Equine dental pathology frequently goes undiagnosed. The reasons for this range from a lack of knowledge and appropriate instrumentation to the common dislike many practitioners have for equine dentistry. Dental pathology, however, is very common as shown in multiple cadaver head studies. Pathologic findings include 60% with periodontal disease and 79% with infundibular caries over age 15; 12.9% with developmental or eruption abnormalities; 29.3% with caries and 6.5% with pulp exposure. These numbers do not include common malocclusions which are frequently the initiating factor in more serious pathology.

The identification of pathology starts with a thorough examination. Adequate sedation, a bright light, and full mouth speculum are a must. The minimum required instrumentation includes a dental mirror, periodontal probe, explorer, alligator forceps, and dental picks. Once a problem area is identified it is important to consider the underlying cause, contributing factors, treatment options, and long term care considerations for preventing the problem from worsening or returning.

The most commonly recognized pathologic conditions in the horse are the various malocclusions. Ramps, hooks, waves, steps, excessive transverse ridges, incisor diagonals, ventral curvatures, and dorsal curvatures are easily recognized by the competent practitioner. Each of these conditions should prompt the practitioner to look beyond the obvious malocclusion and consider the potential sequelae to that malocclusion.

A typical scenario involves a 311 ramp and a 210 step with an excessive transverse ridge at its distal aspect. It is easy to address the malocclusions and overlook the high probability of a significant periodontal pocket in the 310/311 interdental space. A complete treatment plan must address the malocclusions, the periodontal disease, post treatment recheck, and long term care.

Developmental pathologic disorders are present at the time of eruption. Supernumerary teeth are thought to develop from the splitting of the dental bud. The extra tooth will result in an unopposed occlusal surface and focal overgrowth. Periodontal disease is highly probable. Radiology will typically reveal both teeth are in the same alveolus. Extraction is normally required. Oligodontia, congenital absence of one or more teeth, and dysplasia are also possible.

Crowding and tooth impaction are a developmental condition most commonly involving the 07’s and 08’s. These teeth must force their way in between existing teeth. The problem this may create is a deviated tooth and probable periodontal disease due to food packing and stasis. A more insidious issue is the potential for anachoretic pulpitis. Hematogenous or lymphatic bacteria may lodge in the inflamed pulp resulting in apical abscessation.
Acquired pathologic conditions occur after eruption. Practitioners frequently encounter fractured teeth, caries, periodontal disease, pulpar exposure, apical abscesses, and iatrogenic trauma. Other issues such as soft tissue trauma, fractures, and neoplasia are outside the scope of this abstract.

Fractured teeth are very common. The fractures range from a small chip to the entire clinical crown being lost. Large fractures typically result in abscessation and extraction. Small fractures may also cause long term problems. A seemingly insignificant chip at the mesiolingual corner of 310 may result in a corresponding 210 focal overgrowth, feed packing, periodontal disease, and an apical abscess of periodontal origin. The practitioner should develop a treatment plan accordingly.

The most common type of fracture is the slab fracture followed by the midline sagittal fracture. Maxillary cheek teeth are more prone to fracture than mandibular cheek teeth. Most slab fractures do not involve the full length of the tooth, while the sagittal fracture does. Pulpal exposure usually occurs in both fractures with apical infection developing in 24% of slab fractures and 100% of sagittal fractures.

Periodontal disease is common and occurs in about 60% of horses over age 15. It is easy to recognize although a dental mirror is required in the caudal oral cavity. The most common cause of periodontal disease is abnormal mastication forces caused by malocclusions. Overlong crowns inhibit the normal range of motion of mastication and abnormal occlusal forces may increase the interproximal space. Feed packs between the teeth and initiates periodontal disease.

Feed stasis and decay changes the normal oral flora from Gram-positive cocci and rods to Gram-negative aerobes, anaerobes, and spirochetes. Gingivitis ensues followed by increasingly deeper attachment loss. Apical abscessation and tooth exfoliation may eventually occur.

Caries is defined as the demineralization of calcified dental tissues and destruction of their organic parts through the acid produced by micro-organisms. Caries are observed in the infindibula, fractured teeth, teeth with pulpar exposure, incisors, and peripheral cementum. Maxillary cheek teeth with cemental hypoplasia are the most common site for caries.

Teeth with infindibular cemental hypoplasia have feed material pack into the infindibula. The feed decays and produces an acidic environment that leads to decay of the cementum. The decay may progress through the cementum and enamel and into the dentin. The decay may extend to the pulp via the dentinal tubules and cause a pulpitis and apical abscess. The most common catastrophic result of infindibular caries is a complete sagittal fracture of the tooth. This occurs because the mesial and distal infindibula decay and continue to enlarge and coalesce forming a large structural defect. Normal occlusal forces then cause the tooth to fracture.
Pulpar exposure was found to be 6.5% in a 1988 study involving 355 cadaver skulls. Even if this number is twice as high as the population average, open pulps should be diagnosed in 3 of every 100 horses. This condition will nearly always be overlooked if a dental mirror is not used. Sometimes there will be obvious decay around an open pulp horn. Other times the problem is not readily apparent. Close observation reveals a light tan colored area of secondary dentin on the occlusal surface. This discoloration is a potential indicator of an open pulp which should be checked with a dental explorer.

Pulpar exposure is due to occlusal surface attrition that exceeds the rate of secondary dentin deposition. The end result of the pulpar exposure depends on the tooth’s ability to withstand a prolonged bacterial assault. Some teeth will put down a layer of tertiary dentin and seal off the pulp. Other teeth develop an apical abscess.

Iatrogenic dental pathology is probably more common than typically realized. Pulpal insults may be caused by heat, pressure, dessication, chemical exposure, and bacterial infection. The result of a significant pulpar insult may not manifest itself for a long period of time and may be difficult to recognize.

Iatrogenic trauma is also possible with the use of concussive and cutting instruments. Slide hammer type instruments that rely on concussion to fracture off overgrowths on the upper 06’s and lower 11’s have significant potential to fracture the tooth through a pulp horn. Molar cutters have this same potential and also may shatter a tooth if an inappropriate jaw configuration is used.

There is a wide array of common dental pathologic conditions in the horse. Failure to recognize these conditions is normally due to an insufficient examination. Diligent dental examinations and appropriate treatments will lead to improved dental care and an improved quality of life for the horse.

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