Proceedings of the American Association of Equine Practitioners - Focus Meeting

Focus on Upper and Lower Respiratory Diseases

Salt Lake City, UT, USA – 2010

Next Focus Meetings:

July 24-26, 2011 - Focus on Colic
Indianapolis, IN, USA

September 18-20, 2011 – Focus on Dentistry
Location TBD

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Review of Diseases and Surgery of the Nasal Cavities

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Take Home Message

Distinguishing between the many diseases of the nasal cavities of horses is often difficult but is necessary so that a horse with disease of a nasal cavity can be treated appropriately. Diagnosing disease of the nasal cavities generally requires careful clinical, rhinoscopic, and radiographic examinations. Because of the complexity and inaccessibility of the nasal cavities, treating horses for disease of the nasal cavities is often difficult.

High Blowing (False Nostril Flutter)

High blowing is characterized by a loud, vibratory noise produced during expiration by turbulence caused by vibration of the true and false nostrils. High blowing noises are usually produced at the start of exercise or when the horse is excited, usually disappear when the horse is exercised strenuously, and are not associated with respiratory impairment.

Nasal Paralysis

During strenuous exercise, the nostrils dilate as the musculature of the nostrils contracts. Failure of a nostril to dilate can be the result of trauma to the dorsal buccal branch of the facial nerve, such as might occur during extraction of a cheek tooth through a buccotomy. A horse with unilateral or bilateral collapse of the nares exhibits exercise intolerance and make an abnormal respiratory noise that can be localized to the nostrils. A decreased flow of air from the affected naris may be detected.

Mild, bilateral nasal collapse caused by bilateral facial paralysis may be difficult to recognize, but paralysis of one nostril is obvious, because the muzzle is deviated and sometimes the ear and upper eyelid of the affected side are dropped. The condition is confirmed if the horse’s tolerance to exercise improves and abnormal respiratory noise disappears when the nostrils are dilated by abducting the alar cartilages with sutures.\(^1\)

Nasal collapse resulting from neuropraxia of the facial nerve or its branches caused by a trauma to the side of the head usually resolves within days to months. If nasal collapse is permanent, airflow through the affected nostril can be improved by removing the alar fold.\(^2\) The horse’s respiratory capacity can also be improved by excising the lateral alae or creating a permanent tracheostomy, but these two procedures are disfiguring.

Vasomotor Rhinitis

Vasomotor rhinitis is a non-inflammatory, non-allergic, disorder of the nasal cavities characterized by a bilateral, watery, nasal discharge, sneezing, obstructed nasal airflow, loss of
sense of smell, and nasal pruritus. The affected horse may also develop headshaking. Vasomotor rhinitis occurs commonly in human beings, but reports of affected horses are rare. The disorder may be caused by hyper-reaction of the nasal mucosa to stimuli resulting from imbalance in the autonomic control of function of the nasal mucosa. Clinical signs displayed by people affected with vasomotor rhinitis may be induced by changes in temperature or humidity, certain odors, and physical or emotional stress. Vasomotor rhinitis of one affected horse was induced by exercise.

Clinical signs of vasomotor rhinitis can be ameliorated by nebulizing the affected horse before exercise with an adrenergic agonist, such as xylometazoline (5 mg), which may reduce nasal congestion and discharge by constricting the nasal vasculature, or with sodium cromoglycate (80 mg), which stabilizes mast cells, thus preventing excessive release of histamine.

**Apical Infection of a Maxillary Premolar Causing a Nasal Granuloma**

Periapical infection of the second, third, and fourth maxillary premolars (Triadan 106-108 and 206-208) usually results in facial swelling and sometimes in a discharging tract on the side of the face, but it may also result in a tract that discharges into the nasal cavity and a granuloma at the site of the tract’s exit into the nasal cavity.

Clinical signs of a nasal granuloma caused by periapical infection of a maxillary premolar may include halitosis, purulent nasal discharge, and diminished airflow from the affected nasal cavity. Exudate, feed material, or a granuloma may be seen during rhinoscopic examination of the rostrolateral aspect of the nasal cavity. An abnormality of the clinical crown of one of the ipsilateral maxillary premolars, such as occlusal exposure of the pulp, infundibular caries, or fracture may sometimes be seen during oral examination. Radiographic examination of the maxillary dental arcade confirms the diagnosis of periapical infection.

Affected horses are treated by extracting the infected tooth and sealing the oral aspect of its alveolus with an acrylic. The draining tract and granuloma resolve soon after the source of infection has been removed.

**Oronasal Fistula**

An oronasal fistula is an epithelial-lined communication between the oral and nasal cavities that allows contents of the oral cavity to enter the nasal cavity. The fistula is usually the result of failure of an alveolus to heal after a tooth has been removed; failure of the alveolus to heal is usually caused by failure to remove dental or osseous fragments from the alveolus at the time the tooth is removed.

The affected horse has a malodorous, purulent, feed-contaminated discharge from the affected nasal cavity. A probe inserted into the oral aspect of the fistula can be observed or palpated in the nasal cavity. Rhinoscopy may be helpful in identifying the nasal aspect of the fistula, and radiographic examination of the maxillary dental arcade may reveal dental or osseous fragments in the unhealed alveolus.
Treatment is directed at promoting healing of the alveolus by removing dental and osseous fragments and epithelium from the tract. Plugging the oral aspect of the tract with acrylic prevents feed from entering the alveolus and allows the apical aspect of the alveolus to heal. If the fistula fails to heal using these measures, it can be obliterated by plugging it with transposed muscle.8

**Epidermal Inclusion Cysts of the Nasal Diverticulum**

An epidermal inclusion cyst (Fig. 1), or epidermoid cyst, is a congenital, spherical cyst resulting from aberrant location of epithelial tissue. Epidermal inclusion cysts can be found in various sites on the horse, such as the brain9 or cranial pectoral region, but they are most commonly located rostral to the nasoincisive notch between the skin and the mucous membrane in the dorsocaudal aspect of the false nostril.10,11 They are sometimes erroneously identified as a sebaceous cyst, because they contain a thick, grey material resembling sebum.

![Figure 1. Epidermal inclusion cyst in the right false nostril.](image)

Although present at birth, an epidermal inclusion cyst may only become apparent when continuous exfoliation of squamous cells from its epithelial lining causes it to expand. They range from 2 to 5 cm in diameter and bulge into the lumen of the false nostril.12 They are soft and fluctuant, and palpation of them does not cause the horse to show signs of pain. Diagnosis is based on the lesion’s pathognomonic location, its gross appearance, and its odorless, greasy, grey contents obtained by aspiration.10,12

Obstruction of respiration has not been reported; epidermal inclusion cysts are of cosmetic significance only.13 The cyst can be removed with the horse sedated through a cutaneous incision created over the cyst, using local or regional anesthesia. Inadvertent perforation of the thin-walled cyst hampers its excision. The cyst can also be removed using a roaring burr after making a stab incision into the rostroventral aspect of the cyst.14 After expressing the cyst’s contents, a laryngeal burr is inserted into the cyst and rotated so that it engages the cyst’s epithelial lining, which is then everted and excised. The incision is left unsutured.

The cyst can also be removed by chemically destroying it lining by instilling 2 to 4 mL of 10% formalin into the lumen of the cyst after aspirating its contents.15 The cyst gradually regresses.

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8

Proceedings of the AAEP Focus on Upper and Lower Respiratory Diseases - Salt Lake City, UT, USA, 2010
Complications are mild and temporary and include signs of nasal irritation, swelling of the cyst, and mucoid nasal discharge.

**Alar Fold Stenosis (Nasal Flutter)**

The alar fold is a mucocutaneous fold, found in the dorsorostral aspect of the nasal cavity, that extends caudally from the alar cartilage to the rostral aspect of the ventral nasal concha. It divides the external naris into a small, upper compartment, the nasal diverticulum, or false nostril, and a large, lower opening, the true nostril. The alar cartilage, which is attached to the alar fold, is elevated when the *transversus nasi* muscle contracts during deep inspiration. This tightens the alar fold, closing the opening to the false nostril. Improper action of the transversus nasi muscles or excessive size of the alar folds permits entry of air into the false nostrils, causing obstruction of the nasal cavities, a condition referred to as alar fold stenosis.

Alar fold stenosis causes a loud, objectionable respiratory noise during exercise, or sometimes even at rest, in some American Saddlebred horses and Standardbreds. Alar fold stenosis may also occur in young male horses of any breed, but especially miniature horses, if unerupted maxillary canine teeth markedly narrow the nasal vestibula.

Horses with alar fold stenosis make a rattling or vibrating noise that emanates from the nostrils during inspiration and expiration, but especially during expiration. Most horses must be exercised intensely to elicit the noise. In addition to making an abnormal respiratory noise, some horses with alar fold stenosis suffer from exercise intolerance.

The diagnosis of alar fold stenosis can be established with certainty if the abnormal respiratory noise is eliminated when the horse exercises after the alar folds have been elevated with sutures to close the false nostrils. To elevate the folds, a mattress suture (Fig. 2) is placed through each alar fold just caudal to the alar cartilages; the ends of the sutures are tied together, under tension, over a gauze roll placed over the nasal bones.

![Figure 2. A mattress suture placed through the right alar fold. A similar suture is placed through the left alar fold, and the sutures are tied together over the nasal bones.](image-url)
To alleviate the condition, the alar folds are resected with the horse anesthetized and in dorsal or lateral recumbency. The folds can be excised from the alar cartilage to the ventral concha through the external nares, but to better expose the folds, the lateral alae of the nostrils can be incised and the incisions closed after the folds are removed. Dorsal recumbency provides adequate access to both alar folds, if the folds are resected through the external nares. Hemorrhage created is substantial but ceases when the mucosal edge of the incision is joined to the corresponding cutaneous edge of the incision with a continuous suture pattern. The horse can be returned to exercise in two to three weeks.

Resection of the alar folds generally resolves the abnormal respiratory noise and restores exercise tolerance, but if the diameter of the rostral portion of the nasal cavities is inadequate, excision of the folds may not totally relieve the signs.

**Wry Nose (Campylorrhinus lateralis)**

Wry nose (Fig. 3), or campylorrhinus lateralis, is a congenital deviation of the maxillae, premaxillae, nasal bones, vomer bone, and nasal septum. The cause of the deviation is not known, but fetal malpositioning, the result of the inability of the dam to distend its uterus to accommodate the developing foal, has been theorized to be responsible for the condition. The heritability of the wry nose has not been established, and teratogenic causes have not been identified. Wry nose occurs occasionally in all breeds, and male and female foals are affected equally.

The deviation may be mild to severe and may be accompanied by arching of the nasal bones and the hard palate. The affected foal may also suffer from palatoschisis (i.e., cleft palate). The tongue and all or a portion of the mandibular incisors may be visible. The affected foal may have
Deviation results in respiratory impairment, sometimes even when the foal is resting. The nasal cavity on the convex side of the deviation is the most severely restricted.

Most affected foals need no immediate treatment, but administering nutrition through a nasogastric tube may be necessary if the foal is unable to nurse. A foal unable to nurse usually is able to drink a milk replacer from a bucket. A mild deformity may resolve spontaneously as the foal grows. Surgical correction involves straightening the maxillae/premaxillae and nasal bones and removing most of the nasal septum. The premaxillae/maxillae and nasal bones are transected at their point of maximum curvature, and an autogenous rib graft is inserted into the gap created on the concave side of the deformity in the premaxillae/maxillae when the incisors are realigned. The severed premaxillae/maxillae are stabilized with Steinmann pins inserted into their medullary cavity, and the severed nasal bones are stabilized with plates and screws or small Kirschner wires. The deviated nasal septum is removed.

Distraction osteogenesis has also been used to correct deviation of the premaxillae/maxillae. Using this technique, the premaxillae/maxillae are partially transected at their point of maximum curvature, and Steinmann pins are inserted perpendicularly across the premaxillae/maxillae, rostral and caudal to the osteotomies. A double connecting bar is attached on the convex side of the head, and a monolateral distraction external skeletal fixator is attached on the concave side. The rostral and caudal pins on the concave side of the face are distracted periodically. The clamps that attach the Steinmann pins with the double connecting bar on the convex side of the face are also slackened and retightened periodically to accommodate the pressure generated by bone distraction on the concave side. The cosmetic result of distraction osteogenesis is reported to be superior to that of conventional surgical correction.

**Choanal Atresia**

Choanal atresia is a malformation in which one or both nasal cavities fail to communicate with the nasopharynx because of persistence of the buccopharyngeal septum, the membrane separating the nasal and nasopharyngeal cavities during embryonic development. The obstructing septum may be complete or incomplete, and bony or membranous. The obstructing septum of horses seems usually to be membranous, based on the few reports of the condition in this species. The heritability of the choanal atresia in horses has not been established, and teratogenic causes have not been identified.

Choanal atresia results in partial or total inability to breathe through the affected nasal cavity. Because the horse is an obligate nasal breather, a foal with complete, bilateral choanal atresia has severe inspiratory difficulty and soon dies unless it immediately receives a tracheostomy. If the birth is attended, the guttural pouches may be seen to balloon. Complete, bilateral choanal atresia may not be recognized unless the birth is attended. A horse with unilateral choanal atresia or bilateral but incomplete or choanal atresia may show no clinical signs of the condition until it is put into training. When exercised, it may be exercise intolerant or make an abnormal respiratory noise. A unilaterally affected horse may fail to flare the nostril on the affected side during exercise.

Diagnosis is based on clinical signs, the inability to advance a nasogastric tube through the nasal cavity, and endoscopic or contrast radiographic examination of the nasal cavities. Contrast
radiographic examination of the skull, performed after instilling a radiocontrast medium into the affected nasal cavity through the external na ris, outlines the obstructing tissue. During rhinoscopic examination, an obstructing membrane is seen at the level of the caudal na ris, or the entrance between the nasal cavity and the nasopharynx may appear to be narrowed. The nasal portion of the ethmoidal labyrinth may appear atretic and distorted.

A foal affected with complete, bilateral choanal atresia can survive only if it receives a tracheotomy immediately after it is born. The persistent buccopharyngeal septum can be removed through an intranasal approach or through a facial bone flap. The intranasal approach should be reserved for those horses with a thin, membranous septum. The obstruction should be approached through a facial bone flap if the obstruction is bony, or if a membranous obstruction is thick.

Using an intranasal approach and endoscopic guidance, the buccopharyngeal septum is obliterated by electrocoagulation or with a laser, or excised using laparoscopic scissors and forceps. Cicatrization at the site of resection or obliteration is a common sequel but can be lessened by inserting a tube through the nasal cavity into the nasopharynx and maintaining the tube in this position for 4 to 6 weeks. Removing the nasal septum may decrease the incidence of formation of an obstructing cicatrix.

A thick, membranous septum or a bony septum must be removed through osteoplastic flaps, but facial deformity and dental malocclusion may develop due to decreased growth of the maxillae caused by disruption of facial suture lines. Horses with life-threatening choanal atresia can be treated by permanent tracheostomy if surgery is economically impractical or has failed.

**Deformity of the Nasal Septum**

The nasal septum is a cartilaginous plate, covered with a highly vascular mucosa, that extends rostrally from the ethmoidal turbinates to the alar cartilages and separates the right and left nasal cavities. At its dorsal border, the parietal cartilage curves outward from it on each side for a short distance. The ventral border of the nasal septum rests in the bony groove of the vomer bone and palatine processes of the premaxillae.

Septal thickening or deviation obstructs airflow through the nasal cavities. The septum can be thickened from congenital cystic degeneration, hamartoma formation, trauma from fracture of the nasal bones, neoplasia, mycotic infection, and bacterial infection associated with severe respiratory infection. Septal deviation can occur from an expanding mass within the paranasal sinuses and is a feature of wry nose.

Horses with a deformed nasal septum may experience respiratory difficulty during exercise or exhibit abnormal respiratory noise without apparent respiratory impairment. Asymmetry of the face is apparent if the septal abnormality is associated with wry nose or is caused by expansion of a mass within the paranasal sinuses or from trauma to the nasal bones.

Most septal abnormalities can be seen or palpated, and a greater flow of air through one nostril than the other can often be detected. Narrowing of one or both nasal cavities from deformity of
the septum may make insertion of an endoscope into a nasal cavity difficult. The septal cartilage and soft tissues surrounding it are visible on a dorsoventral radiographic projection, but positioning must be precise to observe septal deformity.\textsuperscript{33}

Deformity of the nasal septum does not resolve, and so, affected horses are treated by excising the accessible, rostral three-quarters of the septum.\textsuperscript{31} The horse’s hematocrit should be determined before surgery. Replacing blood during surgery is usually not necessary unless the horse is anemic, but the horse should receive a balanced electrolyte solution intravenously during surgery to avoid hypovolemia caused by severe hemorrhage.

The septum is excised with the horse anesthetized and in lateral recumbency. Gas anesthesia is delivered through an endotracheal tube inserted through a temporary tracheostomy. A small trephine hole (Fig. 4) is made through the nasal bones on the midline of the face, just rostral to the dorsoconchal sinuses, where the nasal bones begin to diverge. The nasal bones are exposed through a straight or curvilinear incision through the skin and periosteum, which are reflected. The hole created in the nasal bones exposes the parietal cartilage of the septum, which is excised or incised longitudinally on each side of the septum to expose the nasal cavities to provide access for the caudal and dorsal septal incisions.

![Figure 4. A small trephine hole is created rostral to the paranasal sinuses to provide access for removing the nasal septum.](image)

The septum can be incised dorsally, ventrally, and caudally using a guarded chisel, a cartilage scissor, or obstetrical wire.\textsuperscript{31,34} Hemorrhage is minimized by removing the nasal septum with obstetrical wire because when using obstetrical wire, three of the four incisions required to remove the septum can be made simultaneously. To remove the septum using obstetrical wire, three separate loops of wire are placed around the ventral, dorsal, and caudal aspects of the septum. The caudal incision should be made at a 60° angle to the nasal bones (towards the nasopharynx) so that the caudal cut edge of the septum resides within the nasopharynx, rather than between the conchae. The caudal, ventral, and dorsal septal incisions are made simultaneously by the surgeon and two assistants with the 3 loops of wire. The dorsal and ventral borders of the septum are incised with the wire to about 3 cm caudal to the rostral end of the cartilage. The rostral incision, made with a scalpel, connects the dorsal and ventral incisions. The septum is removed through either naris using a Vulsellum forceps. The nasal cavity is packed...
tightly with rolled gauze to stop hemorrhage, and the nostrils are sutured closed to retain the packing.

The cutaneous incision is closed with sutures or skin staples, and the endotracheal tube is replaced with a tracheostomy tube before or after the horse recovers from anesthesia. Gauze packing is removed at 12 to 24 hours. The horse can be returned to exercise about 8 weeks after surgery.

Removing the septum restores full respiratory capacity, but most horses continue to make an abnormal respiratory noise at exercise. Most horses continue to make an abnormal respiratory noise at exercise. The nasal bones may collapse into the common nasal cavity if surgery is performed when the horse is less than 6 months old.

**Mycotic Nasal Infection**

Mycotic nasal infection of horses is caused most commonly by the saprophytic fungus, *Aspergillus fumigatus* and less commonly by the saprophytic fungus, *Pseudallescheria boydii*. These fungi are secondary invaders of damaged tissue, and so, they are most likely to infect mucosa of the nasal cavities or paranasal sinuses damaged by accidental trauma, by an expanding mass, or by sinonasal surgery. Horses that live in a cool, humid climate and are confined to a stable containing moldy bedding are most likely to develop mycotic nasal infection. The condition is uncommon but is more often reported in Europe than in North America.

Horses with a mycotic nasal plaque have a chronic, malodorous, unilateral, blood-tinged, mucoid, purulent, or mucopurulent nasal discharge. Affected horses often have an ipsilateral submandibular lymphadenopathy. Mycotic plaques and sometimes destruction of the conchae are identified during rhinoscopy.

Mycotic nasal infection is diagnosed based on clinical signs, identifying fungal plaques endoscopically, cytological examination of exudate or sections of plaques, histologic examination of lesions, and culture of a heavy, pure growth of *Aspergillus fumigatus* or *Pseudallescheria boydii*. Failure to endoscopically identify a mycotic plaque does not eliminate *Aspergillus fumigatus* or *Pseudallescheria boydii* as a cause of clinical signs. Fungal hyphae and conidiophores are seen during histologic examination of lesions or cytological examination of exudate from horses infected with *Aspergillus spp* or *Pseudallescheria boydii*. Antibodies to *Aspergillus fumigatus* serotypes cannot be detected during serologic evaluation of horses affected with nasal mycosis.

Horses affected with nasal mycosis are treated by removing the mycotic plaques and diseased tissue transendoscopically and by administering a concentrated solution of an anti-mycotic drug topically to the infected tissue, through a catheter inserted into the ipsilateral paranasal sinuses or transendoscopically, once or twice daily, for one to two weeks. Anti-mycotic drugs effective topically against *Aspergillus spp* include itraconazole, fluconazole, enilconazole, miconazole, ketoconazole, natamycin, and clotrimazole.

**Fungal Granulomas**
Fungal diseases capable of causing granulomas in the nasal cavities of horses include rhinosporidiosis, conidiobolomycosis, cryptococcosis, and coccidioidomycosis. Although these fungal diseases are generally uncommon, they are encountered frequently in some geographic regions.

Rhinosporidiosis is chronic granulomatous disease of the nasal, vaginal, ocular, or oral mucosa of people, cattle, horses, mules, and other species caused by the fungus, *Rhinosporidium seeberi*. Only the nasal form has been reported to occur in horses. The disease occurs in horses that experience prolonged contact with stagnant water. Reports of horses affected with rhinosporidiosis in the USA have come from southeastern states.

Conidiobolomycosis of horses is a pyogranulomatous disease of the mucosa and submucosa of the nasal cavities and nasopharynx caused by infection by the fungus, *Conidiobolus coronatus* (Fig. 5). Conidiobolomycosis is part of the pyogranulomatous disease complex known as equine phycomycosis. The other diseases of the phycomycosis complex, pythiosis and basidiobolomycosis, occur in tropical and subtropical regions and primarily affect the skin and subcutis. Conidiobolomycosis, in addition to being found in tropical and subtropical areas, also occurs in more temperate climates and is found exclusively in the upper portion of the respiratory tract. Infection by *C. coronatus* most likely occurs while the horse is grazing on soil containing decomposing organic material contaminated by the fungus. The infective conidia may enter mucosa that has been damaged.

![Figure 5. Granulomatous lesions of conidiobolomycosis in the left nasal cavity and left side of the nasopharynx.](image)

Cryptococcosis is granulomatous fungal disease, usually of the skin, brain, or respiratory tract, caused by the yeast-like organism, *Cryptococcus neoformans*, a saprophyte commonly found in soil fouled by bird droppings. The site in horses most frequently infected is the nasal cavity.

Respiratory infection is acquired by the inhalation of contaminated dust.

Coccidioidomycosis, also known as valley fever, San Joaquin fever, and desert fever, is a granulomatous disease found in arid and semiarid regions and is caused by infection with the
fungus, *Coccidioides immitis*. Coccidioidomycosis of horses may be manifested as a nasal granuloma or by generalized debilitation. Horses become infected by inhaling the organism. Clinical signs displayed by horses with a fungal granuloma may include stertorous breathing, dyspnea, halitosis, sneezing, dysphagia, epistaxis, and sanguinolent, mucoid, or mucopurulent discharge from the affected nasal cavity. Granulomas can sometimes be seen at the external nares, but rhinoscopy may be necessary to observe the lesions.

Nasal granulomas of horses caused by infection by *Rhinosporidium seeberi* are pedunculated or sessile, pinkish-tan, and usually < 3 cm in diameter. Most are visible on the nasal mucosa close to the external naris, but smaller, nodular granulomatous lesions may be found scattered throughout the nasal cavities during rhinoscopic examination. The nodules contain 1-mm diameter aggregates of sporangia, which appear as white or yellow dots.

*C coronatus* causes 1- to 5-cm diameter growths, in the nasal cavity or in the nasopharynx. The lesions can sometimes be seen to contain 2- to 5-mm diameter granules. A granulomas caused by *C neoformans* or *C immitis* may appear as a glistening, yellow to yellowish-grey, gelatinous mass during rhinoscopy. Thickly encapsulated, PAS-positive, 5-to 30-µm diameter, yeast bodies are seen during microscopic examination of sections of lesions caused by *C neoformans*. Microscopic examination of lesions caused by *C immitis* reveals 20-to 50-µm diameter spherical bodies with a double-contoured cell wall resembling an oocyst of a coccidium.

Diagnosis of conidiobolomycosis is confirmed by identifying hyphae characteristic of *C coronatus* during histologic examination of infected tissue, by culturing *C coronatus* from a biopsy, or by an immunodiffusion test, using serum from the affected horse. Diagnosis of cryptococcosis is confirmed by identifying the characteristic, thickly-encapsulated, budding yeast bodies of *C neoformans* during cytological examination of nasal exudate stained with PAS or India ink.

Horses affected with rhinosporidiosis are treated by excising or freezing the lesions. Because lesions of conidiobolomycosis are difficult to excise because of their location, horses affected by conidiobolomycosis are treated by administering antifungal drugs parenterally and topically. Drugs used successfully include fluconazole (5mg/kg, per os, q12h, for 6 weeks) and amphotericin B (0.2-1.0 mg/kg, IV, in 1L 5% dextrose over 20 to 30 minutes every other day; or intralesionally [10 to 20 mg/lesion]). Treatment of affected horses with sodium iodide administered systemically (67 mg/kg, IV, once daily) for 2 to 5 days followed by oral administration of organic iodide (ethylenediamine dihydroiodide) (40mg/kg, PO, once daily) indefinitely may also be effective.

Medical treatment of horses with a nasal granuloma caused by *C neoformans* is usually unsuccessful because the gelatinous capsule surrounding the yeast masks the attached opsonic antibody, protecting the organism from phagocytosis. Horses with a nasal granuloma caused by *C neoformans* have been treated by excising or freezing the granuloma, coupled with parenteral treatment with sodium iodide and an antifungal drug, such as amphotericin B or fluconazole (see discussion of treatment of horses with conidiobolomycosis for dosages). Horses affected with coccidioidomycosis can be treated by surgically removing the granuloma in conjunction with...
administration of an antifungal drug (see discussion of treatment of horses with conidiobolomycosis for drugs and dosages).

Nasal Amyloidosis

Amyloidosis is the term for a group of diseases characterized by deposition of a homogeneous, extracellular proteinaceous substance, amyloid, in tissue. The two primary types of amyloid are amyloid AA, which is derived from a serum alpha-globulin, a normal, acute-phase protein produced by the liver, and amyloid AL, which consists of monoclonal, immunoglobulin light chains and fragments of light chains. Amyloid AA can be deposited in various tissues when its seruminal concentration is chronically elevated by inflammation or antigenic stimulation. Nasal amyloidosis is a disease peculiar to horses and is not associated with any underlying disease. Lesions of nasal amyloidosis found in horses are composed of amyloid AL.

Clinical signs of horses with nasal amyloidosis include epistaxis, respiratory impairment, and abnormal respiratory noise. Multiple, mucosa-covered nodules may be seen at the external nares, and the nasal septum and alar folds may be thickened by deposits of amyloid. Lesions may be seen extending into the nasopharynx during rhinoscopy.

The cut surface of lesions is pale yellow and waxy. Histologically, the nasal mucosa is intact, and the submucosa contains an acellular, amorphous, homogeneous, eosinophilic material (i.e., amyloid) and a few histiocytes and multinucleated giant cells. Congo red, a stain specific for amyloid, stains amyloid orange-red.

Medical therapy appears to be useless, and so, the only treatment is to excise the masses (or affected structures, such as the alar folds or nasal septum). Removing the lesions apparently effects cure, but lesions are often surgically inaccessible making complete removal impossible.

Neoplasia

Sinonasal neoplasia of horses is uncommon, but the most common sinonasal neoplasm of horses is the carcinoma (Fig. 6). Because of the diversity of tissue found in the paranasal sinuses and nasal cavities, other neoplasms, such as fibroma, myxoma, chondroma, osteosarcoma, fibrosarcoma, neurofibroma, hemangiosarcoma, and lymphoma, can also be found. Types of carcinomas are the squamous cell carcinoma, adenocarcinoma, and undifferentiated carcinoma. Old horses are more at risk for sinonasal neoplasia than are young horses, and old horses are especially at risk for carcinoma. Fibro-osseous tumors are found most frequently in young horses.
Clinical signs of sinonasal neoplasia typically develop insidiously and can include stertorous respiration, reduced airflow from the affected nasal cavity, and unilateral, malodorous, purulent, mucopurulent, sanguineous, or serosanguineous nasal discharge. The affected horse may have ipsilateral enlargement of the submandibular lymph nodes, epiphora, and distortion of the facial bones and may exhibit lethargy, anorexia, and weight-loss. The neoplasm may be visible at the external naris, but rhinoscopy or sinoscopy may be required to observe the mass. A horse suspected of having sinonasal neoplasia should have its oral cavity examined because some sinonasal carcinomas develop in the hard palate.

Squamous cell carcinoma can be classified histologically as being well-differentiated, moderately differentiated, or poorly differentiated. Grading of sinonasal carcinomas has little value, though, because grading is subjective and results vary with the site of sampling. The grade of differentiation and the tendency to metastasize are poorly correlated. Although they are quite invasive, most sinonasal tumors are slow to metastasize.

Definitive diagnosis of nasal neoplasia is based on results of cytological and histological examination of the abnormal tissue. Care should be taken during cytological examination of a fine-needle aspirate from a nasal mass not to mistake dysplastic cells found in severely inflamed mucosa for neoplastic cells.

Most horses with sinonasal neoplasia are eventually euthanized. Sinonasal neoplasia is usually advanced when recognized, and lesions are relatively inaccessible, making complete surgical excision or cryotherapy difficult or impossible. Horses have been treated successfully for sinonasal carcinoma using cobalt radiotherapy, but multiple treatments requiring general anesthesia are necessary, and the equipment required is not readily available. The life-expectancy of a horse with sinonasal neoplasia depends on the type of neoplasm and the owner’s ability to tolerate the clinical signs displayed by the affected horse.

Osteomas

Osteomas are smooth, solitary, sessile or pedunculated, osseous growths protruded from the surface of a bone, usually a bone formed by intramembranous ossification, such as the bones of the nasal cavities and sinuses. Some pathologists regard them to be hamartomas and as such, osteomas are benign, disordered overgrowths of mature bone. They apparently do not undergo malignant transformation. Histologically, osteomas are composed of a central core of cancellous bone surrounded by a peripheral layer of dense compact bone.

Although female horses can be affected by sinonasal osteoma, in nearly all reports of horses affected with sinonasal osteoma, the affected horse has been male. Most osteomas are probably present at birth, although years may elapse before clinical signs are recognized. They may grow slowly and then may cease growth and remain quiescent for years. Clinical signs of a sinonasal osteoma include mucopurulent nasal discharge, epiphora, restricted air-flow, and facial distortion. Diagnosis is based on results of physical, rhinoscopic, and radiographic examinations. An osteoma may sometimes be discovered incidentally during radiographic examination of the skull performed for reasons other than clinical signs caused by the osteoma.
Removing an osteoma from the nasal cavity may require creating a nasal flap. If removed completely, osteomas do not recur. Incomplete removal does not appear to stimulate growth.

**Osteodystrophia Fibrosa**

Osteodystrophia fibrosa is characterized by deposition of fibro-osseous tissue in bones, especially the facial bones, and is a manifestation of nutritional secondary hyperparathyroidism caused by diets low in calcium or by diets containing 3 or more times as much phosphorus as calcium, regardless of whether the calcium content is deficient. This disease causes thickening of the mandible, maxillae, conchae, and other facial bones, resulting in enlargement of the head. Other names for the disease include miller’s disease, bran disease, and bighead.

Excessive dietary intake of phosphorus inhibits absorption of calcium, by unknown mechanisms, causing hypocalcemia, which in turn, stimulates secretion of parathormone. Parathormone causes resorption of bone and deposition of fibro-osseous tissue preferentially in facial bones. Growing horses are affected most commonly.

Affected horses have bilaterally symmetrical, firm, enlargement of the facial bones dorsal and rostral to the facial crests and thickening of the horizontal rami of the mandible. Deposition of fibro-osseous tissue in the maxillae, conchae, and hard palate occludes the nasal cavities. Horses severely affected may have loose teeth and may be cachectic because of difficulty masticating. They may be lame because of fractures and avulsion of ligaments.

Diagnosis of osteodystrophia is based on characteristic skeletal changes. Radiographic examination may reveal loss of trabeculation of long bones and increased radiolucency of all bones, especially the facial bones. Seruminal concentrations of calcium and phosphorus are usually normal because of the compensatory action of the parathyroid glands, but diagnosis of nutritional secondary hyperparathyroidism can sometimes be aided by detecting a low concentration of calcium and a high concentration of phosphorus in the urine.

Horses affected with osteodystrophia fibrosa should be treated by correcting the dietary calcium: phosphorus ratio to 1.5 to 1, but facial appearance and respiratory capacity of severely affected horses may fail to improve. Resection of occluding nasal conchae has been described, but permanent tracheostomy may be more practical.

**References and Footnotes**


b. Schumacher J, Knoxville, TN (personal observation).