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First Year of Life

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A Review of Equine Dentistry: The First Year of Life

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Take Home Message

Dental and oral disease can severely affect the young horse. Developmental, traumatic, neoplastic, and infectious conditions are commonly diagnosed in the first year of life. In many foals, the long range consequences of dental abnormalities do not manifest clinically until later in life. A detailed oral examination and ancillary imaging techniques will help the observant veterinarian detect abnormalities and develop an effective treatment plan that can allow most foals to lead a productive life.

Introduction

Equine dentistry is a subject that has been studied and practiced by veterinarians since the beginning of the profession in the late 18th century. Most of what has been taught and published previously, involves dental conditions affecting the horse of riding age. Oral and dental abnormalities are often seen in the foal; however, many conditions are not diagnosed until later in life. Typically, recognition of the condition occurs when clinical signs develop due to the severe, long-term consequences of the abnormality. Normally, the most commonly observed clinical signs of dental abnormalities are changes in the horse, such appearance of the head, eating habits, and/or ability to perform its job.

Dental and facial problems seen in the foal can alter the complex development and growth of the equine head. At birth, many congenital defects are in the early stages of development and become evident only with growth over time. Basic knowledge of normal embryology and development biology helps the veterinarian better understand dentofacial problems seen in the young, growing horse.

Dentofacial deformities are seen in the foal and result in a variable degree of patient morbidity. These defects can be congenital, developmental, or acquired. Such deformities are seldom life threatening unless they compromise the foal's ability to nurse or breathe normally.

This review will cover the development, diagnosis, and general treatment recommendations for the more common dental problems detected during the horse's first year of life.

Embryology and Development

The mandible and maxilla form from the tissues of the first brachial arch. In early embryonic development of most higher vertebrates, the cartilage of the first brachial arch extends as a solid hyaline cartilaginous rod surrounded by a fibrocellular capsule from the developing ear region (otic capsule) to the midline of the fused mandibular processes. The mandible and maxilla

originate from a single center of membranous ossification and each form a neural element. These neural elements are the mandibular branch of the trigeminal nerve innervating the mandible and the infraorbital branch of the maxillary nerve innervating the maxilla. The jaws also develop an alveolar element related to the germinating teeth.

At about 45 days of embryologic development, a continuous band of thickened epithelium forms around the mouth over the presumptive mandible and maxilla. These horseshoe shaped, primary epithelial bands correspond in position to the future dental arches of the maxilla and mandible. The epithelial bands give rise to two subdivisions: the vestibular laminae and the dental lamina. Within the dental laminae, a series of epithelial outgrowths into the ectomesenchyme sets the position of future deciduous teeth. From this point, the tooth progresses through three stages of development: the bud, cap, and bell.[1]

The tooth germs give rise to the deciduous incisors and premolars. These teeth form as a result of further proliferative activity within the dental laminae at its deepest extremities. This increased proliferative activity leads to another tooth bud for the permanent tooth on the lingual aspect of the deciduous tooth germ. The permanent tooth bud remains dormant for some time.

The deciduous canine teeth and the factors that control sexual dimorphism of canine teeth in the horse are not understood. The first premolars, also known as the wolf teeth, are not present in the newborn foal and are presumed to be rudimentary permanent teeth. The upper wolf teeth typically do not erupt until 6-18 months of age in both colts and fillies.

The molars of the permanent dentition have no deciduous precursors so their tooth germs do not originate in the same manner. Instead, when the jaws have grown long enough to handle incorporating more teeth, the dental laminae burrows posteriorly beneath the lining epithelium of the oral mucosa into the ectomesenchyme. This backward extension successively gives off epithelial outgrowths that with associated ectomesenchymal response form the tooth germs of the first, second, and third molars. Aberrations in this pattern of development result in missing teeth or the formation of extra teeth.

The jaw of the foal can only accommodate a few teeth due to its short length relative to an adult horse. The adult equine maxilla and mandible require not only a larger number of teeth but also larger diameter, longer crowned teeth. However, once the enamel organ of a tooth is formed, its size cannot increase. Therefore, two dentitions, deciduous and permanent, are needed. Hypsodont teeth are tall crowned teeth which have a limited growth period but prolong eruption throughout the life of the animal. Therefore, in order to develop in the jaw, considerable movement is required to bring them into the occlusal plane and maintain them in position. This movement has been described in three phases: pre-eruptive, eruptive, and post-eruptive tooth movement.[1] Superimposed on these movements is progression from primary to permanent dentition involving the shedding of deciduous dentition. Premature loss of a deciduous tooth occasionally can lead to early permanent tooth eruption, which is usually not a problem. However, premature tooth loss can also lead to permanent tooth malformation, displacement, damage, or death.

The normal foal is born with the ability to stand and nurse within hours of birth. Suckling takes a coordinated effort of lips, tongue, and oral cavity. At birth, the foal has central incisors and three premolars that are in the early stages of eruption. These 16 teeth continue to erupt and come into wear during the first few weeks of life, filling the oral cavity. With head growth and as the foal begins to masticate forage, the intermediate (02s) incisors erupt at about 6-8 weeks of age. The foal erupts corner incisors (03s) and has a mouth full of 24 deciduous hypsodont teeth by 6-9 months of age. The 18 palatal ridges are close together and the normal transverse ridge pattern of the upper and lower cheek teeth are quite prominent by 6 months of age. At this time, most domesticated foals are weaned from the dam. The dental buds of permanent hypsodont teeth are in place and in various stages of development. An enamel organ forms and the mineralization process of the permanent teeth continues until approximately four years of age.[2]

The steps leading to the development of permanent dentition are complex, requiring a balance among tooth formation, tooth growth, and the maintenance of function. A disruption of the normal pattern of tooth development and eruption is considered to indicate some systemic or local abnormality and can be of some diagnostic significance. A basic understanding of normal embryology and developmental biology helps the veterinarian better understand most of the dentofacial problems seen in a young growing horse.

Congenital Defects

Every equine practitioner should be aware of the large number of congenital craniofacial abnormalities seen in the newborn foal. However, also know that at birth a number of congenital defects are in the early stages of development and may not be detected clinically until later in life. Some craniofacial abnormalities are solely developmental; therefore, the deformity does not begin until after birth. Dental and facial problems seen in the foal can alter the development of the adult equine head.

The more severe congenital deformities have been reported in the literature and given common broad terms, such as wry nose, parrot mouth, monkey mouth, cleft palate, polydontia, etc. These terms poorly describe the complex and continual progression of deformities involving the hard tissues of the head, which are the bone, cartilage, and teeth. These tissues are derived from pluripotent cranial neural crest (CNC) cells. Genetic disorders, environmental insults, or a combination of both, can alter the determination of CNC cells and result in craniofacial malformations.

The development of a foal is complex and a delicately balanced process. As stated, craniofacial malfunctions, which produce congenital or developmental defects, are induced by a number of factors surrounding the horse. Some of these malformations have a genetic basis that is poorly understood. Environmental factors, including teratogens, must be considered. If the effects of a teratogen are exhibited during the first four weeks of gestation, the outcome may be detrimental to the embryo. During this four week period of time, rapid embryo development is occurring. If such a high number of cells are damaged then death of the embryo occurs. However, if only a few cells are damaged during this early period then repair is possible. During the next stage of development (8-12 weeks) when histodifferentiation and organ differentiation are taking place,

teratogenic agents are most likely to produce fetal malformation. The later growth stage (12 weeks until birth) is not as susceptible to teratogenic agents.

Craniofacial deformities can be associated with other congenital defects. The degree and severity of the condition will determine the clinical presentation and prognosis. Such conditions have been studied closely over the past few years. The progression of the deformities over the life of the horse has been recorded and surgical intervention has been used to manage the negative consequences of the conditions.

Defects of the Mandible and Maxilla

Abnormal shortening of the mandible compared to the maxilla with the upper and lower incisors not in full occlusion is referred to as **parrot mouth**. This condition is one of the more common congenital defects seen in the foal.[3,4,5] Parrot mouth is considered an inherited disorder and has shown some familial tendency in thoroughbred horses. Other congenital defects such as autosomal trisomy and limb contractures are rarely seen in combination with parrot mouth. Profound incisor and premolar malocclusion with marked rostral (overjet) and ventral (overbite) projection of the maxillary incisors is classified as a Deep Bite Class II Malocclusion (Fig.1a/b). This condition is aesthetically displeasing to horsemen and can be functionally debilitating (Fig. 2a/b). Evidence obtained from recent studies in rats and reports in human medicine, demonstrates a relationship between dental malocclusion, dysfunction of the masticatory system, and the alignment of the spinal column. With early detection, this condition can be corrected using a combination of acrylic and metal bite plates utilized as a functional incline plane.[4] Tension band wiring techniques with or without a bite plate have also been successfully used to correct this malocclusion in the young rapidly growing foal under six months of age.[5] Osteodistraction of the mandible has been used to correct the condition in older foals and a large size camelid.[6] Foals diagnosed with dental malocclusion will benefit from regular oral examinations to detect dental overgrowths. Corrective dental reductions are indicated before elongations cause functional disturbances with jaw growth and/or mastication.

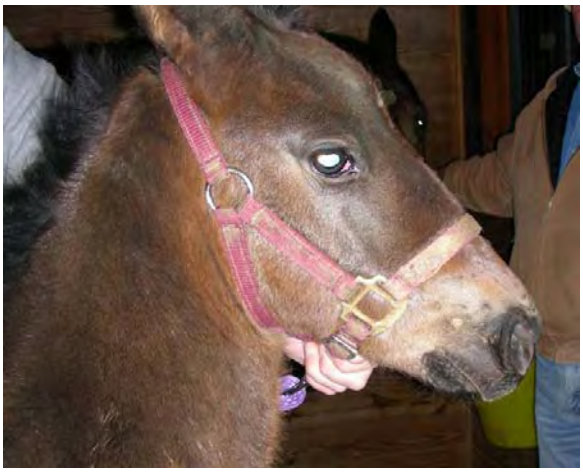


Fig. 1a. Lateral view of a 1 month old foal with a Class II Malocclusion. Notice the over development of the upper lip in contrast to the lower. This parrot mouth appearance is usually evident by 30-90 days of age.

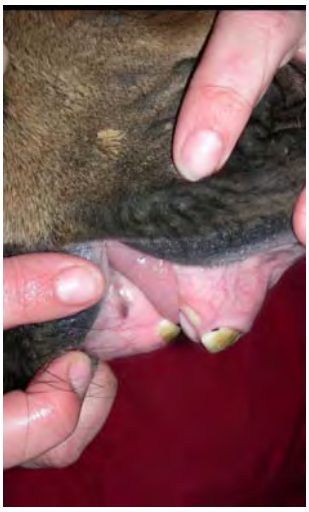


Fig. 1b. Open mouth view of the same 1 month old foal showing the overjet of the upper incisors in relation to the lower incisors.

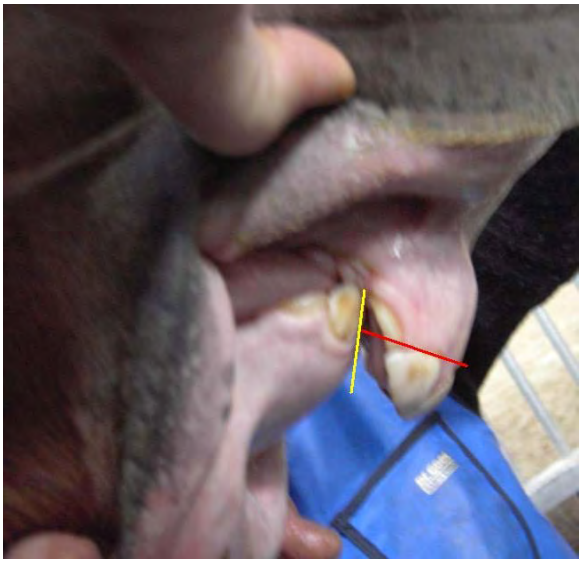


Fig. 2a. Open mouth view of an 8 month old parrot mouth foal with a deep bite Class II Malocclusion. This foal has a severe overjet (red line) and overbite (yellow line) of the incisor teeth.

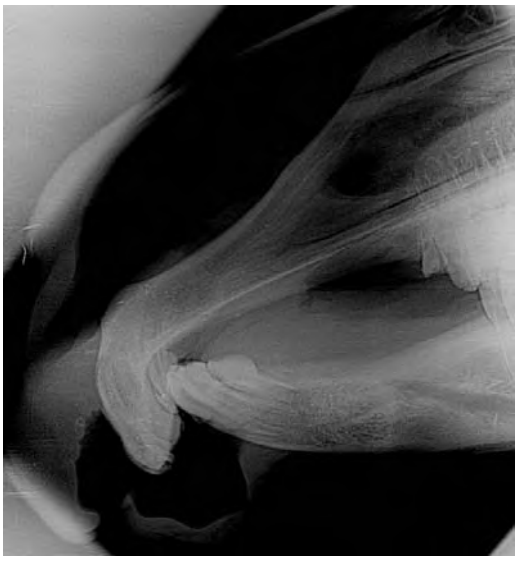


Fig. 2b. Lateral skull radiographs of the same 8-month-old foal. A downward curvature of the premaxilla and trapping of the lower incisors behind the upper incisors is obvious. Deciduous 2nd premolar (06) malocclusions and dental elongations (hooks) can be seen.

A shortened maxilla in relation to the mandible is termed **monkey mouth**, undershot jaw, or Class III Malocclusion. This condition is seen more commonly in miniature horses and pony breeds. It has also been associated with achondroplastic dwarfism in cattle. This congenital anomaly has been reported in combination with other deformities of the head and musculoskeletal system. Traumatic events during the first year of life can cause the monkey mouth to develop in foals that were normal at birth. (Fig. 3a/b/c) Principles of therapy in the young, growing horse should consist of: 1) encouraging or accelerating rostral growth of the maxilla, 2) supporting the nasal bone and nasal septum, 3) slowing rostral growth of the mandible, 4) preventing abnormal interference of the upper and lower arcades during mastication, and 5) preventing abnormal wear patterns, which lead to overgrowths of the teeth. Many of the same orthodontic and surgical principles used to correct parrot mouth have been used to correct monkey mouth.

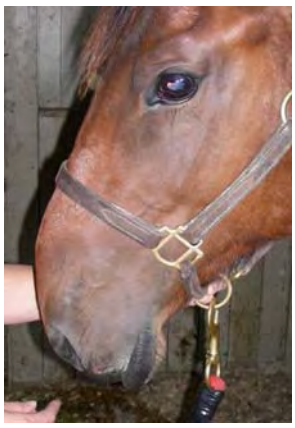


Fig. 3a. A 5-month old foal 60 days after a compression injury to the face from running into a wall. The foal has compromised airflow of one nostril due to a palpably deviated nasal septum.

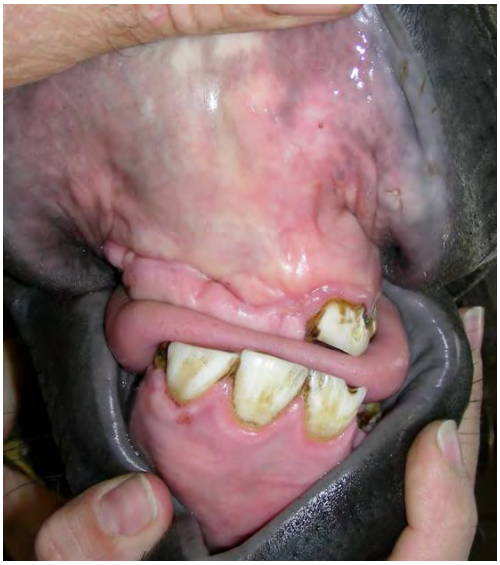


Fig. 3b. Open mouth view of the same foal showing undershot lower jaw and missing upper right deciduous incisors.

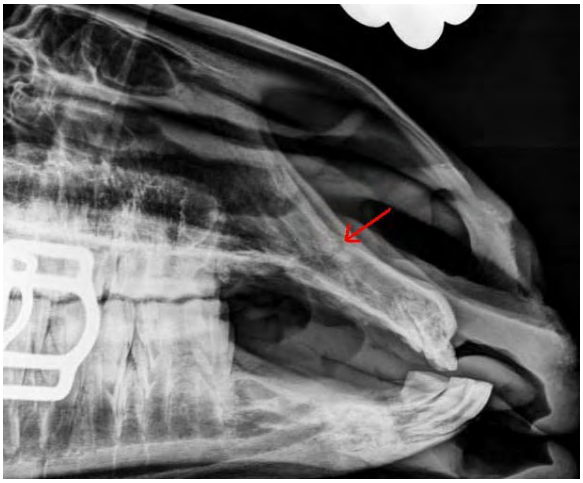


Fig. 3c. Lateral radiograph showing a Class III Malocclusion with the lower incisors rostral to the damaged upper incisor arcade. A bony callus can be seen in the healing fractured nasal and palatine processes of both premaxillae (red arrow). The right upper deciduous incisors are missing.

Wry nose or campylorrhinus lateralis is the congenital shortening and deviation of the maxillae, premaxilla, nasal bones, vomer bone, and nasal septum. (Fig. 4a/b) The cause of this condition is unknown. A genetic component has not been reported, but the condition may be seen in combination with other congenital defects, such as cleft palate and limb contractions. The deviation may be mild or severe and may be accompanied by abnormal arching of the nasal bones and the hard palate. Severely affected foals have difficulty nursing and exhibit respiratory stridor even at rest. Traumatic events early in life have also been known to cause this condition to develop.

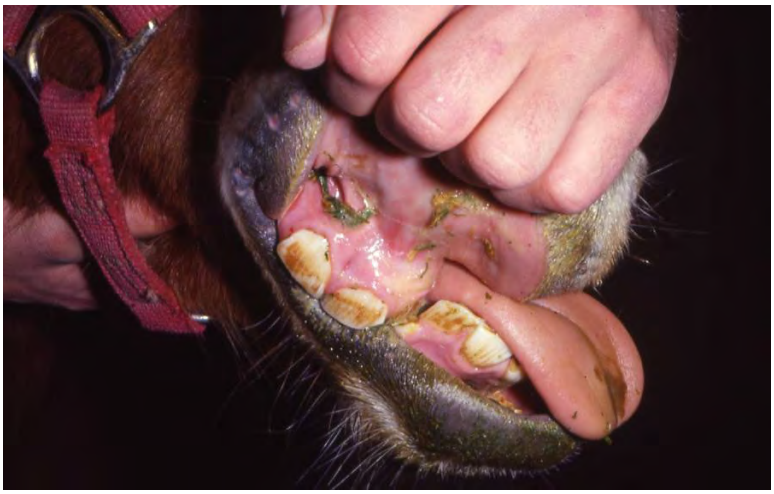


Fig. 4a. Open mouth view of a 4 month old foal born with a wry nose. The upper incisor arcade is displaced at a 75 degree angle to the lower arcade. The cheek teeth are in good occlusion but showing a shear wear pattern on the concave arcade.

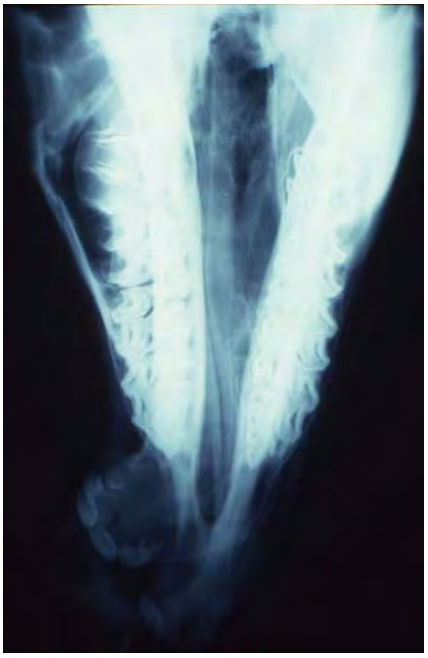


Fig. 4b. Dorsoventral skull radiograph of the 4 month old foal with a wry nose. The deviated nasal septum and convex side nasal occlusion can be appreciated.

Mildly affected foals with wry nose need no immediate treatment to survive, but severely affected foals may require intensive and extensive nursing care and possibly a tracheotomy. Slight deviations have been seen to spontaneously straighten as the foal grows. Moderate to severe deviations usually become more debilitating over time. These cases are best managed by orthodontic and/or surgical treatment to resolve respiratory obstruction or improve mastication and dental occlusion.

Surgical success in the treatment of wry nose has been achieved using a two-stage procedure performed at three month intervals.[7] The first stage involves straightening the pre-maxilla and alining the incisor teeth. The second stage involves removal of the deviated nasal septum. Distraction osteogenesis has also been used to correct this condition in a yearling.[8] A recent modified surgical correction technique requiring only one general anesthesia achieved good cosmetic and functional results allowing a successful athletic career.[9]

Dental Growths and Tumors

Ossifying Fibroma

Fibroosseous lesions of the young horse's oral cavity have been reported in the literature. Equine juvenile mandibular ossifying fibroma has been described as a separate entity based on characteristic radiographic and histological findings. (Fig. 5a/b) While the rostral portion of the mandible is the site of predilection, these tumors can occur at other locations in the mandible as well as the maxilla and premaxilla.[10] Many treatment options have been proposed because local recurrence is common after surgical debulking. More long term case studies need to be conducted to determine which tumors are candidates for simply surgical excision and which tumors require more aggressive surgical treatment, such as mandibulectomy or megavoltage

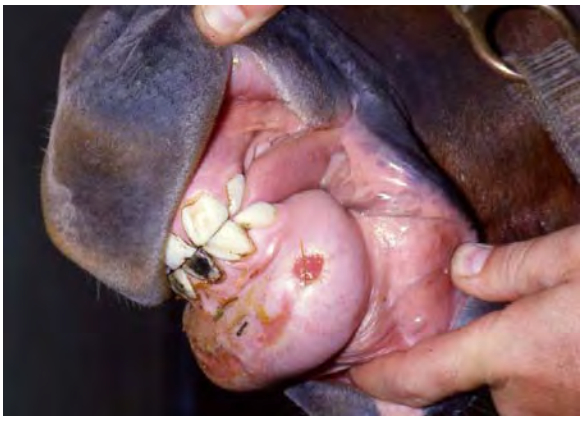


Fig. 5a. Lower jaw of a 9 month old foal with an equine juvenile mandibular ossifying fibroma. The labial mucosa is cracked from being stretched due to this rapidly growing mass.

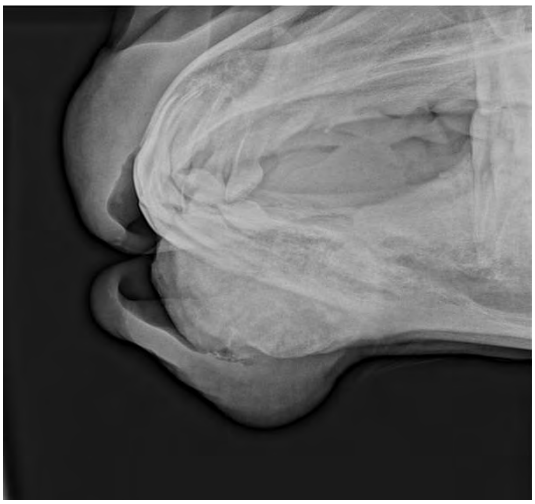


Fig. 5b. A lateral radiograph of a 7 month old foal with the characteristic appearance of an ossifying fibroma.

radiation. The long term effect of treatment on permanent tooth eruption and jaw growth has not been studied.

Heterotopic Polydontia (temporal teratoma or adenoma, dentigerous cysts, periauricular cysts, conchal fistula, or ear tooth)

Aural fistula or draining tract from the rostral pinna is most often associated with an ectopic tooth in the area of the temporal bone.[11] This congenital dentigerous cyst is recognized as a swelling at the base of the ear accompanied by a draining tract lined with stratified squamous epithelium and containing dental tissue. (Fig. 6a/b/c) The cyst is believed to originate from failure of closure of the first brachial cleft. The lesions may be present at birth but may not be recognized clinically until the horse is mature. Radiology or computed tomography can be helpful in confirming the diagnosis. Careful surgical removal of the entire cyst and dental tissue is curative.[12]

Fig. 6a. An 8 month old American Saddlebred colt with mucoid drainage from the rostral pinna of the ear. This is characteristic of a conchal fistula. The round, soft dentigerous cyst can be seen just below the base of the ear.

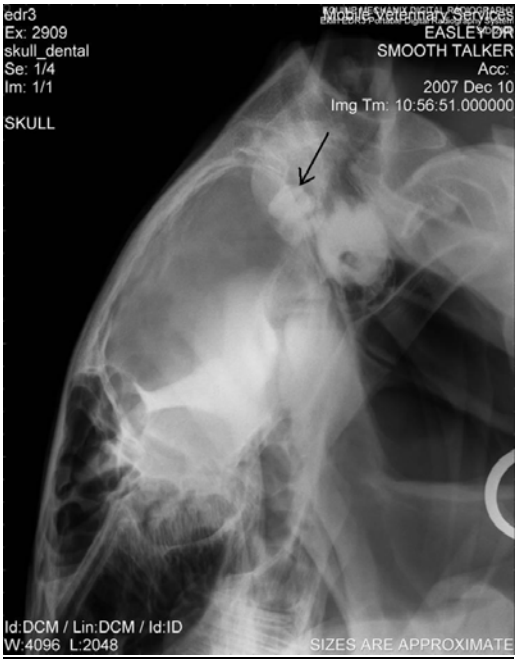


Fig. 6b. A lateral skull radiograph showing a density consistent with calcified dental tissue just over the temporal bone rostral to the ear (black arrow). This is characteristic of a dentigerous cyst.

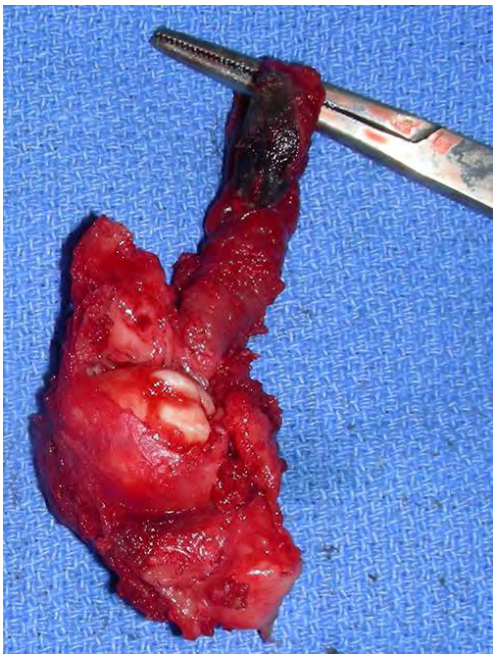


Fig. 6c. Fistulous tract and dental material surgically removed from this foal's temporal region.

Bone and Sinus Cysts

Cysts of the bone and soft tissue can affect foals of any age. The more common soft tissue cysts are conchal fistulas with no dental tissue, subepiglottic cysts, thyroglossal cysts, and paranasal sinus cysts. An epidermal inclusion cyst located in the dorsolateral aspect of the nasal diverticulum, also known as an atheroma, has been seen in the young horse. Surgical management consists of removing the cyst completely and addressing the care of hard or soft tissues.

Paranasal sinus cysts are most often seen in horses under two years of age but have been in horses ranging in age from nursing foals to young adults. (Fig. 7) The most common clinical features are dyspnea, facial swelling, and nasal discharge. As the cyst expands, pressure causes distortion of the ventral nasal conchae, internal sinus structures, maxilla, nasal septum, and developing dental buds of permanent teeth. The mandible may also be involved in some cases. The cysts are typically filled with yellow, viscous, acellular fluid. Radiographs or head imaging with CT or MRI will usually outline the extent of the cystic lesion. Management consists of surgical excision of the cyst through a sinus flap, establishment of ventral sinus drainage, and removal of any damaged or displaced teeth. The prognosis is guarded to favorable depending on the extent of distortion of adjacent structures. Regression of the nasal obstruction and facial deformity is more likely following surgery in the young rapidly growing horse.

Cystic lesions of the jaws present as bony swellings. (Fig. 8a/b) Mandibular aneurysmal bone cysts are rare. Odontogenic cysts are noninflammatory cysts lined with epithelium and may or

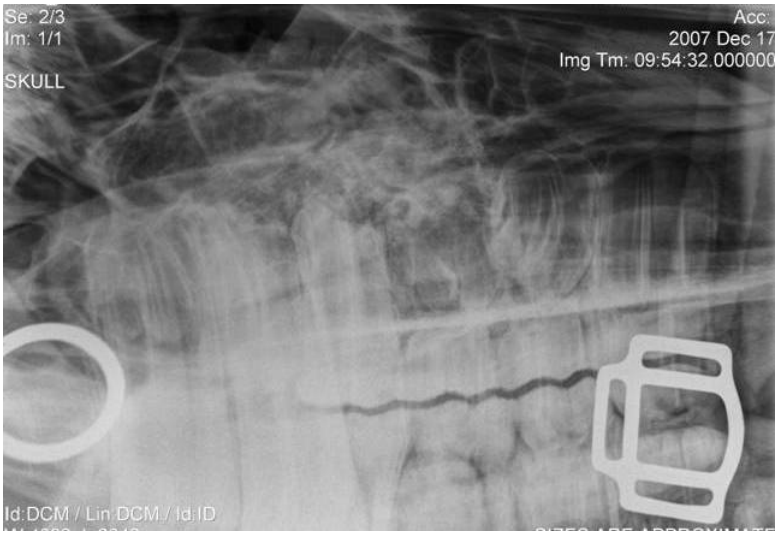


Fig. 7. A lateral oblique skull film of an 11 month old foal with a firm enlargement over the maxillary sinus. The frontomaxillary sinuses are filled with a lobulated fluid material. The permanent first molar apex appears blunted. The dental buds of premolars 3 and 4 are displaced from the expansion of the sinus cyst.

may not be associated with tooth roots. Fibrous dysplasia of the jaw has been reported in man and similar lesions have been seen in the foal. This lesion is not a true neoplasm but a slowly expansive, space-occupying mass in the lower jaw.[13] Suspicious jaw enlargements should be subjected to a full range of diagnostic tests including radiology, gamma scintigraphy, biopsy, and where feasible, CT and MRI.

Oral and dental tumors are uncommon but several have been reported in the neonate or young growing equine. Complex/compound odontomas are irregular tumor-like masses of dental tissue and radiographic appearance is characteristic. Multiple small lobulated masses within a well-defined cyst-like structure at the root of a maxillary tooth are typical. Complete surgical excision early in the course of the disease should resolve the problem. Repeat surgeries may be necessary and care should be taken to minimize damage to associated dental and paranasal sinus structures. There is at least one report of an osteosarcoma involving the mandible of a 6-month-old colt.[13]

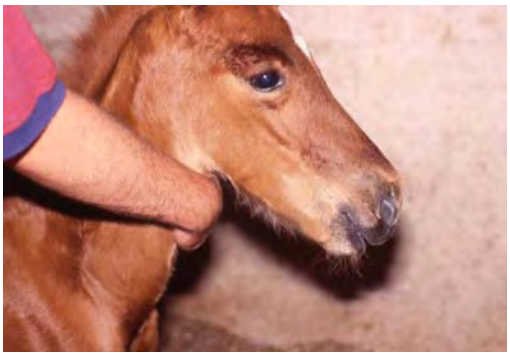


Fig. 8a. A 1 month old foal with a firm enlargement in the interdental region of the right hemi-mandible.

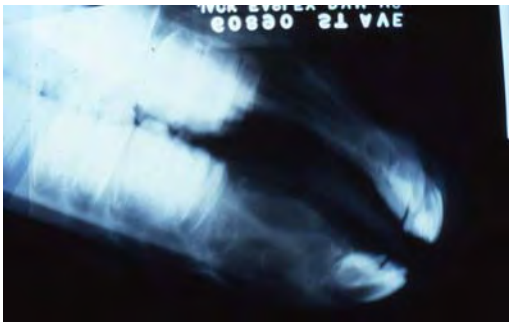


Fig. 8b. A lateral skull radiograph showing a cystic bone lesion in the interdental space of the mandible. The cyst was surgically opened and contained mucoid material. The cavity was curetted, and the defect healed without complications.

Fig 9a. A 14 day old foal that showed signs of neonatal maladjustment syndrome. He recovered but was left with the tongue protruding from the left side of the mouth due to right-sided hypoglossal paresis.

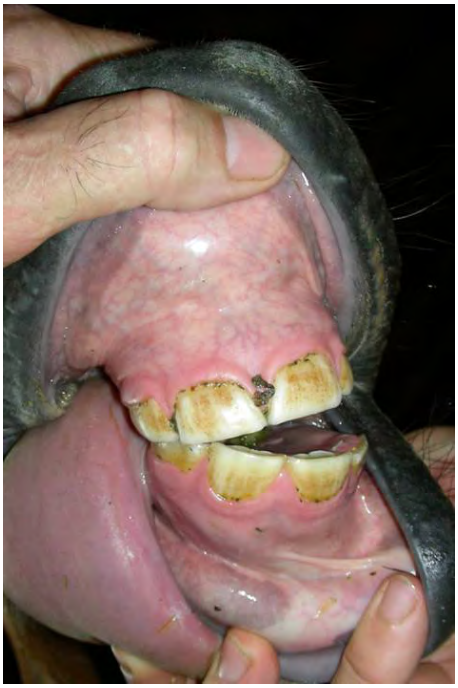
Oral, Facial, and Dental Injuries

Non-traumatic soft tissue lesions can be seen at birth or develop over the first year of life. Oral fungal plaques commonly called thrush caused by Candida albicans have been reported in immunocompromised foals. Lip clefts, defects, and warty growths have been seen in the newborn.

Tongue paralysis and hemiparesis have been seen in the foal secondary to trauma or cerebral hypoxia. (Fig. 9a/b) Traumatic lacerations to the lips and cheeks are not uncommon in the precocious foal. A wound may involve any combination of soft tissues, bone, and teeth. These wounds require careful evaluation and may benefit from surgical repair with accurate apposition of each tissue layer. Orocutaneous or orosinus fistulas can occur if careful and complete tissue closure is not achieved.[7]

Trauma to the head can have obvious effects on soft tissue and bone but underlying dental tissues are often damaged. Fracture or avulsion of deciduous teeth can lead to acute oral malodor following initial insult. If these teeth are lost or removed, therefore no longer occupying a space in the mouth, then interproximal dental drift and abnormal elongation of opposing teeth will occur. Permanent tooth buds can be damaged, displaced, or crowded, impeding normal eruption. Long-term ramifications of these problems should be anticipated but may not show up clinically until later in life.

The inquisitive nature of the foal and the use of its lips and nose to gather environmental information predispose the foal to trauma of the maxilla and mandible. Distraction injuries from catching the jaw on a solid object and pulling back or compression fractures from a kick or fall are common occurrences. Dentoalveolar injuries with avulsion of teeth and/or bone fragments are often seen with jaw fractures. Denuded or displaced jaw fragments can lead to bone necrosis and sequestra formation.



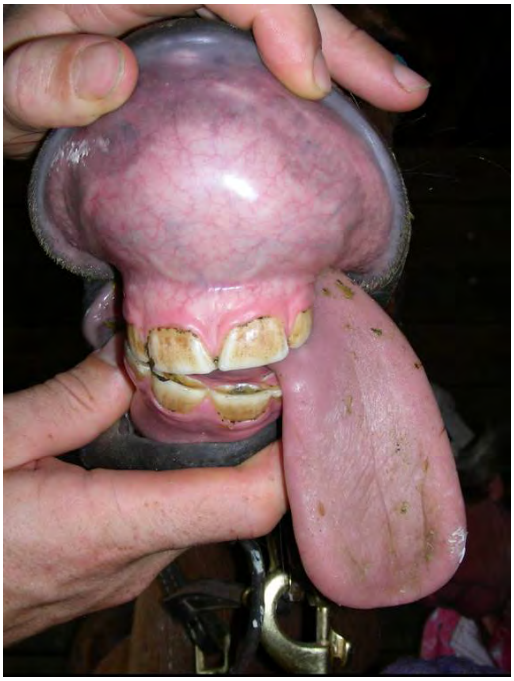


Fig. 9b/c. This foal regained full use of the tongue by 9 months of age. The pressure from constant weight of the tongue hanging from the left side of the mouth caused a ventral deviation to the rostral portion of the left side of the mandible. This lead to a permanent diagonal incisor bite.

Multiple options are available for repair ranging from conservative management, wire fixation, intra-oral splints, and internal or external fixation. The complication rate for these fractures is high but the long term prognosis for life and function is generally favorable. The decreased holding power of foal bone compared to that of the adult makes the use of bone screws and threaded pins less effective for fracture fixation. Threaded implants can also complicate jaw growth and tooth eruption if left in place for extended periods of time.

On a lesser degree of reporting, dentoalveolar defects have been documented in the young growing foal. Oligodontia meaning missing teeth and polydontia meaning additional teeth are rarely seen in the foal. Mixed epithelial defects of tooth and skin as well as displaced and dysplastic teeth are described in the literature as they relate to foals in the first year of life.

Foal Examination and Routine Dental Care

Examination of the newborn foal should consist of evaluation of the head for symmetry. The lips, tongue, cheeks, and palate should be assessed. The occlusion of the upper and lower teeth should be examined. Radiographs or other imaging modalities may be required to develop a more complete understanding of some craniofacial defects. The oral cavity can usually be superficially assessed without the use of sedation or a mouth speculum. If a detailed oral examination is required, such as in the case of suspected cleft palate or post trauma, mild sedation with xylazine 0.5 mg/lb and a Haussman or McPherson mouth speculum works well.

Smaller sized speculums are available commercially for use on ponies and miniature horses, but these speculums are not necessary as the larger models can be used on foals.

When examining any horse, a permanent record of all dental findings is crucial. Most veterinarians find it useful when using computerized records to use The Triadan Dental Numbering System. The Triadan System for numbering deciduous teeth utilizes the prefix of 5, 6, 7, and 8 to identify the four quadrants of the mouth beginning on the upper right arcade and continuing in a clockwise fashion around the mouth. Therefore, the foal's right upper arcade is designated as 501, 502, 503, 506, 507, and 508.

When performing an oral exam on a foal, the most important things to look for are normal tooth alignment, eruption sequence, and tooth development. Any deviation from normal should alert the veterinarian of possible underlying systemic or local abnormalities and the need for further diagnostic workup. Diagnostic tools may include radiographs, nuclear scintigraphy, sinocentesis, biopsy, or blood work.

Smaller floats and instruments for use on the foal are available and marketed for ponies or miniature horses. Power instruments are seldom needed with young horses. However, if severe elongations in the back of the mouth need to the reduced then the flexible shaft grinders with diamond burrs make this job easier.

Floating foals' teeth is usually limited to correction of dental elongations resulting from incorrect jaw alignment and often leading to abnormal mastication. Such cases might be a shear mouth seen in a wry nose foal or hooks seen on the upper 06s and/or lower 08s in a foal with a parrot mouth. Abnormally positioned tall transverse ridges or ramps seen in the parrot mouth or monkey mouth foal can actually lock the maxilla and mandible in an abnormal position and prevent rostral growth of the shorter jaw. If a foal has traumatically broken teeth or genetically displaced teeth in one jaw, consequently some of these foals may have tall teeth that need reduction. As foals get older and reach the age of nine to twelve months, a space forms behind the 08s prior to eruption of the 09s. As a result, they often accumulate feed in the caudal recesses of the mouth. Flushing may be necessary to prevent feed stagnation and quidding.

Conclusion

Evolutionary and developmental biologists, geneticists, and tissue engineers provide information on the molecular mechanisms crucial for the patterning and formation of craniofacial structures and morpho-regulation of tooth number, size, shapes, and differentiation. This work will change the way we understand, treat, and possibly prevent congenital and developmental malformations. It may also establish a foundation for craniofacial tissue manipulation and regeneration.[4]

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