cementum

Under polarised light, undecalcified (ground) transverse sections of equine CT show two distinct regions to be present in peripheral cementum. Adjacent to the peripheral amelocemental junction, the crystalloid nature of the cementum is observed to be irregular in hydroxyapatite crystal orientation. This is similar to maxillary CT infundibular cement. Beyond this, its nature changes to become regular, with crystals having a similar concentric orientation. It is in this zone that 'peripheral lines' may be observed in decalcified transverse sections. These two zones of regular and irregular peripheral cementum are more pronounced near the occlusal surface in sections of older teeth.

## References

- Baker, G.J. & Allen, M. L. (2002) The use of power equipment in equine dentistry. (48th American Association of Equine Practitioners Conference Proceedings) 438-441.
- Berkovitz, B.K.B. & Moxham, B. (1981) Early stages of tooth development. Dental anatomy and embryology. 166-174 Blackwell Scientific Publications.
- Burns, R.C. & Herbranson, E. J. (2002) Tooth morphology and cavity preparation. Pathways of the pulp. 8 (7) 173-229 Mosby.
- Castelnuovo, J. & Tjan, A. H. (1997) Temperature rise in pulpal chamber during fabrication of provisional resinous crowns. *J. Prosthet. Dent.* 78 (5) 441-446.
- Dacre, I.T. (2004a) A Pathological, Histological and Ultrastructural Study of Diseased Equine Teeth. Royal (Dick) School for Veterinary Studies.
- Dacre, I.T. (2004b) Equine dental pathology. Equine Dentistry. 2nd (9) Elseiver.
- Dixon, P.M. (1999) Dental anatomy. Equine Dentistry. 1st (1) 3-28 W.B. Saunders.
- Dixon, P.M. & Copeland, A. N. (1993) The radiological appearance of mandibular cheek teeth in ponies of different ages. *Equine Vet Edu.* 5 (6) 317-323.
- Fawcett, D.W. (1987) A textbook of histology. 603-618 W.B. Saunders Company.
- Ferguson, M. (1990) The dentition throughout life. The dentition and dental care. Vol.3 (1) 1-18 Oxford Heinemann Medical Books.
- Freeman, E. (1998) Periodontium. Oral Histology. 5 (13) 253-288 Mosby
- Johnson, G. & Brannstrom, M. (1974) The sensitivity of dentin. Changes in relation to conditions at exposed tubule apertures. *Acta Odontol. Scand.* 32 (1) 29-38.
- Jones, S.J. (1981) Human tissue: cement. Dental anatomy and embryology. 193-209 Blackwell Scientific Publications.
- Jones, S.J. (1990) The pulp-dentine complex. The dentition and dental care. 193-209 Oxford Heinemann Medical Books.

- Kempson, S.A., Davidson, M., & Dacre, I. T. (2003) The effect of three types of rasps on the occlusal surface of equine cheek teeth: a scanning electron microscopic study. *J. Vet. Dent.* 20 (1) 19-27.
- Kierdorf, H. & Kierdorf, U. (1992) A scanning electron microscopic study on the distribution of peritubular dentine in cheek teeth of Cervidae and Suidae (Mammalia, Artiodactyla). *Anat. Embryol. (Berl)*. 186 (4) 319-326.
- Kilic, S. (1995) A light and electron microscopic study of calcified dental tissues in normal horses. 1-193 University of Edinburgh.
- Kilic, S., Dixon, P. M., & Kempson, S. A. (1997a) A light microscopic and ultrastructural examination of calcified dental tissues of horses: 2. Ultrastructural enamel findings. *Equine Vet. J.* 29 (3) 198-205.
- Kilic, S., Dixon, P. M., & Kempson, S. A. (1997b) A light microscopic and ultrastructural examination of calcified dental tissues of horses: 3. Dentine. *Equine Vet. J.* 29 (3) 206-212.
- Kilic, S., Dixon, P. M., & Kempson, S. A. (1997c) A light microscopic and ultrastructural examination of calcified dental tissues on horses: 4. Cement and the amelocemental junction. *Equine Vet. J.* 29 (3) 213-219.
- Kirkland, K.D., Baker, G. J., Marretta, S., Eurell, J. A., & Losonsky, J. M. (1996) Effects of aging on the endodontic system, reserve crown, and roots of equine mandibular cheek teeth. *Am. J. Vet. Res.* 57 (1) 31-38.
- Lowder, M.Q. & Mueller, P. O. (1998) Dental embryology, anatomy, development, and aging. *Vet. Clin. North Am. Equine Pract.* 14 (2) 227-45, v.
- Mitchell, S. (2004) Peripheral cementum of normal equine cheek teeth: a qualitative and quantitative study. 1-146 University of Edinburgh.
- Muyllé, S., Simoens, P., & Lauwers, H. (2001) The distribution of intratubular dentine in equine incisors: a scanning electron microscopic study. *Equine Vet. J.* 33 (1) 65-69.
- Muyllé, S., Simoens, P., & Lauwers, H. (2002) A study of the ultrastructure and staining characteristics of the 'dental star' of equine incisors. *Equine Vet. J.* 34 (3) 230-234.
- Paphangkorakit, J. & Osborn, J. W. (2000) The effect of normal occlusal forces on fluid movement through human dentine in vitro. *Arch. Oral Biol.* 45 (12) 1033-1041.
- Raab, W.H. (1992) Temperature related changes in pulpal microcirculation. *Proc. Finn. Dent. Soc.* 88 Suppl 1 469-479.
- Schramme, M., Robinson, J., Boswell, J., & Butson, R. (1999) Results of endodontic therapy for periapical infection in nineteen equine cheek teeth. 8 7-11.
- Shafer, W.G., Hine, M. K., & Levy, B. M. (1983) Diseases of the pulp and periapical tissues. A textbook of oral pathology. 4 (8) 479-510 W.B. Saunders Co.
- Ten Cate, A.R. (1998a) Dentinogenesis. *Oral Histology.* 5 (8) 128-149 Mosby, Inc.
- Ten Cate, A.R. (1998b) Hard tissue formation and destruction. *Oral Histology.* 5 (5) 69-77 Mosby
- Torneck, C.D. (1998) Dentin-pulp complex. *Oral Histology.* 5 (9) 150-196 Mosby, Inc.