Diseases of the Nasal Cavities and Paranasal Sinuses (1-Mar-2002)

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Introduction

Diseases affecting the nasal cavities and paranasal sinuses in the horse are especially clinically significant compared to other species since the horse is an obligate nasal breather. Diseases affecting this region may result in impairment of nasal airflow and may ultimately lead to loss of performance in athletes. More commonly, such diseases can lead to the production of chronic nasal discharge and facial distortion.

The horse has a requirement for rapid delivery of large volumes of air, at the correct temperature and humidity for maximal athletic performance, during which time airflow can increase up to 60 times resting values. Significant airflow resistance exists in the upper respiratory tract and 80 - 90% of this resistance occurs in the nasal cavity [1], most of which is believed to be attributable to the collapsible nostrils. Nasal airway diameter is increased during exercise by dilation of the nostrils, retraction of the false nostrils and vasoconstriction of the venous plexi covering the nasal conchae [2].

Anatomy of the Nasal Cavities and the Paranasal Sinuses

The Nasal Cavities - The horse has large, mobile nostrils, to facilitate the obligatory nasal breathing, which like the nasopharynx and larynx are unsupported [3], and require muscular action to dilate, especially during inspiration at exercise. The nostrils are supported medially by the comma shaped alar cartilages, which are connected by a loose joint or by fibrous tissue to the nasal septum [4]. This anatomical arrangement allows great mobility of the muzzle and nostrils as compared to other species [5]. The prominent medial ala of the cartilage supports the alar fold, which extends caudally from it, dividing the nostril in two, with the blind dorsal compartment termed the "false nostril".

The nasal septum, which is composed of hyaline cartilage, divides the nasal cavity sagittally and is continuous caudally with the perpendicular plate of the ethmoid bone. Ventrally, the septum inserts into a groove on the palatine processes of the incisive bones and on the maxilla and vomer bones caudally [2]. The nasal septum is covered on its ventral aspects by an extensive venous plexus, which *in vivo* protrudes laterally into the common nasal meati (the "swell body"). The dorsal and ventral conchae divide the nasal cavity longitudinally into the dorsal, middle and ventral meati, of which the ventral meatus has the largest diameter (Fig. 1). The caudal aspect of the ventral nasal meatus communicates with the nasopharynx through the almost horizontally angled choanae.

Figure 1. Endoscopic view of the ventral nasal meatus of a clinically normal horse, showing the convex ventral aspect of the ventral concha. - To view this image in full size go to the IVIS website at www.ivis.org . -

The two nasal conchae (turbinates) in the horse consist of single scrolls of thin bone in a more simple arrangement (Fig. 2) than that in some other species, where there are secondary scrolls [6]. The conchae are attached laterally and scroll in opposite directions (medially) towards the middle meatus. The dorsal concha is the largest and the space enclosed within its caudal aspect (the dorsal conchal sinus; formerly termed - the turbinate part of the frontal sinus) is continuous caudally with the frontal sinus. The rostral aspect of the dorsal and ventral conchae are divided into scroll-like cells that communicate with the middle meatus. The space enclosed within the ventral concha, termed the ventral conchal sinus, communicates with the

rostral maxillary sinus. The conchae, and in particular their more ventral aspects, are covered by an extensive submucosal venous plexus. The rostral aspect of the ventral concha is continuous with the alar fold. Both conchae are attached rostrally to the nasal bone by folds of highly vascularized mucous membrane at the level of the first cheek teeth (2nd premolars).
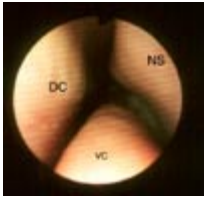

Figure 2. Endoscopic view of the middle nasal meatus, showing the swell body of the nasal septum (NS), the dorsal concha (DC) and the ventral concha (VC). - To view this image in full size go to the IVIS website at www.ivis.org . -

The six endoturbinates (ethmoturbinates) form the ethmoid labyrinth which extend rostrally from the cribiform plate of the ethmoid. They enclose spaces, termed ethmoidal sinuses, which sometimes communicate with the sphenoidal and palatine sinuses ventrally and the caudal maxillary sinus laterally, but do not communicate directly with the nasal cavity (J. McCann, 1999 unpublished observation). Twenty-five delicate ectoturbinates with secondary lamellae lie between the endoturbinates [4].

The Paranasal Sinuses - The exact function of the paranasal sinuses is not clear in any species. Numerous hypotheses of their function include: to add resonance to vocalization, to equalize pressure differences within the nasal cavity, to condition inhaled air, protection of intracranial structures from trauma, and reduction in the weight of the skull without sacrificing strength.

The paranasal sinuses in the horse consist of paired frontal, maxillary, dorsal conchal (DCS), ventral conchal (VCS), ethmoidal sphenoidal and palatine sinuses. The frontal and maxillary sinuses are air filled spaces formed by the evagination of the frontal and maxillary bones into the embryonic nasal cavity and these sinuses communicate directly with the nasal cavity [6]. The previously described DCS and VCS are enclosed within the nasal conchae. The left and right frontal sinuses are completely separated by a thick, midline bony septum. The frontal sinuses extend caudally to a line just rostral to the temporomandibular joint in the adult, or to the caudal aspect of the bony orbit in the foal [7]. The dorsal margins of the maxillary sinuses are clinically described as being delineated by an imaginary line joining the medial canthus of the eye to the nasoincisive (nasomaxillary) notch. Rostrally, the conchal part of the frontal sinuses extends to where the nasal bones diverge towards the orbit [8], rostral to the suture line between the nasal and frontal bones. A convexity exists on the floor of the frontal sinus due to the presence of the underlying ethmoidal labyrinth. A large, oval fronto-maxillary opening that connects the caudal maxillary and frontal sinuses lies on the lateral floor of the frontal sinuses, slightly rostral to the medial ocular canthus.

The maxillary sinuses in the horse are unique in being divided into separate rostral and caudal parts by a thin, cribiform bony septum, which is variable in position [4] and is often incomplete in the donkey. This septum is angled obliquely and its rostral aspect can vary in position, usually overlying the apices of the 2nd and 4th maxillary cheek teeth, commonly *circa* 5 cm caudal to the rostral aspect of the facial crest in the adult horse [8,9]. The infraorbital canal that runs longitudinally through the maxillary sinuses divides the maxillary sinuses into medial and lateral compartments. In younger horses, the air spaces of the sinuses are largely obliterated by the reserve crowns of the 3rd to 6th cheek teeth (4th premolar to 3rd molar) which are embedded in the alveolar bone, ventral to the infraorbital canal. As the teeth erupt in older horses, the lateral and medial compartments become separated by a thin bony plate that lies ventral to the infraorbital canal, and the air space within the sinuses gradually increases.

The rostral border of the rostral maxillary sinus (RMS) is usually at the level of the third cheek tooth. The ventral conchal sinus (VCS) forms the medial compartment of the rostral maxillary sinus. However, the VCS extends caudally, medial to most of the length of the caudal maxillary sinus. It drains dorsally over the infraorbital canal into the rostral aspect of the rostral maxillary sinus via a narrow conchomaxillary opening. The caudal portion of the VCS contains several lamellae and its caudal aspect forms the bulla of the ventral conchal. The rostral and caudal maxillary sinuses communicate with the middle nasal meatus through a curved, slit-like orifice that is divided by a bony plate continuous with the septum dividing these sinuses [2,10]. However, some authors describe an ostia common to both compartments [6,9]. These ostia are bounded dorsally by a fold of mucous membrane which is continuous with the margin of the fronto-maxillary opening, and ventrally by fold of mucous membrane continuous with the ventral concha [11]. The caudal maxillary sinus usually communicates with the sphenoidal and palatine sinuses (which lie ventral to the ethmoturbinate labyrinth and the base of the skull) through a

variably sized opening, caudomedial to the infraorbital canal.

All the paranasal sinuses are lined with a well vascularized mucoperiosteum, which is innervated by the ophthalmic nerve in the frontal and sphenopalatine sinuses, and by the maxillary nerve in the maxillary sinus [12]. The nasal cavity and paranasal sinuses are lined by ciliated columnar epithelium with variable numbers of mucus producing cells and acinar glands [9,13,14].

Examination of the Nasal Cavities and Paranasal Sinuses
Clinical Examination - Investigation of sinonasal diseases should commence by obtaining a thorough history and performing a clinical examination. Many diseases affecting this region are chronic and the duration and progression of signs are particularly important diagnostically. The majority of horses present with a history of facial swelling or nasal discharges, which are often chronic, unilateral and purulent in nature [15]. Additionally, the presence or absence of ipsilateral submandibular lymphadenopathy and epiphora, head-shaking, abnormal respiratory noises and nasal airflow obstruction may all be relevant.

A full clinical examination of the horse for signs of systemic disease should be performed. Examination of the upper respiratory tract should include palpation of the submandibular lymph nodes, percussion of the maxillary and frontal sinus areas, and use of material, such as fine cotton, to detect airflow asymmetry between the left and right nostrils. An examination of the oral cavity using a full mouth speculum is essential if sinonasal disease secondary to dental disease or sinonasal neoplasia is suspected. Nasal swabs can be collected from any nasal discharges and cultured for bacteria, particularly if *Streptococcus equi var. equi* (Strangles) infection is suspected.

Endoscopic Examination - Examination of the nasal cavities with a flexible endoscope is an essential part of a thorough investigation. Nasal endoscopy is possible in conscious horses although restraint in the form of nose or ear twitches or chemical sedation may be necessary in some horses. Examination of both left and right nasal cavities via both the ventral and middle nasal meati should be performed. Examination of the aspect of the middle meatus into which the nasomaxillary ostia drain from the rostral and caudal maxillary sinuses, sometimes termed the "maxillary drainage angle", is particularly important (Fig. 3). Compression of the nasal meati by expansive lesions, inflammation of the nasal mucosa, exudation from the caudal drainage angle, distortion or erythema of the nasal conchae or endoturbinates, or exudation from the ethmoturbinates may also be indicative of sinonasal disease. Endoscopic examination should include examination of the guttural pouches where relevant, although chemical sedation may sometimes be necessary to perform this safely. Endoscopy of the lower respiratory tract is advisable to differentiate sinonasal discharges from pulmonary secretions, which are more commonly, but not always bilateral, and which usually accumulate in the lower trachea.
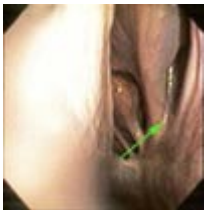

Figure 3. Endoscopic view of the caudal nasal cavity showing the "drainage angle" (arrow) where exudates from the rostral and caudal maxillary sinuses discharge. - To view this image in full size go to the IVIS website at www.ivis.org . -
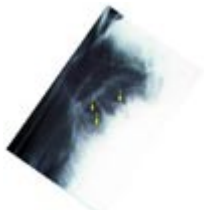

Figure 4. Lateral radiograph of the head of a horse with sinusitis showing multiple fluid lines (arrows) in several compartments of the maxillary sinuses. - To view this image in full size go to the IVIS website at www.ivis.org . -

Radiographic Examination - Horses with suspected sinus disease, e.g., those with nasal discharges or gross facial swellings should have a radiographic examination [16]. Diagnostic radiographs of the paranasal sinuses and surrounding structures are possible with portable machines, due to the good radiographic contrast between the air in the sinuses and the more radiodense adjacent structures. Standard views include straight lateral, lateral 30ºdorso-lateral oblique, lateral 15ºventro-lateral oblique (open-mouthed), and dorsoventral projections. Radiographs should always be taken in sedated horses. The horse's head can

be supported on a stand to reduce movement. Fluid exudates within the paranasal sinuses may be observed as horizontal fluid lines on the straight lateral views (Fig. 4). The oblique projections allow examination of the apical (lateral 30ºdorso-lateral oblique) and occlusal (lateral 15ºventral-lateral open-mouthed oblique) areas of the maxillary cheek teeth. Dorsoventral radiographs enable detection of nasal septum deviation, and comparison of the left and right nasal chambers, and the presence of masses or exudates in the ventral conchal sinuses.

Sinoscopy - Sinoscopy (intrasinus endoscopy) allows the direct visualisation of normal structures and possibly of lesions within the frontal and maxillary sinuses and is a useful ancillary technique to complement those already described. Sinoscopy may be performed with a rigid arthroscope [17] or more usually a flexible endoscope [18]. A small osteotomy is created over the frontal or rostral maxillary sinuses. The site for a frontal osteotomy lies rostral to an imaginary line joining the lateral canthi of the eyes and caudal to an imaginary line joining the medial canthi, 2 cm abaxially from the midline. A rostral maxillary osteotomy may be created approximately 2 cm dorsal to the facial crest, caudal to its rostral limit, or where radiographs indicate the extent of the RMS to lie. After clipping and aseptically preparing the site, 1 ml of local anesthetic e.g., mepivicaine (Intra-Epicaine, Arnolds, UK) is administered subcutaneously. A 2.5 cm longitudinal skin incision is made through skin and periosteum. A modified approx. 10 mm diameter drill bit or trephine is used to create a portal sufficient in diameter to allow passage of the endoscope. The endoscope is then passed via this osteotomy portal in the frontal or maxillary bones into the sinuses. The presence of exudates within the sinuses can obscure sinoscopic examination and the view may be improved after lavage of the sinuses to remove purulent exudates, followed by a period of 30 - 60 minutes to allow drainage of excess lavage fluid.

Scintigraphy - Gamma scintigraphy may also be used to confirm other clinical or ancillary diagnostic findings, e.g., to assist with the diagnosis of sinusitis secondary to dental disease when radiographs of dental apices are inconclusive. Focal regions of increased radiopharmaceutical uptake may be observed in the infected dental apex and the surrounding alveolar bone (Fig. 5). Early reports [19] suggest that this technique is more sensitive than radiology for the detection of early dental apical infection. In addition, information about the size and extent of sinus cysts and tumors may be gained from scintigraphy.
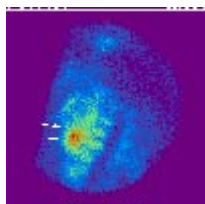

Figure 5. Bone phase gamma scintigraph using Technetium 99-MDP, showing a region of increased radiopharmaceutical uptake over the second and third maxillary cheek tooth (Triadan 407, 408) (arrows). - To view this image in full size go to the IVIS website at www.ivis.org . -

Diseases of the Nasal Cavities
Congenital Defects - The most common congenital defect of the nasal passages involves a lateral deviation of the muzzle termed "wry nose" due to gross deformities of the nasal, incisive and maxillary bones, and the nasal septum [20] (Fig. 6). Less commonly, the rostral mandible may be affected, or affected animals may also have a cleft palate. Affected foals may have difficulty sucking, but often even gross displacement of the incisors does not preclude sucking. Abnormal breathing noises may be present even at rest, due to the marked curvature of the nasal septum, which protrudes towards the convex side of the face. The etiology of this disorder is unknown [21] but genetic factors and adverse intrauterine conditions have been suggested as causes [22]. Surgical correction has been attempted by creation of osteotomies in the premaxilla and orthodontic realignment of the incisors, with some improvement in nasal airflow and in cosmetic appearance [23]. Such treatments are technically difficult, and the result usually does not enable normal athletic activity [24].

Figure 6. Adult mare showing severe wry nose. - To view this image in full size go to the IVIS website at www.ivis.org . -

False Nostril Cyst - These lesions, also referred to as atheromata, or epidermal inclusion cysts of the false nostril are usually observed in animals of 1 - 3 years of age. Sebaceous glands are not usually identified histologically in their lining, and the terms sebaceous cyst, or atheroma are therefore inappropriate. The condition is rare [25] and usually unilateral. These non-painful lesions occur on the lateral or dorsolateral aspect of the nasal diverticulum (Fig. 7). Treatment is not always necessary, since airflow obstruction (and abnormal respiratory noises) are not a feature of these lesions. However, surgical removal may be indicated for cosmetic reasons or if the lesions impinge on a noseband. Aspiration of the cysts usually results in recurrence or occasionally in local abscessation [24]. Removal of the cyst lining in toto, is the treatment of choice, and can be performed in the standing horse [21,26]. The skin over the lesion is aseptically prepared and desensitized using regional or perineural infraorbital infiltration of local anesthetic. A longitudinal incision is carefully made over the cyst and the cyst is dissected free without incising its lining. Histology of the lining of such lesions reveals keratinised or non-keratinised squamous epithelium [14,27].

Figure 7. A false nostril epidermal inclusion cyst (arrows) usually presents as an asymptomatic unilateral swelling of the lateral aspect of the false nostril. - To view this image in full size go to the IVIS website at www.ivis.org . -

Nasal Paralysis - Paralysis of one or both nostrils may be caused by damage to branches of the facial nerve [21]. This can include nose-band trauma, bilaterally to the dorsal buccal branches of the facial nerve, neoplasia, guttural pouch mycosis, nostril lacerations, facial abscesses or iatrogenic cryosurgical damage to the facial nerve or its dorsal buccal branches [24,28].

Alar Fold Abnormalities - Abnormally large or flaccid alar folds have also been rarely reported to cause airflow obstruction exercise intolerance and abnormal respiratory noises during fast exercise, especially in Standardbreds [29]. In severely affected cases, abnormal vibrating type respiratory noises may also be present at rest [30]. Direct visual examination of these folds immediately after fast exercise may reveal the presence of excessively prominent alar folds. Temporarily suturing the false nostril open to confirm the diagnosis is advised, prior to attempting surgical correction [21]. Resection of the alar folds is reported to offer a reasonable prognosis [21,31,32].

Amyloidosis - Amyloidosis is a group of diseases involving the deposition of an extracellular material, termed "amyloid", in a variety soft tissues [33]. Amyloidosis is rare in horses, with only 9 cases reported out of a total of 16,000 cases referred to a Veterinary hospital in The Netherlands [33]. The condition has been reported in horses of all ages [34]. In the horse the mucosa of the nasal septum is a predilection site for amyloid deposition, although widespread URT amyloidosis has been reported. Horses with nasal amyloidosis can develop epistaxis - due to ulceration of the mucosa over the amyloid deposit, abnormal respiratory noise, exercise intolerance, and inappetance. Lesions of the nasal mucosa may be visible by direct or endoscopic examination. Confirmation of the diagnosis is by histology of biopsy sections, where amyloid may be identified in polarized light following Congo-red staining. Treatment with topical or systemic corticosteroids has been unsuccessful, but surgical excision has occasionally been more successfully performed [34,35].

Mycotic Rhinitis - Equine sinonasal diseases associated with fungal infection are rare in the horse in the UK. Greet [36] first described 3 cases of mycotic rhinitis in horses caused by *Aspergillus fumigatus*, and subsequent reports are sparse. Of ten cases described by McGorum [37] *Aspergillus fumigatus* was cultured from six, *Pseudallescheria boydii* from one, and *Penicillium* sp. from a single case [37]. *Pseudallescheria boydii*, an opportunistic saprophyte has also been isolated from an equine nasal granuloma [38], and a frontal sinus lesion [39].
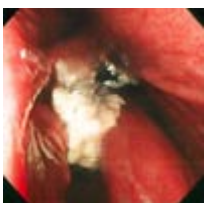
Figure 8. Mycotic plaques in the middle nasal meatus of a horse suffering from mycotic rhinitis (*Aspergillus fumigatus*). - To view this image in full size go to the IVIS website at www.ivis.org . -

Aspergillus Fumigatus is ubiquitous in dead vegetation including hay and straw. The mechanism of infection of the nasal chambers or paranasal sinuses by usually saprophytic fungi is not clear, but previous trauma from surgery or nasogastric tube passage may be factors in some cases [36,40,41]. Equine nasal aspergillosis (Fig. 8) does not usually result in raised serum antibody titres, and consequently diagnosis by current serological techniques is unreliable.

Mycotic rhinitis due to other fungal organisms is common in warm humid climates. Mycotic nasal granulomas are not uncommon in the Southern United States, Australia and Canada. Mycotic granulomas involving the nasal cavities and other organs have been reported due to: *Cryptococus neoformans* [40,42], *Coccidiodes immitis* [43-45] *Rhinospordium seeberi* [46] *Condiobolus coronatus* (*Entomopthora coronata*) [47] *Condiobolus lamprauges* [48] and *Hyphomyces destruens* [49]. Such mycotic granulomas are characterised by the presence of necrotic foci or "kunkers" within proliferative granulation tissue. They have been associated with nasal discharge, which may vary from purulent to sanguineous, and is frequently malodorous. Nasal airflow obstruction may occur with advanced lesions [44].

The treatment of superficial nasal mycotic lesions carries a good prognosis although recurrence is possible. Topical treatment of nasal aspergillosis with nystatin [36] or natamycin [37] has been reported [18]. Surgical removal of a phycomycotic nasopharyngeal granuloma, in combination with sodium iodide infusion has also been described [50,51].

Miscellaneous Diseases of the Nasal Cavities
Foreign bodies, such as twigs in the nasal chambers may result in nasal inflammation and partial nasal obstruction [52]. Deviations of the nasal septum occur secondary to trauma or to space occupying sinonasal lesions [18,53,54]. Rarely, extensive primary nasal septal deviation and thickening may cause partial respiratory obstruction necessitating removal of the septum. Surgical techniques for nasal septum removal have been described. These very traumatic procedures are accompanied by profuse hemorrhage [26,55] and collapse of the nasal cavity following septum removal of the septum is a potential long-term complication [56]. Proliferation of the maxillary bones due to nutritional secondary hyperparathyroidism causing nasal airway obstruction may be due to excessive dietary phosphorus [57-59], due to a diet consisting of excessive amounts of bran or grazing on certain tropical grasses.

Diseases of the Paranasal Sinuses
Sinusitis - Inflammation of the paranasal sinuses is relatively uncommon and may be due to primary bacterial or mycotic infections [18,60,61] or secondary to dental disease [18,61,62], facial trauma, maxillary cysts and sinonasal neoplasia [16,63]. Equine sinusitis is usually unilateral but bilateral disease has been reported [18,64,65]. There is apparently no breed, age or gender predisposition.

Clinical signs reported with sinusitis include; unilateral purulent nasal discharge (Fig. 9), ipsilateral submandibular lymph node enlargement and epiphora. Less commonly observed signs include: facial swelling, exophthalmos, abnormal respiratory noises, head-shaking and exercise intolerance [18,64,65]. The nasal discharge in primary sinusitis is usually purulent and odorless [60], but malodorous nasal discharges occurred with 42% of primary sinusitis [18] and has been associated with inspissation of pus in the VCS [66].

Figure 9. A unilateral purulent nasal discharge (arrows) is the most common clinical sign of sinusitis. - To view this image in full size go to the IVIS website at www.ivis.org . -

Culture of exudates from primary sinusitis cases has yielded a variety of bacteria including *Streptococcus equi var equi*, *Streptococcus equi var zooepidemicus* [66,67], *Corynebacterium* sp. [68], *Staphylococcus* [18,60,66], *Pseudomonas aeruginosa*, *Bacteroides* sp., *Peptostreptoccus* sp. [18,67] and *Eschericia coli* [60,66] although the etiological significance of many of these isolates is unclear.

Sinusitis is frequently associated with apical infections of the maxillary cheek teeth [18,60,61,65], which occurs most

frequently in horses aged 4 - 7 years [69]. Sinusitis can also occur secondarily to idiopathic dental fractures, severe diastemata, and supernumerary cheek teeth. Nasal discharge is frequently fetid when associated with dental secondary sinusitis, or nasal granulomas caused by 1st or 2nd (or occasionally 3rd) maxillary cheek tooth apical infection [70]. Anaerobes including; *Bacteroides fragilis*, *B. melaninogenicus*, *B. oralis*, *Fusobacterium mortiferum* have been cultured from nasal discharge with such dental infections [71].

Nasal endoscopy of horses with sinusitis usually reveals purulent exudate in the caudal nasal cavity, draining from the nasomaxillary ostia of the rostral or caudal maxillary sinuses (Fig. 10). Gross accumulation of exudates in the ventral conchal sinus results in swelling of the ventral concha, which may eventually prevent passage of the endoscope. Displacement of the nasal septum may even occur in severe cases. Straight lateral radiographs frequently reveal multiple fluid lines in some compartments of the paranasal sinuses. Oblique projections are necessary to radiographically separate the left and right rows of maxillary cheek teeth for radiographic examination of their dental apical areas. Radiography is an insensitive technique for diagnosis of dental apical infections. As noted previously, the presence of infection of a tooth apex can sometimes be confirmed by Gamma scintigraphy, which appears to be more sensitive than radiography, particularly in the early stages of the disease.

Figure 10. Endoscopic view of the "drainage angle" in the caudal nasal cavity showing purulent exudation (arrow), which originated from the maxillary sinuses. - To view this image in full size go to the IVIS website at www.ivis.org . -

Acute cases of primary sinusitis may spontaneously resolve or may respond to antimicrobial drug administration, with the organisms most commonly isolated from this type of sinusitis, being sensitive to penicillin. Horses with chronic sinusitis (2 or more months duration) [18] frequently have gross thickening of the sinus mucosa, which restricts normal nasomaxillary drainage and may have inspissated exudate in the affected sinus. Such cases are usually not responsive to antibiotics therapy. Lavage of the paranasal sinuses may be performed via a catheter sutured into an osteotomy in the frontal sinus, (for lavage of the frontal and caudal maxillary sinuses), or into the RMS. Such cases may respond to lavage with 5 - 10 l of water, saline or dilute disinfectants such as 0.05% povidine-iodine solution, once to twice daily for 5 - 10 days.

Cases with gross thickening of the sinus mucosa or with accumulations of inspissated pus in the sinus cavity may require surgical debridement, removal of inspissated exudate or sinonasal fistulation and drainage (Fig. 11). The frontal, maxillary and ventral conchal sinuses are most easily approached via a large nasofrontal bone-flap osteotomy [72] (Fig. 12). A maxillary osteotomy may be used in older horses, but the reserve crowns of the maxillary cheek teeth limit the access via this approach in younger animals. Bone flap osteotomies may be created under general anesthesia or in the standing horse under chemical restraint [73].

Figure 11. Sinoscopic view of inspissated purulent material in the ventral conchal sinus. - To view this image in full size go to the IVIS website at www.ivis.org . -

Figure 12. Skin incision for a nasofrontal surgical approach to the frontal and caudal maxillary sinuses, prior to creating a bone-flap. - To view this image in full size go to the IVIS website at www.ivis.org . -

After making a rectangular or curved incision through the skin and periosteum the three-sided bone flap is created with an oscillating saw, chisels or Gigli wires and then reflected back on the fourth (uncut side) with its periosteum and skin still attached (Fig. 13). Alternatively with smaller osteotomies, the fourth side, may be cut and the bone fragment may be discarded after elevating the periosteum.

Inspissated pus and grossly thickened mucosa is removed and sinus is lavaged. Drainage into the nasal cavity may be improved by creation of a fistula through the dorsomedial wall of the ventral concha, into the nasal cavity. Even when performed on the less vascular dorsal aspect of the medial conchal wall, this fistulation is usually accompanied by profuse hemorrhage, necessitating packing of the nasal cavity with gauze. The bone flap is replaced in situ (if retained) and secured with one or two wire sutures tightened in preplaced drill holes. The periosteum is closed with absorbable suture and the skin closed with staples or non-absorbable sutures. A simple plastic or Foley catheter, passing into a separate osteotomy in the frontal sinus, allows post-operative lavage of the sinuses.

Figure 13. Nasofrontal bone-flap, cut on three sides and elevated to reveal chronic sinus empyema with mucosal thickening in a case of sinusitis. - To view this image in full size go to the IVIS website at www.ivis.org . -

Sinusitis secondary to maxillary dental apical infections necessitates removal of the affected cheek tooth before resolution of the sinusitis will occur. Careful identification of the affected tooth using radiography (Fig. 14) or gamma scintigraphy is necessary to absolutely confirm the diagnosis. Infected cheek teeth may be removed via oral extraction, repulsion or via a lateral buccotomy. The latter technique is suitable for the rostral three teeth, but risks iatrogenic damage to the buccal nerve and parotid ducts. Extraction *per os* has considerably reduced complications as compared to repulsion, and additionally it may be accomplished in the standing horses. Dental extractions involving the caudal maxillary cheek teeth can be performed under general anesthesia via a bone-flap osteotomy or via a trephine. Intra-operative radiographs should be taken during and after dental repulsion to assist with dental punch placement and to identify the presence of remaining bone or dental fracture fragments, which have the potential to sequestrate. If dental extraction is performed *per os*, in horses with dental sinusitis, lavage of the affected paranasal sinuses should also be performed post operatively. Many cases of dental sinusitis require multiple treatments before total resolution of clinical signs, although the long-term prognosis is good [18].

Figure 14. Lateral 30ºdorso-lateral oblique radiograph showing sclerotic radio-opacities and loss of radiographic definition of the apical area of the fourth maxillary cheek tooth (arrows). - To view this image in full size go to the IVIS website at www.ivis.org . -

Sinus Cysts - Sinus cysts are expansive fluid-filled space-occupying lesions which, occur uncommonly in the sinuses of horses of all ages [74-76]. Congenital intra-sinus cysts have also been reported [77,78]. The etiology of these lesions is unclear and no breed or sex predisposition has been identified. It has been suggested that they are developmental in origin and associated with dental tissues [20] but evidence for this theory has not been found [79], although one of the cases described by Dixon [75] was attached to dental alveoli. A common etiology between these lesions and ethmoidal hematoma has been suggested [76], but little evidence for this association has been reported. Equine sinus cysts most commonly occur in the maxillary sinuses, but they have been reported in the other sinuses.

A consistent clinical feature caused by the expansive nature of the lesion is distortion of the facial, maxillary and conchal bones [8,76, 80]. This may result in gross facial swelling, epiphora and exophthalmos due to thinning of the overlying maxillary or frontal bones, and nasal obstruction due to the expansion of the lesion within the sinuses and conchae (Fig. 15). A nasal discharge, which varies from mucopurulent to purulent, is often present and is thought to be due to sinusitis secondary to obstruction of nasomaxillary drainage [68].

Figure 15. Unilateral facial swelling due to the expansion on a cyst within the maxillary sinuses. Sinus cysts also cause nasal airflow obstruction in many such cases. - To view this image in full size go to the IVIS website at www.ivis.org . -

Diagnosis is assisted by endoscopy, which may reveal distortion of nasal conchae. Radiographic features of sinus cysts include the presence of a rounded, expansive, soft-tissue density lesion in the frontal or maxillary sinuses. Sinoscopy via a frontal osteotomy may also demonstrate the lesion within the maxillary or frontal sinuses (Fig. 16). Distortion and thinning of the surrounding bones may be evident as the lesion increases in size, and secondary distortion of adjacent dental apices within the sinuses may be present. The contents of the cysts frequently appear radiographically as a homogenous soft tissue density shadow, sometimes containing spicules of mineralized tissue. A radiodense, partially mineralized capsule is sometimes visible, and extralesional fluid lines may be present if the lesion is associated with secondary sinus empyema. Centesis of the lesion via a sinusotomy or by sinus puncture through the thin bone using e.g., a 16G needle is diagnostic, yielding a viscous, usually sterile, translucent yellow fluid which is odorless and may contain some leucocytes [41,75,76]. Treatment of the lesion by surgical drainage may be effective in some cases [75,76,81] but removal of the lesion *in toto*, via a nasofrontal or maxillary osteotomy approach, under general anesthesia or standing chemical restraint (Fig. 17), is the treatment of choice [41,75,76].

Figure 16. Sinoscopic view (from the frontal sinus) of a maxillary sinus cyst expanding to almost occlude the frontomaxillary opening. - To view this image in full size go to the IVIS website at www.ivis.org . -

Figure 17. A maxillary bone-flap osteotomy has been performed in this horse under standing chemical restraint, resulting in the release of yellow viscous fluid from a maxillary cyst. - To view this image in full size go to the IVIS website at www.ivis.org . -

Histological examination of sinus cysts has revealed extensive resorption and remodelling of the bones surrounding the cyst; replacement of the normal bony septa within the sinus by fibrous tissue and replacement of the loose intrasinus connective tissue with bony spicules. The cysts themselves are lined by ciliated columnar respiratory epithelium with focal areas of ulceration, and evidence of sub-epithelial hemorrhage, and chronic inflammation [76,79].

Progressive Ethmoidal Hematoma (PEH)
Progressive ethmoidal hematoma (PEH) were first clinically described in detail by Cook and Littlewort [82], and Platt [83] comprehensively described the histology of these lesions. Despite many subsequent case reports of this lesion [84-88] their etiology still remains unclear. Angiomatoid lesions in the submucosa of the adjacent sinuses have been suggested as a possible cause of these lesions, resulting in areas of organizing submucosal hemorrhage. The lesions have been reported in horses 3 - 18 years of age [83] most commonly occurring in mature horses.

PEH lesions most frequently arise from the ethmoidal labyrinth, but lesions have been reported as arising within the sinuses [82] where they are usually attached to the sinus mucoperiosteum, and rarely invade the nasal passages. PEH lesions expand to fill the cavity in which they are developing, and ulceration of their walls results in intermittent low-grade hemorrhage. Pressure resorption of the surrounding bone may also rarely occur as the lesion expands. The lesions are usually unilateral, occurring with equal frequency on either side but bilateral lesions have also been reported.

Clinically, cases of PEH present with a chronic (e.g., several weeks or months), low-grade, unilateral, intermittent epistaxis, often with dark blood. In advanced cases, severe nasal obstruction and abnormal respiratory noise may be present, even at rest and may necessitate a tracheostomy [86]. However, facial distortion is rare even in advanced cases, in contrast to the facial swelling observed with sinus cysts. Head-shaking has been reported in conjunction with PEH, and lesions may rarely extend to involve the cerebral cortex resulting in neurological signs [21,41].

Diagnosis is straightforward if the lesion can be viewed endoscopically, usually lying rostral to the ethmoidal labyrinth (Fig.

18). The lesions have a distinct gross appearance varying in color from dark red to yellow-gray depending on the type and distribution of the hemoglobin pigments following the most recent intralesional hemorrhage. Absence of PEH lesions in the nasal cavity when viewed endoscopically does not preclude a diagnosis of PEH [89] as the lesions may be within the paranasal sinuses. Radiographically, a well-circumscribed soft tissue density is sometimes visible, usually extending from the ethmoturbinates in such cases (Fig. 19).

Figure 18. Endoscopic view of a progressive ethmoidal hematoma in the caudal nasal cavity. - To view this image in full size go to the IVIS website at www.ivis.org . -
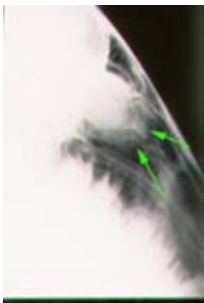

Figure 19. Radiograph showing a progressive ethmoidal hematoma (arrows) as a rounded soft-tissue radio-opacity lying in front of the orbits. - To view this image in full size go to the IVIS website at www.ivis.org . -

PEH lesions are covered with a respiratory epithelium, which may be ulcerated in some areas, that overlies a fibrous submucosa, forming a capsule 0.2 - 1 cm thick. Within the capsule some areas are of recent or organizing hemorrhage, while other firmer areas contain blood vessels and fibrous tissue [14].

Earlier this century, PEH lesions were considered neoplastic and affected animals were destroyed when lesions were identified [82]. Currently, surgical removal is frequently performed via maxillary or nasofrontal flaps [82,87] (Fig. 20). The proximity of the lesions to the cribiform plate and thus to the brain means that trauma to the olfactory lobes, and other structures during surgery is a potential risk [18,87]. More recent treatments for this disorder include surgical excision of the lesion trans-endoscopically with a Neodymium-Yttrium Aluminium Garnet laser [90]. Trans-endoscopic injection of the lesion with a 10% formaldehyde solution [41,91], has been successfully used but many lesions require multiple treatments (Fig. 21). However, neurological signs following damage to the olfactory lobes has been reported following formalin injection [92]. Recurrence after surgical removal are reported from 13% [87] to 50% [88] and 8% after trans-endoscopic laser ablation [90]. Mycotic rhinitis following surgical removal of PEH has been described [18,87], but is usually self-limiting.
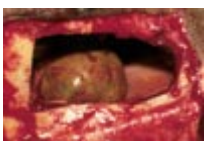

Figure 20. A nasofrontal flap osteotomy has been created to enable excision of the green, rounded, progressive ethmoidal hematoma visible in the frontal sinus of this horse. - To view this image in full size go to the IVIS website at www.ivis.org . -

Figure 21. This progressive ethmoidal hematoma in the caudal nasal cavity is being transendoscopically injected with 10% formalin. - To view this image in full size go to the IVIS website at www.ivis.org . -

Sinonasal Neoplasia
Neoplasia of the nasal and paranasal sinuses is a relatively rare condition in the horse [93-96] and reports of multiple cases of neoplasia involving the equine upper respiratory tract are few [94,97-101].

Squamous cell carcinoma is the most frequently reported tumor of this region in the horse with lesions reported originating in

the nasal cavities [94], paranasal sinuses [54,102] or pharynx [103]. Carcinomas originating in the equine rostral nasal cavity [74] and ethmoid labyrinth [104] and frontal sinus have been reported [101,105,106].

Other tumor types recorded include spindle cell sarcoma [107], mastocytomas, hemangiosarcoma [108], and angiosarcoma [109]. Lymphosarcoma involving the nasal cavities and URT have also been reported [101,110-112], with the nasopharynx being the most common site of this tumor.

A group of fibro-osseous lesions, often of uncertain tissue origin, have been reported in the nasal cavities and paranasal sinuses of horses. These include osteomas, which have been found in the frontal and maxillary sinuses [101,113,114] osteochondromas [115], fibromas [116] and fibrosarcomas [101,117]. Tumors of dental tissue origin with involvement of the maxillary sinuses have been reported, although such neoplasms more frequently affect the mandibular or rostral maxillary cheek teeth [118]. Dental tumors are more common in older animals [119], but have also been described in foals [120].

Clinical signs associated with neoplasia are similar to those of other expansive lesions in this area. Nasal discharge, facial swelling (Fig. 22), epiphora and nasal obstruction are all reported frequently. Head-shaking, exophtalmus and epistaxis are less commonly observed [41,106]. Surgical resection of benign lesions such as osteomas via a nasofrontal flap may carry a good long-term prognosis [41,100]. Benign melanomas causing partial nostril obstruction may be removed under standing chemical restraint (Fig. 23). However, the aggressive nature and surgically inaccessible anatomical location of most carcinomas and sarcomas, leads to a guarded prognosis after surgical resection [101]. Beta radiotherapy with Cobalt 60 has been attempted with limited success [121].
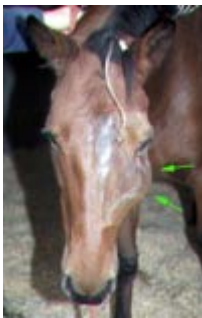

Figure 22. Sinonasal tumors are often accompanied by gross distortion of the frontal and maxillary bones (arrows) resulting in large facial swellings. They invariably also have nasal airflow obstruction due to expansion within the nasal cavity. - To view this image in full size go to the IVIS website at www.ivis.org . -

Figure 23. An unusual melanoma on the margins of the nostril which resulted in abnormal respiratory noises and airflow obstruction. - To view this image in full size go to the IVIS website at www.ivis.org . -

Traumatic Injuries of the Nasal Cavities and Paranasal Sinuses
Fractures involving the premaxilla (incisive bone) are common in foals [122] and depression fractures involving the frontal and maxillary sinuses have often been reported in adult horses [41,123,124]. Traumatic hemorrhage into the sinuses may lead to an unexpectedly prolonged (> 4 weeks) intermittent epistaxis. Open fractures may frequently lead to secondary sinusitis [125], and the presence of intra-sinus sequestra may result in chronic suppuration [126]. Horses with non-displaced fractures may be treated conservatively with a satisfactory result, particularly if the skin remains intact. Head fractures are frequently depression fractures of the frontal or maxillary bones, where repair is possible by elevating the depressed bone flap and, if necessary, immobilizing it in the reduced position with stainless steel wires. Reconstruction of the soft tissues over the fracture accelerates healing. The use of synthetic polymers for reconstruction of a facial fracture in a horse has been reported [127].

Nasofrontal Suture Exostoses
Swellings of the frontal region of the head due to periostitis of the suture lines between the nasal and frontal and more rarely the nasal, lacrimal and malar bones have been described [16,41,128,129]. They occur in many breeds but the incidence appears to be particularly high in Thoroughbreds and Thoroughbred crosses [25]. Although most are possibly traumatic in origin, including following nasofrontal osteotomy, the exact etiology of such lesions remains unknown in other cases. Cases

have presented with bilateral, firm, non-painful swellings, rostral to the eye, accompanied by epiphora in some cases (Fig. 24).

Figure 24. Symmetrical firm painless swellings at the level of the medial canthus are typical of a nasofrontal suture exostosis. - To view this image in full size go to the IVIS website at www.ivis.org . -

Differentiation from facial fractures and sinusitis is usually possible by clinical and radiological examination. Radiographs frequently demonstrate proliferative periosteal changes of the incompletely closed suture line. The swellings usually remodel and regress gradually without treatment over 1 - 2 years, but in some cases continued instability has resulted in progressive increases in the size of these swellings.

References

1. Robinson NE, Sorenson PR. Pathophysiology of airway obstruction in horses: a review. J Am Vet Med Assoc 1978; 172:299-303.
2. Sisson SB, Grossman JD. The nasal cavity. In: The anatomy of the domestic animals. Philadelphia and London: WB Saunders Co, 1953; 78-84.
3. Dixon PM. Nasal cavity. In: Equine respiratory endoscopy. Boehringer Ingelheim, Bracknell, 1993; 22-29.
4. Taylor J. The horse. In: The head and neck. London: Oliver and Boyd, 1955; 133-135.
5. Sisson S, Grossman DJ. The respiratory system. In: The anatomy of the domestic animals. Philadelphia and London: WB Saunders Company, 1959; 517-558.
6. King AS, Riley VA. The skull. In: A guide to the physiological and clinical anatomy of the head, Liverpool, University of Liverpoool, 1980; 1.1-1.14.
7. Nickel R, Schummer A, Seiferle E. In: Eingewide, Berlin: Verlag Paul Parey, 1975.
8. Freeman DE. Paranasal sinuses. In: Beech J, ed. Equine respiratory disorders. Philadelphia: Lea and Febiger, 1991; 275-305.
9. Dyce KM, Sack WO, Wensing CJG. The respiratory apparatus. In: Textook of veterinary anatomy. Philadelphia, London: WB Saunders Co, 1989; 143.
10. Cook WR. Some observations on diseases of the ear, nose and throat in the horse, and endoscopy using a flexible fibreoptic endoscope. Vet Rec 1974; 94:533-541.
11. Dyce KM, Sack WO, Wensing CJC. The head and ventral neck of the horse. In: Textbook of veterinary anatomy, Philadelpia, London: WB Saunders Co, 1989; 462-486.
12. Budras K-D, Sack WO, Rock S. The head. In: Anatomy of the horse an illustrated text, London: Mosby-Wolfe, 1994; 28-41.
13. Pirie M, Pirie HM, Wright NG. (1990), A scanning electron-microscopic study of the equine upper respiratory-tract. Equine Vet J 1990; 22:333-337.
14. Tremaine WH, Clarke CJ, Dixon PM. Histopathological findings in equine sinonasal disorders. Equine Vet J 1999; 31:296-303.
15. Lane JG, Gibbs C, Meynik SE, et al. Radiographic examination of the facial, nasal and paranasal sinus regions of the horse: I indications and procedures in 235 cases. Equine Vet J 1987; 19:466-473.
16. Gibbs C, Lane JG. Radiographic examination of the nasal and paranasal sinus regions of the horse. Part 2: Radiological findings. Equine Vet J 1987; 19:474-482.
17. Ruggles AJ, Ross MW, Freeman DE. Endoscopic examination of normal paranasal sinuses in horses. Vet Surg 1991; 20:418-423.
18. Tremaine WH, Dixon PM. (2001), A long-term study of 277 cases of equine sinonasal disease. Part 1: details of horses, historical, clinical and ancillary diagnostic findings. Equine Vet J 2001; 33:274-282.
19. Weller R, Livesey L, Maierl J, et al. Comparison of radiography and scintigraphy in the diagnosis of dental disorders in the horse. Equine Vet J 2001; 33:49-58.
20. Boulton CH. Equine nasal cavity and paranasal sinus disease : A review of 85 cases. J Equine Vet Sci 1985; 5:268-275.
21. Goble DO, Geiser DR, Jones RD. Examination, diagnosis and treatment of equine upper respiratory disorders, Part 1. J Equine Med Surg 1979; 3:162-169.
22. Vandeplassche M, Simoens P, Roeters R, et al. Aetiology and pathogenesis of congenital torticollis and head scoliosis in the equine foetus. Equine Vet J 1984; 16:419-424.

23.   Valdez H, McMullan WC, Hobson HP, et al. Surgical correction of deviated nasal septum and premaxilla in a colt. J Am Vet Med Assoc 1978; 173:1001-1004.

24.   Freeman DE. Nasal passages. In: Beech J., ed. Equine respiratory disorders. Philadelphia, London: Lea and Febiger, 1991; 253-274.

25.   Dixon PM. Swellings of the head region in the horse. In Pract 1991; 257-263.

26.   Nickels FA, Tulleners EP. Nasal passages. In: Auer J, ed. Equine surgery. Philadelphia, London: WB Saunders Co, 1992; 433-446.

27.   Gordon LR. Cytology and histology of epidermal inclusion cysts in the horse. J Equine Med Surg 1978; 2:370-372.

28.   Dixon PM. Tracheostomy in the horse. In Pract 1988; 10:249-253.

29.   Gaughan EM. Surgery of the upper respiratory tract. In: Kobluk CN, Aanes TR, and Geor RJ, ed. The horse: Diseases and clinical management. Philadelphia, London: WB Saunders Co, 1995; 243-256.

30.   Robinson NE, Sorenson PR, Goble DO. Patterns of airflow in normal horses and horses with respiratory disease. Proc Am Assoc Equine Pract 1975; 21:11-21.

31.   Haynes PFSurgery of the equine respiratory tract. In: Jennings PB, ed. The practice of large animal surgery. Philadelphia: WB Saunders Co, 1984; 388-487.

32.   Hawkins JF, Tulleners EP, Evans LH, et al. Alar fold resection in horses: 24 cases (1979-1992). J Am Vet Med Assoc 1995; 206:1913-1916.

33.   van Andel ACG, Gruys E, Kroneman J. Amyloid in the horse: A report of nine cases. Equine Vet J 1988; 20:277-283.

34.   Shaw DP, Gunson DE, Evans LH. Nasal amyloidosis in four horses. Vet Path 1987; 24:183-185.

35.   Mould JRB, Munroe GA, Eckersall PD, et al. Conjunctival and nasal amyloidosis in a horse. Equine Vet J 1990; Suppl 10:8-11.

36.   Greet TRC. Nasal aspergillosis in three horses. Vet Rec 1981; 109:487-489.

37.   McGorum BC, Dixon PM, Lawson GHK. A review of ten cases of mycotic rhinitis. Equine Vet Educ 1992; 4:8-12.

38.   Brearley JC, McCandlish IAP, Sullivan M, et al. Nasal granuloma caused by *Pseudallescheria boydii*. Equine Vet J 1986; 18:151-153.

39.   Johnson GR, Schiefer B, Pantekoek JFCA. Maduromycosis in a horse in Western Canada, Can Vet J 1975; 16:341-344.

40.   Watt DA. A case of Cryptococcal granuloma in the nasal cavity of a horse. Austr Vet J 1970; 46: 493-494.

41.   Tremaine WH, Dixon PM. A long-term study of 277 cases of equine sinonasal disease. Part 2: treatments and results of treatments. Equine Vet J 2001; 33:283-289.

42.   Corrier DE, Wison SR, Scrutchfield WL. Equine cryptococcal rhinitis. Compend Contin Educ Pract Vet 1984; 6:556-558.

43.   DeMartini JC, Riddle WE. Disseminated Coccidiomycosis in two horses and a pony. J Am Vet Med Assoc 1969; 155:149-156.

44.   Reed SM, Boles CL, Dade AW, et al. Localised equine nasal coccidiomycosis granuloma. J Equine Med Surg 1979; 3:119-123.

45.   Hodgkin EC, Conaway DH, Ortenburger AI. Recurrence of obstructive nasal coccidioidal granuloma in a horse. J Am Vet Med Assoc 1984; 184:339-340.

46.   Myers DD, Simon J, Case MT. (1964), Rhinosporidiosis in a horse. J Am Vet Med Assoc 1964; 145:345-346.

47.   Bridges CH, Romane WM, Emmons CW. Pycomycosis of horses caused by Entomophthera coronata, J Am Vet Med Assoc 1962; 140:673-677.

48.   Humber RA, Brown CC, Korngay RW. Equine zygomycosis caused by Conidiobolus lamprauges. J Clin Micro 1989; 27:573-576.

49.   Hutchins DR, Johnston KG. Phycomycosis in the horse. Austr Vet J 1972; 48:269-277.

50.   McMullan WC, Joyce JR, Hanselka DV, et al. Amphotericin B for the treatment of localised subcutaneous phycomycosis in the horse. J Am Vet Med Assoc 1977; 170:1293-1297.

51.   Zamos DT, Schumacher J, Loy JK. Nasopharyngeal conidiobolomycosis in a horse. J Am Vet Med Assoc 1996; 208:100-101.

52.   Greet TRC. Differential diagnosis of nasal discharge. Equine Vet Educ 1992; 4:23-25.

53.   Servantie D, Sautet JY. Hamartoma of the nasal septum in a yearling. Equine Pract 1986; 8:11-15.

54.   Howie F, Munroe G, Thompson H, et al. Palatine squamous cell carcinoma involving the maxillary sinus in two horses. Equine Vet Educ 1992; 4:3-7.

55.   Tulleners EP, Raker C. Nasal Septum resection in the horse. Vet Surg 1983; 12:41-47.

56.   Hardy J. Upper respiratory obstruction in foals, weanlings, and yearlings. Vet Clin North Am Equine Pract 1991; 7:105-122.

57.   Joyce J, Pierce K, Romane W. Clinical study of nutritional secondary hyperparathyroidism in horses. J Am Vet Med Assoc 1971; 158:2033-2042.

58. Schryver H, Hintz H, Lowe J. Calcium and phosphorus in the nutrition of the horse. Cornell Vet 1974; 64:494-515.
59. Clarke CJ, Roeder PL, Dixon PM. Nasal obstruction caused by nutritional osteodystrophia fibrosa in a group of Ethiopian Horses. Vet Rec 1996; 139:568-570.
60. Mason BJE. Empyema of the equine paranasal sinuses. J Am Vet Med Assoc 1975; 167:727-731.
61. van der Velden MA, Verzijlenberg K. Chronic purulent maxillary sinusitis in horses. Tijdschr Diergeneeskd 1984; 109:793-799.
62. Scott EA Sinusitis. In: Robinson NE, ed. Current Therapy in Equine Medicine 2. Philadelphia: WB Saunders Co, 1987; 605-607.
63. Mansmann RA, Wheat JD. The diagnosis and treatment of equine upper respiratory diseases. Proc Am Assoc Equine Pract 1973;18:388-487.
64. Coumbe KM, Jones RD, Kenward JH. Bilateral sinus empyema in a six year-old mare. Equine Vet J 1987; 19:559-560.
65. Lane JG. Management of sinus disorders, part 1. Equine Vet Educ 1993; 5:5-9.
66. Schumacher J, Honnas C, Smith B. Paranasal sinusitis complicated by inspissated exudate in the ventral conchal sinus. Vet Surg 1987; 16:373-377.
67. Ruggles AJ, Ross MW, Freeman DE. Endoscopic examination and treatment of paranasal sinus disease in 16 horses. Vet Surg 1993; 22:508-514.
68. Schumacher J, Crossland LE. Removal of inspissated purulent exudate from the ventral conchal sinus of three standing horses. J Am Vet Med Assoc 1994; 205:1312-1314.
69. Dixon PM, Tremaine WH, Pickles K, et al. Equine dental disease part 4: a long-term study of 400 cases: apical infections of cheek teeth. Equine Vet J 2000; 32:182-194.
70. Lane JG. A review of dental disorders of the horse, their treatment and possible fresh approaches to management. Equine Vet Educ 1994; 6:13-21.
71. Mackintosh ME, Colles CM. Anaerobic bacteria associated with abscesses in the horse and donkey. Equine Vet J 1987; 19:360-362.
72. Freeman DE, Orsini PG, Ross MW, et al. A large frontonasal flap for sinus surgery in the horse. Vet Surg 1990; 19:122-130.
73. Scrutchfield WL, Schumacher J, Walker M, et al. Removal of an osteoma from the paranasal sinuses of a standing horse. Equine Pract 1994; 16:24-27.
74. Leyland A, Baker JR. Lesions of the nasal and paranasal sinuses of the horse causing dyspnoea. Br Vet J 1975; 131:339-346.
75. Dixon PM. Equine maxillary cysts. Equine Pract 1985; 7:25-33.
76. Lane JG, Longstaffe JA, Gibbs C. Equine paranasal sinus cysts: a report of 15 cases. Equine Vet J 1987; 19:534-544.
77. Sanders-Shamis M, Robertson JT. Congenital sinus cyst in a foal. J Am Vet Med Assoc 1987; 190:1011-1012.
78. Beard WL, Robertson JT, Leeth B. Bilateral congenital cysts in the frontal sinus of a horse. J Am Vet Med Assoc 1990; 196:453-454.
79. Gunn HM. Histochemical observations on laryngeal skeletal muscle fibres in "normal" horses. Equine Vet J 1972; 4:144-148.
80. Caron JP. Diseases of the nasal cavity and paranasal sinuses. In: Colahan PT, Mayhew IG, Merritt AM, and Moore JN, eds. Equine Medicine and Surgery. Goleta, Ca: American Veterinary Publications, 1991; 386-397.
81. O'Connor JJ. Operations. In: Dollar'sVeterinary Surgery. London: Baillere, Tindall and Cox, 1930; 222-231.
82. Cook WR, Littlewort MCG. Progressive haematoma of the ethmoid region in the horse. Equine Vet J 1974; 6:101-108.
83. Platt H. Haemorrhagic nasal polyps of the horse. J Path 1975; 115:51-55.
84. Hanselka DV, Young MF. Ethmoidal hematoma in the horse. Vet Med Small Anim Clin 1975; 70:1289-1291.
85. Etherington WG, Vasey JR, Horney FD. Ethmoid hematoma of the equine. Can Vet J 1982; 23:231-234.
86. Specht TE, Colahan PT, Nixon AJ, et al. Ethmoidal hematoma in nine horses. J Am Vet Med Assoc 1990; 197:613-616.
87. Greet TRC. Outcome of treatment in 23 horses with progressive ethmoidal haematoma. Equine Vet J 1992; 24:468-471.
88. Bell BTL, Baker GJ, Foreman JH. Ethmoid hematoma in horses: characteristics, cause and treatement. Compend Contin Educ Pract Vet 1993; 15:1391-1399.
89. Sullivan M, Burrell MH, McCaudlish IAP. Progressive haematoma of the maxillary sinus in a horse. Vet Rec 1984; 114:191-192.
90. Rothhaug PG, Tulleners EP. ND-YAG laser treatment of 25 progressive ethmoid haematomas and other masses of ethmoidal origin 1986-1995. Vet Surg 1996; 437.
91. Schumacher J, Yarbrough T, Pascoe J. Transendoscopic chemical ablation of progressive ethmoidal hematomas in standing horses. Vet Surg 1998; 27:175-181.
92. Frees KE, Gaughan EM, Lillich JD, et al. Severe complication after administration of formalin for treatment of progressive ethmoidal hematoma in a horse. J Am Vet Med Assoc 2001; 219:950-952, 939.

93. Cotchin E. Neoplasms of the domesticated animals, Common Wealth Agricultural Bureaux, England, 1956; 17.

94. Madewell BR, Priester WA, Gillete EL, et al. Neoplasms of the nasal passages and paranasal sinuses in domestic animals as reported by 13 Veterinary Colleges. Am J Vet Res 1976; 37:851-856.

95. Sundbergh JP, Burustein T, Page EH, et al. Neoplasms of Equidae. J Am Vet Med Assoc 1977; 170:150-152.

96. Priester WA, Mackay FW. The occurrence of tumors in domestic animals. Natl Cancer Inst Monogr 1980; 54:59-99.

97. Cotchin E. Spontaneous neoplasms of the upper respiratory tract in animals. In: Muir CS, Shanmugaratnam K, eds. Cancer of the naso-pharynx, New York: Medical Examination Publishing Co., Flushing, 1967; 203-259.

98. Stunzi H, Hauser B. Tumours of the nasal cavity (International histological classification of tumours of domestic animals). Bull World Health Org 1976; 53:257-263.

99. Hilbert BJ, Little CB, Klein K, et al. Tumours of the paranasal sinuses in 16 horses. Austr vet J 1988; 65:86-88.

100. Head KW, Dixon PM. Equine nasal and paranasal sinus tumours. Part 1: review of the literature and tumour classification. Vet J 1999; 157:261-278.

101. Dixon PM, Head KW. Equine nasal and paranasal sinus tumours: part 2: a contribution of 28 case reports. Vet J. 1999; 157:279-294.

102. Riegg A. Squamous cell carcinoma involving the facial sinuses and air passages in a horse. Vet Med 1946; 41:142-143.

103. Jones DL. Squamous cell carcinoma of the larynx and pharynx in horses. Cornell.Vet 1994; 84:15-24.

104. Acland HM, Orsini JA, Etkins S, et al. Congenital ethmoid carcinoma in a foal. J Am Vet Med Assoc 1984; 184:979-981.

105. Reynolds BL, Stedham MA, Lawrence JM, et al. Adenocarcinoma of the frontal sinus with extension to the brain in a horse. J Am Vet Med Assoc 1979; 174:734-736.

106. Hill FWE, Moulton JE, Schiff PH. Exophthalmos in a horse resulting from an adenocarcinoma of the frontal sinus. J S Afr Vet Assoc 1989; 60:104-105.

107. Mason BJE. Spindle-cell sarcoma of the equine para-nasal sinuses and nasal chamber. Vet Rec 1975; 96:287-288.

108. Richardson JD, Lane JG, Nicholls PK. Nasopharyngeal mast cell tumour in a horse. Vet Rec 1994; 134: 238-240.

109. Malikides N, Reppas G, Hodgson JL, et al. Mast cell tumours in the horse: four case reports. Equine Pract 1996; 18:12-17.

110. Meschter CL, Allen D. Lymphosarcoma within the nasal cavities of an 18 month old filly. Equine Vet J 1984; 16:475-476.

111. Lane JG. Palatine lymphosarcoma in two horses. Equine Vet J 1985; 17:465-467.

112. Adams R, Calderwood-Mays MB, Peyton LC. Malignant lymphoma in three horses with ulcerative pharyngitis. J Am Vet Med Assoc 1988; 193:674-676.

113. Gorlin RJ, Meskin LH, Brodey R. Odontogenic tumours in man and animals. pathological classification and clinical behaviour- a review. Ann NY Acad Sci 1963; 108:722-771.

114. Schumacher J, Smith BL, Morgan SJ. Osteoma of paranasal sinuses of a horse. J Am Vet Med Assoc 1988; 192:1449-1450.

115. Adair HS, Duncan RB, Toal RL. Solitary osteochondroma of the nasal bone in a horse. Cornell Vet 1994; 84:25-31.

116. Barber SM, Clark EG, Fretz PB. Fibroblastic tumour of the premaxilla in two horses. J Am Vet Med Assoc 1983; 182:700-702.

117. Hultgren BD, Schmotzer WB, Watrous BJ, et al. Nasal - maxillary fibrosarcoma in young horses: a light microscopic study. Vet Path 1987; 24:194-196.

118. Pirie RS, Dixon PM. Mandibular tumours in the horse: a review of the literature and 7 case reports. Equine Vet Educ 1993; 5:287-294.

119. Head KW, Dixon PM. Equine nasal and paranasal sinus tumours. Part 1: review of the literature and tumour classification. Vet J 1999; 157:261-278.

120. Roberts MC, Groenendyk S, Kelly WR. Ameloblastic odontoma in a foal. Equine Vet J 1978; 10:91-93.

121. Walker MA, Schumacher J, Schmitz DG, et al. Cobalt 60 radiotherapy for treatment of squamous cell carcinoma of the nasal cavity and paranasal sinuses in three horses. J Am Vet Med Assoc 1998; 212:848-851.

122. Hardy J. Upper respiratory obstruction in foals, weanlings, and yearlings. Vet Clin North Am Equine Pract 1991; 7:105-122.

123. Sullins KE, Turner AS. Management of fractures of the equine mandible and premaxilla (Incisive bone). Compend Contin Educ Pract Vet 1982; 4:S480-S489.

124. Denny HR. Traumatic conditions of the head. In: Proceedings of 23rd Annual Congress of the British Equine Veterinary Association, 1984; 52-53.

125. Dixon PM. Nasal cavity. In: Equine Respiratory Endoscopy, Boehringer Ingelheim, Bracknell 1993; 22-29.

126. Lane JG. The management of sinus disorders: Part 2. Equine Vet Educ 1993; 5:69-73.

127. Valdez H, Rook JS. Use of fluorocarbon polymer and carbon fiber for restoration of facial contour in a horse. J Am Vet

Med Assoc 1981; 178:249-251.

128. Speirs VC. Diseases of the paranasal sinuses. In: Robinson NE, ed. Current Therapy in Equine Medicine 3. Philadelphia, London: WB Saunders Co, 1992; 271-274.

129. Trotter GW. Paranasal sinuses. Vet Clin North Am Equine Pract 1993; 9:153-169.

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