EQUINE DENTAL ANATOMY AND
PHYSIOLOGY OF MASTICATION

Jack Easley, DVM, MS, Diplomate, DABVP (Equine)
Equine Veterinary Practice, LLC
Shelbyville, KY. 40066 USA

Dental disease and its prevention and treatment comprises approximately 10% of the general equine practitioner's work. The most common disease affecting the equine hypsodont teeth is the development of dental overgrowths. Brachydont dentition (canines and wolf teeth) are less often diagnosed with dental disease. Gingivitis and periodontal disease as seen in many young horses can affect any tooth during the early stages of eruption especially at the time of deciduous tooth shedding. Periodontal disease in the older horse cheek teeth is often associated with crown attrition, diastema, and abnormalities of wear. Diseased canine and incisor teeth in the older horse are often affected by odontoclastic tooth resorption and hypercementosis (EOTRH).

Periapical infections of the cheek teeth can be a significant problem especially in younger and middle-aged horses. This type of dental disease has historically been associated with dental impactions and associated mandibular and maxillary remodeling (so called 3 and 4 year old eruption bumps). Apical infections (termed tooth root infections in older horses) involve the maxillary cheek teeth and have falsely been associated with food accumulation and fermentation deep in the infundibulum. Similarly, dental fracture or splitting of a cheek tooth has been erroneously linked to progression of infundibular caries. An ultrastructural look at the architecture of the equine cheek teeth has further delineated the etiology of infundibular disease and dental fracture.

Recent research sheds new light on dental developmental and traumatic displacements. Such displacements play a major role in diseases of the teeth and can lead to severe biting problems as well as abnormalities of wear. Abnormal dental development, eruption, and variation in position are leading causes of abnormal dental wear patterns and associated disease. Undiagnosed abnormal dental wear sets up a progressive cascade of events. These can eventually lead to diastema (spaces) between teeth that accumulate stagnant feed and can lead to progressive periodontal disease.

Studies using special imaging techniques and thermal measuring devices have shown how floats and power grinders affect the equine tooth. Iatrogenic damage to the tooth surface must be avoided to prevent subsequent dental disease. Several new studies on the thickness of cheek teeth secondary dentine in different aged horses has shown a wide variation with as little as 3 mm overlying some pulp horns. These findings show that horses of all ages should have their cheek teeth reduced with great caution. Bit
seating of the triadan 06 should be minimal to avoid thermal damage or physical exposure of the pulp horns.

The Triadan system for dental nomenclature has been widely adapted for horses and is illustrated in Figure 1. To identify deciduous dentition, the figure 4 is added to the first number (e.g. the right central deciduous incisor [Triadan 501] is replaced by the permanent incisor [Triadan 101] at 2.5 years of age). A pulp identification system (most cheek teeth have 5 pulps, the 06s have 6 pulps and the 11s have 6 or 7 pulps) is shown in Figure 2.

Right

Upper teeth

Left

Lower teeth

Figure 1. The Triadan system of equine dental nomenclature for permanent teeth. To identify deciduous teeth, 4 is added to the first number.
The foal’s dentition, with complex enamel folding on its hypsodont (long crowned teeth) is capable of grinding hard forage immediately after birth, although this will not occur to a significant degree for months. Their cheek teeth differ from those of brachydont species in that the cross sectional areas of the (three) deciduous cheek teeth are almost the same size as permanent cheek teeth, and the 3 cheek teeth fully occupy the maxillae and mandible. The jaws of the foal are not big enough to accommodate all of the deciduous incisors at birth, but the central incisors (01s) usually cut through the overlying gingiva within a week of birth. As the premaxillae and mandible grow, later there is room for the 02s to erupt at 4-6 weeks and for the 03s to erupt at 6-9 months.

The simple (brachydont) canine and first premolar teeth and the more complex (hypsodont) incisors and cheek teeth of the horse contain enamel, dentine and cementum. All teeth are held fast to the alveolar bone by a periodontal ligament attached by Sharpy’s fibers to the peripheral cementum of the tooth. Teeth have one or more pulp chambers that reduce in size with age and contain the vascular and nervous tissues.

An understanding of endodontic anatomy in the mature horse is critical in evaluating dental disease. The middle four cheek teeth (07, 08, 09 and 10) contain five pulp horns arising from a common pulp chamber. The upper cheek teeth have 3 roots and the lower 2 roots. The first cheek tooth in all four rows (06s) has an additional smaller pulp horn, rostral to the others. The lower 6th cheek tooth (311 and 411) has a caudal pulp horn. The 6th maxillary cheek tooth (111 and 211) has an irregular caudal extension (or two) to an adjacent pulp horn.
Enamel is the material that gives the tooth its shape and is the product of ameloblasts. Enamel is hardest and most dense material in the body. Enamel with its high mineral content (96-98%) and absence of cellular inclusions can be regarded as an inert tissue. The ameloblasts die off once the tooth is fully formed. Equine enamel is composed of two main types: Equine Types I and II and small amounts of Equine Type III.

Enamel is quite wear resistant but brittle. The thickness of enamel remains consistent throughout the length of the tooth crown but the folds and rows become closer together as the tooth tapers toward the apex. The incisors and upper cheek teeth contain central columns of enamel referred to as infundibula.

The bulk of the tooth is comprised of dentin which is 70% mineral and 30% organic components. Primary dentin lies between the layers of internal and external enamel. Odontoblasts lining the pulp cavities produce secondary dentin that is responsible for closing the pulp chambers as the tooth matures. With age, this eventually fills with secondary dentin making the tooth stronger and less susceptible to fracture. Tertiary dentin fills in the pulp chambers preventing pulp exposure as the hypsodont tooth crown wears down toward the apex. A balance between crown attrition and dentin deposition is critical to dental heath. The pulp cavity houses the vascular and nervous tissue of the tooth and is lined with odontoblasts. As the tooth matures, the large common pulp chamber becomes narrow until it divides into 5-7 pulp horns and 2-3 separate roots. Root formation in hypsodont teeth continues for many years after tooth eruption. After the roots initially form on these continually erupting teeth, the overall tooth length shortens due to crown attrition. The pulp horns filled with secondary dentin can be identified at the occlusal surface of hypsodont teeth as a dark brown stained area surrounded by cream colored primary dentin.

Cement and the periodontal ligament form a single functional unit anchoring the tooth in the dental socket or alveolus. Prior to eruption the infundibula are lined with cementoblasts that get their blood supply from the coronal aspect of the dental sac. After tooth eruption, the dental sac is disrupted and the blood supply to the infundibular cement and exposed crown cement is lost (rendering it inert or a dead substance). The cement surrounding the reserve crown and root that is in the alveolus is continuously replaced in order for the tooth to erupt throughout life. The periodontal ligament is comprised mainly of collagen fibers produced by both the cementoblasts and the fibroblast housed within the tooth socket.

The incisive bone, maxilla and mandible house the tooth sockets. Only a thin shelf of bone lines between the rostral maxillary sinuses and the caudal roots of the upper fourth premolars and the first molars. A thick plate of bone lies between the caudal maxillary sinuses and the roots of the second and third upper molars.

SUMMARY
Equine dental disease has not changed but the way we understand it, has certainly evolved. New electron microscopic studies of dental structures have provide researchers and veterinary practitioners more thorough and detailed knowledge regarding factors that lead to dental disease. Such advances lead to new methods of prevention and treatment.

REFERENCES AND SUGGESTED READING MATERIALS


Equine Dental Anatomy and Physiology of Mastication IVMA 2011