

Tutorial Article

Epistaxis in the horse

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Introduction

Epistaxis is a clinical sign which occurs with a wide range of disorders of the equine upper and lower respiratory tract. Determination of the source and cause of the haemorrhage is essential, in order to differentiate potentially life-threatening conditions (e.g. arterial haemorrhage due to guttural pouch mycosis) from those which are likely to be self-limiting (e.g. mild sinonasal trauma). A full history, along with thorough clinical and endoscopic examination, will usually give the clinician enough information to diagnose the cause of epistaxis. In some cases, however, radiography, sinuscopy, lesional biopsy, examination of tracheal secretions or bronchoalveolar lavage fluid (BALF), computed tomography or magnetic resonance imaging may also be required in order to make a definitive diagnosis.

This article describes the methods of investigation and potential causes of epistaxis in the horse.

Investigation of horses with epistaxis

History

The **duration** of epistaxis, whether it is unilateral or bilateral, and an **estimate of the volume of blood** lost are the most important features of the history to help ascertain whether the case constitutes an emergency. Additionally, the nature of the blood, i.e. if fresh/bright red or old/dark/brown, may give some clue as to the nature and duration of the causal lesion. In the absence of evidence of head trauma, profuse epistaxis at rest most commonly occurs due to guttural pouch mycosis, while epistaxis during or after fast exercise is usually attributable to exercise-induced pulmonary haemorrhage (EIPH). A history of epistaxis with sudden development of facial swelling, particularly if associated with an open wound, is highly suspicious of trauma.

The **presence and nature** (e.g. mucopurulent or purulent, unilateral or bilateral) of other types of nasal discharge occurring between episodes of epistaxis should also be recorded. The presence of intercurrent neurological signs of dysphagia or Horner's syndrome are indicative of guttural pouch mycosis.

Clinical signs

If the owner estimates that the horse may have lost a substantial amount of blood (e.g. >4–5 l in a 500 kg horse), the horse should first be examined to ascertain its **cardiovascular status**. Pale mucous membranes, tachycardia, weak peripheral pulses and generalised weakness are all indicative of hypovolaemic shock, and appropriate emergency therapy should be instituted. The packed cell volume (PCV) may not decrease until 24 h after an episode of haemorrhage, and this parameter is therefore not reliable for assessment of acute hypovolaemia due to blood loss.

The nature and amount of epistaxis should be noted, and whether it is **unilateral or bilateral**. Consistently unilateral epistaxis indicates sinonasal haemorrhage, whereas significant guttural pouch or pulmonary haemorrhage will usually be bilateral. The submandibular lymph nodes should also be palpated for evidence of unilateral or bilateral enlargement. **Airflow at each nostril** should be checked to assess presence and degree of respiratory obstruction (usually originating within the nasal cavity). This is easily performed by holding a small piece of cotton wool in front of each nostril in turn, and observing the amount of movement generated by airflow. The head should be inspected for evidence of skin damage or asymmetry due to swelling or trauma. Subcutaneous emphysema may be detected if the integrity of the sinonasal cavities has been disrupted. **Swellings** of the parotid area may be due to guttural pouch distension, or abscessation of lymph nodes in the parotid region.

An assessment of cranial nerve function should be made if guttural pouch mycosis is suspected (**Table 1**). Facial paralysis may also rarely be associated with epistaxis if there has been traumatic injury to both the facial nerve and the sinonasal area. More severe cases of skull trauma (e.g. if the horse rears and falls over backwards) may exhibit clinical signs consistent with damage to the central nervous system.

Endoscopy

Respiratory endoscopy is the key diagnostic technique for ascertaining the source of haemorrhage in cases of epistaxis. A small diameter (8–10 mm), 1 m long endoscope

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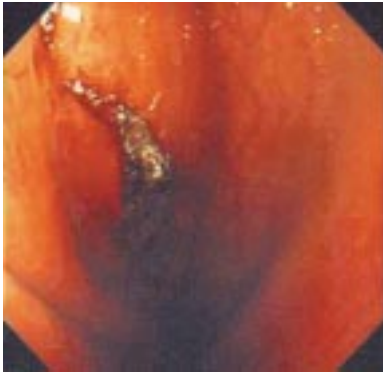


Fig 1: Endoscopic view of the dorsal rostral nasal cavity of a horse, which presented with unilateral epistaxis and a depression fracture of the nasal bones following trauma. Note the disruption of the mucosa of the dorsal aspect of the nasal cavity which is the source of haemorrhage.

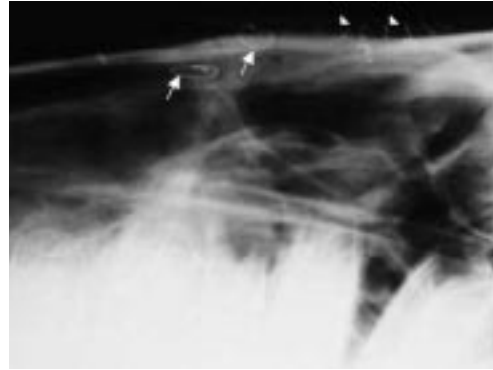


Fig 4: 30° dorsolateral-lateral oblique view of the sinuses of the horse in Figure 3. Under general anaesthesia, depression fractures of the skull have been elevated and fixed in place with cerclage wire (arrows). The skin staples are also visible (arrowheads).



Fig 2: Endoscopic view of the caudal aspect of the right middle meatus of a horse which has sustained trauma to its right maxillary sinuses. Fresh blood can be seen emanating from the sinus drainage angle.



Fig 5: Endoscopic view of the right ethmoturbinate area showing a PEH with a characteristically green/purple coloured capsule. This horse presented with a history of chronic, intermittent, low-grade unilateral epistaxis.



Fig 3: Lateral radiograph of the paranasal sinuses of a horse which sustained marked trauma to its frontal and maxillary bones. Note the fluid lines (arrows) present in the conchofrontal and caudal maxillary sinuses due to intrasinus haemorrhage.

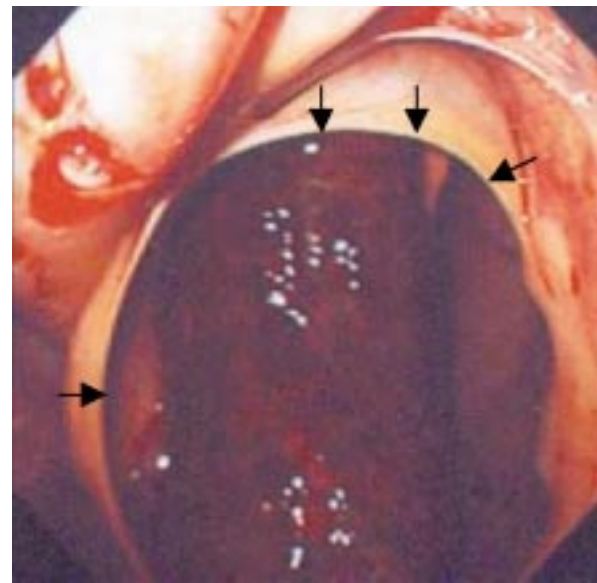


Fig 6: Endoscopic view of a large PEH within the left maxillary sinus, as viewed by sinoscopy via a trephine hole in the frontal sinus. Note the sharply demarcated border of the frontomaxillary opening (arrows).

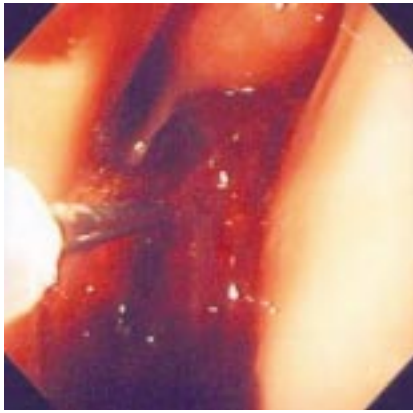


Fig 7: Endoscopic image showing transendoscopic, intralesional injection of a PEH with formalin. Note the collapsed appearance of the PEH, with formalin leaking from its capsule.

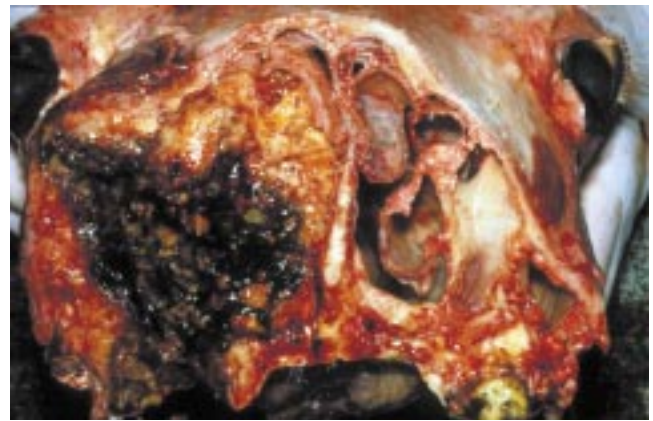


Fig 10: Transverse section through the skull of a horse with a large invasive sinonasal squamous cell carcinoma which has completely filled the right nasal cavity and paranasal sinuses. Note also the involvement of the hard palate.

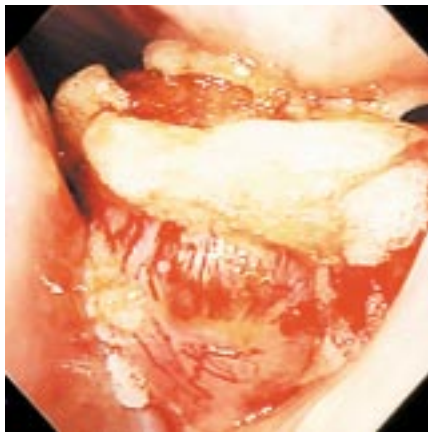


Fig 8: The ventral meatus of this aged horse, which presented with intermittent unilateral epistaxis and nasal discharge, is almost completely obstructed by a soft tissue mass. The mass was biopsied transendoscopically and found to be a lymphoma.

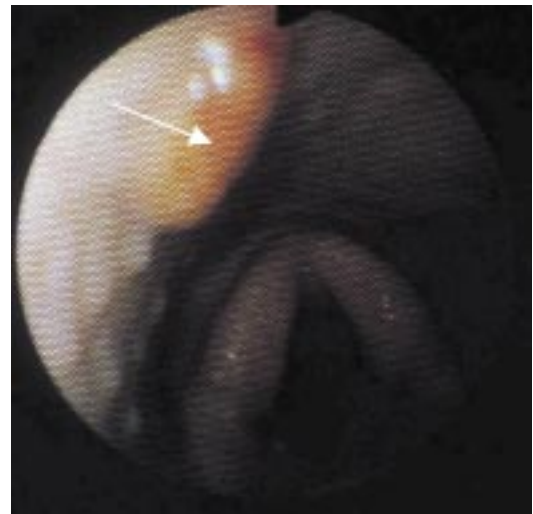


Fig 11: Endoscopic view of the nasopharynx of a horse showing a mass protruding from the right dorsolateral wall of the pharynx (arrow). A biopsy revealed this mass to be a lymphoma.

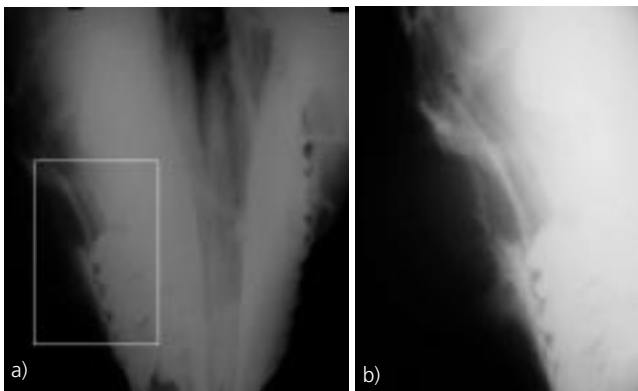


Fig 9: a) Dorsoventral radiograph and b) enlargement of boxed area from (a) of a 15-year-old mare which presented with intermittent, unilateral epistaxis and marked soft tissue facial swelling at the site of a previously extracted cheek tooth. Note the area of bony destruction of the maxilla surrounded by soft tissue swelling, which is highly suggestive of neoplasia. Histopathology of a biopsy of the mass revealed it to be a squamous cell carcinoma.



Fig 12: Endoscopic view of the middle meatus of a horse which had unilateral mucopurulent discharge and 2 episodes of mild epistaxis. Fungal plaques and fibrin can be seen. The mucosa underneath these plaques was deeply ulcerated, and was the source of the epistaxis.

TABLE 1: Clinical signs of neuropathies seen in horses with GPM

Cranial nerve	Clinical/endoscopic signs of neuropathy
VII (Facial)	Facial asymmetry due to paralysis of muscles of ear, eye and muzzle
IX (Glossopharyngeal)	Pharyngeal hemiplegia, pharyngeal dysphagia
Pharyngeal branch of X (Vagus)	Pharyngeal hemiplegia, pharyngeal dysphagia, dorsal displacement of the soft palate
Recurrent laryngeal branch of X	Ipsilateral laryngeal hemiplegia
XI (Accessory)	Pharyngeal hemiplegia, pharyngeal dysphagia
XII (Hypoglossal)	Reduced tongue tone (may be difficult to assess if unilateral)
Cranial sympathetic nerve	Horner's syndrome: ptosis, hemifacial sweating (in warm climates), miosis, enophthalmos

(e.g. human gastroscope) is most useful for examining the upper respiratory tract. In suspected cases of EIPH where blood may not be evident in the upper trachea, a longer (circa 1.7 m) endoscope (e.g. 12 mm diameter human colonoscope) may be required in order to inspect the carina and mainstem bronchi, to collect tracheal respiratory secretions and to perform bronchoalveolar lavage.

The ventral, middle and dorsal nasal meati should all be examined for evidence of trauma, mycotic lesions, abnormal exudate and intra- or extraluminal narrowing (due to trauma or sinonasal lesions). The sinonasal 'drainage angle' at the caudal aspect of the middle meatus should be carefully examined for presence of blood or haemorrhagic exudate, indicating intrasinus haemorrhage. The adjacent ethmoidal labyrinth should be inspected for the presence of progressive ethmoidal haematomas (PEH), or direct haemorrhage from this area.

As the endoscope is advanced into the nasopharynx, the **ostia of the guttural pouches** should be inspected for evidence of haemorrhagic or mucopurulent discharge. Haemorrhage or discharges which are present in the nasopharynx may originate from the lungs, and a close endoscopic inspection of the caudal nasopharynx and the trachea should allow differentiation of pulmonary disease from guttural pouch disease. A flexible biopsy instrument or guide wire inserted caudodorsally for a length of 10 cm through the dorsal part of the ostium facilitates passage of the endoscope into the auditory tube and guttural pouch. **Each guttural pouch should be carefully inspected** for a possible source of epistaxis, e.g. mycotic plaques overlying blood vessels or rupture of the *rectus capitis/longus capitis* muscles. The floors of the guttural pouches should be inspected for pooling of blood or other discharges, enlarged lymph nodes and tumours.

Nasopharyngeal and laryngeal disorders are very rarely associated with epistaxis, but these areas should be inspected for evidence of neurological deficits which may be secondary to guttural pouch mycosis (GPM) and for other lesions such as trauma from foreign bodies, or tumours. **In cases of EIPH**, blood can often be seen emanating from the larynx, and the

stream of blood can often be traced distally into the trachea (accumulating in the rostral aspect of the thoracic trachea ['sump']) and the mainstem bronchi.

Radiography

Radiography is a useful tool in cases of equine epistaxis for the **diagnosis of traumatic skull fractures**, detection of fluid or soft tissue densities within the sinuses or guttural pouches and avulsion fractures of the basisphenoid and occipital bones (associated with *rectus capitis/longus capitis* rupture). It is particularly useful for evaluation of cases where the nasal cavity is obstructed, preventing passage of an endoscope.

Standard views of the skull include straight lateral, 30° dorsolateral-lateral obliques and dorsoventral views. If skull fractures are suspected, oblique views taken at varying angles may be necessary to highlight the fracture lines; however, due to the complexity of bones within the skull, and particularly within the paranasal sinuses, nondisplaced fractures are difficult to detect radiographically. Superimposition of soft tissue densities within the sinuses due to intrasinus haemorrhage and mucosal inflammation may further obscure fracture lines.

Sinoscopy

Direct sinus endoscopy is a simple and inexpensive technique which is indicated in cases of epistaxis where blood or discharge is seen emanating from the sinus drainage angle. One study showed that sinoscopy provided diagnostic information in 70% of cases of sinus disorders (Tremaine and Dixon 2001a). A trephine hole into the frontal sinus, directly over the frontomaxillary aperture, provides a useful endoscopic portal in the majority of cases. This approach allows inspection of the frontal, dorsal conchal, caudal maxillary and entrances to the sphenoidal and palatine sinuses. If only the rostral maxillary and/or ventral conchal sinuses are involved, sinoscopy of these sinuses is usually unrewarding. However, lesions which may cause epistaxis (e.g. trauma, PEH) are rarely confined to the rostral maxillary sinus and ventral conchal sinus. In horses under age 7 years, the reserve crowns of the cheek teeth can almost completely fill the rostral maxillary sinus, thereby increasing the risk of iatrogenic damage to the teeth while making the trephine hole, and markedly reducing the 'space' within the rostral maxillary sinus, consequently making sinoscopy almost impossible. Even in the older horse with shorter cheek teeth reserve crowns, the rostral maxillary sinus often remains a small structure which limits the endoscopic field of view.

Other imaging modalities

Computed tomography (CT) (Tietje *et al.* 1996) or **magnetic resonance imaging (MRI)** (Arencibia *et al.* 2000) are very useful for evaluating the complex, 3-dimensional structures of the equine head. Although these modalities are presently restricted to a limited number of veterinary referral centres, their use is increasing in equine clinical work. Another

major limitation of CT and MRI is the requirement for general anaesthesia. Osseous swellings, upper respiratory tract growths and dental disorders are ideal applications for CT scanning (Tucker and Farrell 2001). MRI excels in the evaluation of soft tissues and is the optimal imaging modality for neurological disorders of the head in humans and small animals (Tucker and Farrell 2001).

Scintigraphy is becoming a widely available imaging modality, and although its use for the diagnosis of equine dental disease has been established (Weller *et al.* 2001), there are few studies evaluating its use for other equine sinonasal disorders which may cause epistaxis, such as PEH and neoplasia. Scintigraphy has also been used experimentally to detect and quantify exercise-induced pulmonary haemorrhage (Votion *et al.* 1999).

Causes of equine epistaxis

Facial and sinonasal trauma

Kicks, falls, injuries from inanimate objects or iatrogenic damage by stomach tubes or endoscopes may all result in trauma to the lining of the sinuses or nasal cavity. Due to the highly vascular nature of these structures, such injuries commonly result in epistaxis, with 65% of horses suffering from sinonasal trauma presenting with a sanguinous nasal discharge (Tremaine and Dixon 2001a) which may persist intermittently for up to 1 month (Dixon 1993). If the nasal cavity is affected, it is often possible to see disruption of the nasal mucosa endoscopically (**Fig 1**). If the sinus mucosa has been disrupted and intrasinus haemorrhage results, blood usually emanates from the 'drainage angle' in the middle meatus (**Fig 2**). In 30% of cases of sinonasal trauma, no blood may be seen at the ostia at the time of endoscopy (Tremaine and Dixon 2001a), but straight lateral skull radiographs may reveal fluid lines within the sinus cavities (**Fig 3**) and possible fracture lines which, combined with the history of epistaxis, is suggestive of intrasinus haemorrhage.

In the majority of cases, the degree of trauma is usually not severe enough to warrant surgical intervention to provide haemostasis, maintain a patent airway, or drain the sinuses, as most cases will self-resolve within a few weeks. Rarely, neurological signs occur due to concurrent central nervous system (CNS) damage. Depression fractures of the maxilla and nasal bone are common, and large displaced fragments should be elevated and fixed in place with cerclage wire if an optimal cosmetic result is desired (**Fig 4**), especially if the fracture is open. During surgery, small loose bone fragments should be removed and a catheter implanted (usually into the frontal sinus) to lavage blood clots and debris from the sinuses post operatively.

Progressive ethmoidal haematoma

Progressive ethmoidal haematomas (PEHs) are non-neoplastic masses of unknown aetiology, which are aptly described as '**haemorrhagic polyps**' (Head and Dixon 1999). They consist of sinusoidal, blood-filled cavities lined with respiratory mucosa, within a loose trabecular network of fibrous tissue

which is encapsulated by normal respiratory epithelium (Schumacher *et al.* 1998; Tremaine *et al.* 1999). They most commonly originate in the ethmoidal labyrinth and grow rostrally into the caudal nasal cavity. They may also originate on the lateral aspect of the ethmoturbinates and expand into the maxillary sinuses, from the dorsal aspects of the ethmoturbinates with expansion into the conchofrontal sinus, or from the ventral ethmoturbinates, expanding into the sphenopalatine sinus (Tremaine *et al.* 1999).

The most common clinical presentation of PEH is of chronic, intermittent, low grade, unilateral, dark coloured, serosanguinous nasal discharge, which may be mucopurulent at times. Other less common clinical signs include abnormal respiratory noises, dyspnoea and, rarely, distortion of the facial bones, or CNS signs. Endoscopy is the most useful imaging modality for PEHs which originate within the nasal cavity (**Fig 5**), and sinuscopy may reveal intrasinus PEHs (**Fig 6**). Discrete lesions may be visible on lateral skull radiographs; however, radiographic interpretation of the ethmoidal area is difficult due to superimposition of lesions over the globes, orbits and ethmoidal labyrinths, and may result in small PEH lesions being missed.

Laing and Hutchins (1992) reported that 3 of 10 untreated cases of PEH required euthanasia 6 to 24 months following diagnosis. Traditionally, **surgical excision** via a nasofrontal or maxillary bone flap has been used for treatment of the disorder, but recurrence has been reported in up to 38% of cases (Tremaine and Dixon 2001b). More recently, **intralesional injection of 4% formaldehyde solution** has been shown to be a very successful technique which can be performed in the standing sedated horse (Schumacher *et al.* 1998; Tremaine and Dixon 2001b) (**Fig 7**), although more than one injection is usually required for complete regression of large lesions. **Development of neurological signs** necessitating euthanasia has been reported following intralesional injection of formaldehyde in a horse where a PEH had eroded the cribriform plate (Frees *et al.* 2001). Transendoscopic photovaporisation of PEHs using a Nd:YAG laser has also been described (Tate 1991).

It must be remembered that, although treatment of PEH may cause long-term remission of clinical signs, recurrence of pre-existing lesions or development of new lesions (on the ipsilateral or contralateral side) is common, whichever method of treatment is employed.

Neoplasia of the upper respiratory tract

Neoplasia of the equine respiratory tract is relatively uncommon but should be considered as a cause of epistaxis, especially in the older horse presenting with facial swelling and purulent nasal discharge. Accurate diagnosis of respiratory neoplasms is important, because most cases are associated with a grave prognosis, in contrast with the more common non-neoplastic growths of this area (Tremaine and Dixon 2001b).

The most frequently occurring tumour of the equine sinonasal area is squamous cell carcinoma (Priester and Mackay 1980; Head and Dixon 1999). Although epithelial

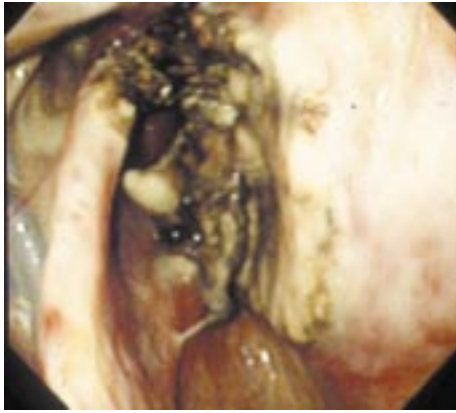


Fig 13: Endoscopic view of the right guttural pouch showing a fungal plaque situated on the dorsomedial aspect, overlying the internal carotid artery and 9th, 10th, 11th and 12th cranial nerves.

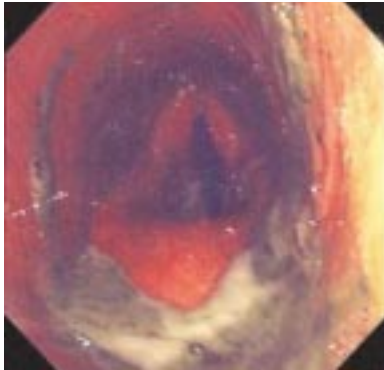


Fig 14: Endoscopic view of the nasopharynx of a pony suffering from GPM with neurological damage, showing pharyngeal pooling of saliva and food material and left-sided laryngeal paralysis.

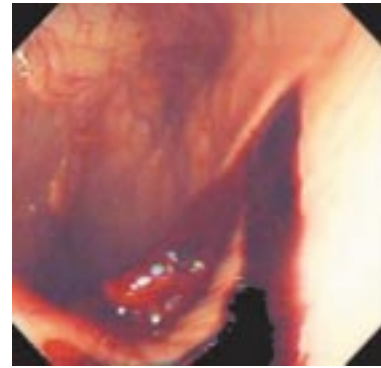


Fig 15: Endoscopic view of the nasopharynx of a horse which suffered a episode of profuse epistaxis a few hours previously. Note the stream of blood, and a blood clot emanating from the left guttural pouch ostium.

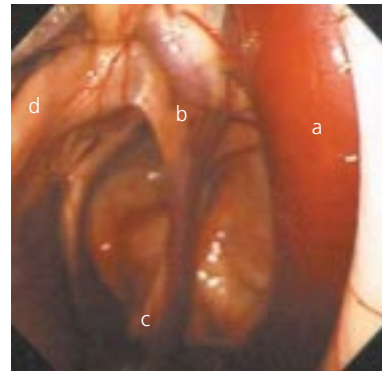


Fig 16: Endoscopic view of the medial compartment of the right guttural pouch, showing the longus capitus muscle (a) on its medial aspect. The internal carotid artery (b), which is closely associated with cranial nerves IX, X, XI and XII (c), and the dorsal aspect of the stylohyoid bone (d), are also visible.

tumours (such as squamous cell carcinoma) are more common in older horses, fibro-osseous tumours of this area occur most commonly in young horses (mean age 4 years; Dixon and Head 1999). Clinical signs depend on the exact location and type of neoplasm, but commonly include facial deformity (81%), nasal discharge (77%), respiratory obstruction (55%) and epistaxis (22%); dysphagia and neurological signs occur less often (Tremaine and Dixon 2001a). This is in contrast to canine sinonasal neoplasia, where epistaxis is present in most affected animals. Systemic signs such as anorexia and weight loss may also be seen in advanced cases.

Endoscopy (**Fig 8**) and radiography are useful to define the location and extent of these lesions, and radiographs may show additional changes which are more suggestive of neoplasia, such as bone lysis caused by aggressive tumours (**Fig 9**). If neoplasia is suspected, the mucosa of the hard palate should be carefully inspected *per os*, as this is a common site from which sinonasal squamous cell carcinomas originate (**Fig 10**). **A definitive diagnosis may be made** only by biopsy and histopathology of the lesion. Endoscopic biopsies are noninvasive, but are frequently misleading, because they often reveal only secondary

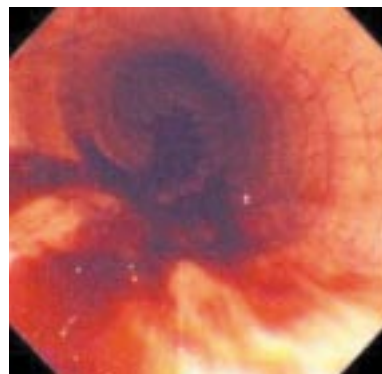


Fig 17: Endoscopic view of the distal trachea of a horse which was pulled up during a race and subsequently showed bilateral epistaxis. A stream of blood, which is less than half the tracheal width (Grade 3 EIPH), is present.

pathological processes and surface changes rather than the deeper underlying tumour parenchyma (Scarratt and Crisman 1998; Head and Dixon 1999). Larger incisional or excisional biopsies obtained via a trephine hole or sinonasal bone flap are most useful.

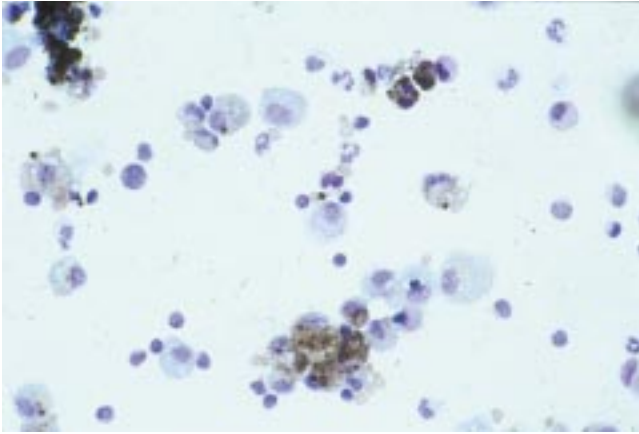


Fig 18: Cytological preparation of bronchoalveolar lavage fluid taken from a horse which was presented with poor racing performance and intermittent epistaxis after exercise. Note the presence of both free erythrocytes, indicative of recent haemorrhage, and (brown-staining) haemosiderophages, indicative of previous haemorrhage, which may have occurred during the last 6 months (Leishmann's, x400).

Treatment of sinonasal tumours most commonly involves surgical excision, but cryotherapy, immunotherapy and radiation therapy have also been reported (House *et al.* 1976; Theon 1998). The prognosis depends largely on the nature of the neoplasm, but the aggressive nature of equine epithelial sinonasal tumours, along with limited surgical exposure and difficulty in obtaining clean tumour-free margins, allowed sinonasal tumour regrowth in 69% of cases within 6 months (Dixon and Head 1999).

Lymphosarcoma (lymphoma) is the most frequently occurring tumour (**Fig 11**) in the equine nasopharyngeal and laryngeal areas. Clinical signs are often attributable to dysphagia and respiratory obstruction and include nasal discharge, dyspnoea and abnormal respiratory noises. Often, secondary bacterial infection and/or tissue necrosis results in a bilateral nasal discharge which may contain blood. Surgical and laser (Jones 1994) excision of pharyngeal/laryngeal tumours has been attempted, but these neoplasms are often diffuse, aggressive and tend to metastasise locally, resulting in a poor prognosis.

Foreign bodies

Foreign bodies are rarely inhaled or swallowed in horses, but damage to the mucosa of the upper respiratory tract from inhaled foreign bodies may result in epistaxis, which is usually transient.

Other disorders of the sinonasal area

Sinonasal infection with *Aspergillus fumigatus* is rare in the horse, and is usually secondary to other sinonasal disorders, e.g. ethmoidal haematoma, or occurs following sinonasal surgery. A **malodorous, unilateral or bilateral, purulent to mucopurulent discharge results**, and epistaxis is

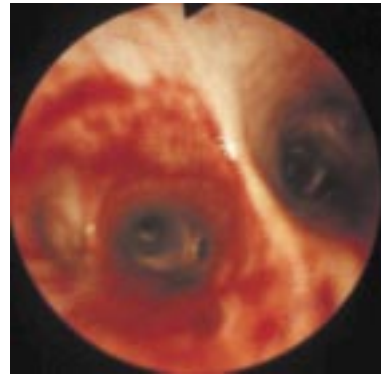


Fig 19: Endoscopic view of the distal trachea of a horse suffering from acute haemorrhagic necrotising pneumonia. Blood is emanating from both primary bronchi, making the endoscopic appearance similar to that of EIPH (however, the clinical presentation is quite different).

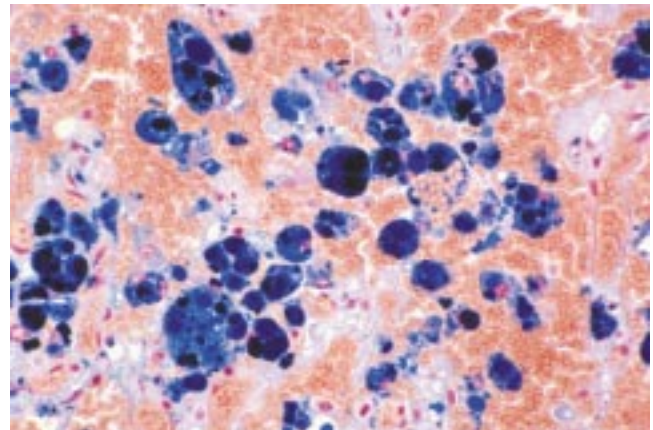


Fig 20: Histopathological section of the lung of a horse with acute necrotising haemorrhagic pneumonia showing erythrocytes completely filling the alveoli, large amounts of haemosiderin (blue-staining) within macrophages and a marked influx of white blood cells (Prussian Blue, x400).

reported to occur in 8% of horses (McGorum and Dixon 1992; Tremaine and Dixon 2001a). **Fungal plaques** may be seen in the nasal cavity (**Fig 12**) or within the sinuses. When aspergillosis is a secondary disease, treatment of the primary condition will often result in resolution of the fungal infection. Cases of primary aspergillosis respond well to topical antifungal treatment (nystatin or natamycin) (Greet 1981; McGorum and Dixon 1992; Tremaine and Dixon 2001b).

In the study by Tremaine and Dixon (2001a), 10% of horses with primary sinusitis and 11% with sinus cysts were reported to have a sanguinous nasal discharge. Other rare causes of epistaxis include sinonasal fibrous dysplasia (a benign, fibro-osseous lesion of unknown aetiology) (Livesey *et al.* 1984), nasal amyloidosis (the presence of homogenous extracellular protein masses) (van Andel *et al.* 1988), ulcerative rhinitis, nasal polyps and turbinate bone necrosis (Cook and Littlewort 1974).

Guttural pouch mycosis

The opportunistic pathogen *Aspergillus fumigatus* also appears to be responsible for mycotic infection of the guttural pouch with erosion of major blood vessels which lie within it. Epistaxis of large volumes of bright red arterial blood at rest is the most common clinical sign associated with guttural pouch mycosis (GPM), and is usually attributable to erosion of the internal carotid artery at or just below the sigmoid flexure, where the fungus has a predilection site (**Fig 13**). Less commonly, the external carotid or maxillary arteries (in the lateral compartment) may be eroded by the fungal plaque. Several bouts of haemorrhage usually precede a fatal episode. Although GPM is usually unilateral, epistaxis may be unilateral or bilateral, often depending on the volume of haemorrhage into the nasopharynx (i.e. if a profuse bleed occurs, blood will often flow from both nostrils), or if erosion of the median septum of the guttural pouch has occurred.

Emergency treatment for hypovolaemic shock should be instituted immediately for horses which have lost a significant volume of blood (e.g. greater than 4 l in a 500 kg horse). This should include venous catheterisation with a large bore (13 gauge or larger) catheter and rapid administration of large volumes of i.v. fluids, usually isotonic (0.9% w/v) saline or Hartmann's solution, or hypertonic (7.5% w/v) saline (4 ml/kg bwt) followed by isotonic saline or Hartmann's. Whole blood can also be administered if a suitable donor horse and equipment for collection are available. If haemorrhage shows no sign of slowing and the horse is at risk of fatal hypovolaemia, the ipsilateral common carotid can be temporarily ligated in the caudal cervical region as a salvage procedure until the horse is transported to a surgical facility for occlusion of the internal carotid artery, but there is a risk of cerebral damage with this procedure.

Other clinical signs of guttural pouch mycosis include more chronic mucopurulent or purulent nasal discharge, swelling of the ipsilateral submandibular lymph nodes and parotid region, hyperextension of the head and neck and dyspnoea. Neurological signs (**Table 1**) such as pharyngeal dysfunction resulting in dysphagia (**Fig 14**) (glossopharyngeal, vagus and accessory nerves), with nasal return of saliva/food/water, laryngeal hemiplegia (recurrent laryngeal branch of the vagus nerve), Horner's syndrome (cranial sympathetic nerve) and persistent dorsal displacement of the soft palate (pharyngeal branch of the vagus nerve) can occur when there is damage to the cranial nerves which run within the guttural pouch. Facial paralysis occasionally occurs in horses with GPM, with damage to the facial nerve as it courses over the dorsal surface of the lateral compartment of the pouch. Dysphagia and dyspnoea may alternatively be attributable to partial obstruction of the nasopharynx due to gross (ventral) distension of the guttural pouches, but this is uncommon with GPM. Atlanto-occipital arthropathy and stylohyoid arthropathy may also occur in horses with GPM (Dixon and Rowlands 1981; Walmsley 1988).

Endoscopically, blood, blood clots (**Fig 15**) or mucopurulent discharge may be seen emanating from the

ostium of the affected pouch. The cartilaginous flap of the ostium may be eroded and irregular in appearance in cases of chronic guttural pouch infection. If there has been a recent haemorrhage and a stream of blood or blood clot can be seen at the ostium, great care should be taken when advancing the endoscope into the pouch in case the blood clot at the site of arterial wall erosion is disrupted and a fatal haemorrhage ensues. However, once the horse is in a facility where surgical occlusion of the affected artery can be performed, the pouches should be examined endoscopically in order to confirm that GPM is the cause of epistaxis, to rule out the possibility of *rectus/longus capitis* muscle rupture, and to ascertain whether the fungal plaque is situated on the internal or external carotid artery (Caron *et al.* 1987). If no plaque is evident, the median septum and the contralateral pouch should be evaluated carefully, as erosion of the median septum in some cases of GPM may result in blood emanating from the contralateral ostium in the nasopharynx.

Within the guttural pouch, grey, black or white fungal and fibrinous plaques may be present. Occasionally, fungal erosion of the stylohyoid bone may result in stylohyoid osteopathy and/or pathological fracture.

If left untreated, there is a 50% mortality rate for horses suffering from GPM (Cook 1968). Treatment of GPM involves occlusion of the affected artery by simple ligation (Church *et al.* 1986; Greet 1987), insertion of a balloon catheter (Freeman and Donawick 1980a,b; Caron *et al.* 1987) or coil embolisation (Leveille *et al.* 2000). The use and efficacy of topical antifungal therapy in addition to arterial occlusion is controversial (Greet 1987; Speirs *et al.* 1995; Tremaine and Dixon 2001b), but it seems logical that topical application of an effective antifungal agent (e.g. natamycin) may prevent further nerve damage.

Rupture of the longus capitis and rectus capitis ventralis muscles

The *longus capitis* and *rectus capitis ventralis* muscles are flexor muscles of the head. They originate from the transverse processes of the 3rd, 4th and 5th cervical vertebrae, and the ventral arch of the atlas, respectively, and insert onto the basilar portion of the occipital bone and the body of the basisphenoid (Dyce *et al.* 1987; Sweeney *et al.* 1993). The insertion of these muscles lies dorsal to the pharynx and above the median septum of the guttural pouches (**Fig 16**). Avulsion and rupture of a portion of the *longus capitis* or *rectus capitis ventralis* muscles from the basisphenoid or occipital bones may occur in horses which rear and fall over backwards (Knight 1977; Sweeney *et al.* 1993).

Neurological signs associated with damage to the CNS are often present in horses with this injury.

Following the traumatic incident, the initial episode of epistaxis is usually transient, but can be severe, with milder episodes of epistaxis occurring later. A large haematoma forms within the guttural pouches, which may cause ventral deviation of the dorsal pharyngeal wall. There may also be external swelling of the retropharyngeal area, which is painful

on palpation. Endoscopically, blood clots or fresh blood may be seen emanating from one or both ostia of the guttural pouches, and it is therefore important to differentiate this condition from GPM by endoscopic examination of the affected pouch. Lateral radiographs of the base of the skull may show avulsed bone fragments which originate from the ventral part of the basisphenoid. In the absence of neurological signs, conservative treatment may be successful (Sweeney *et al.* 1993).

Exercise-induced pulmonary haemorrhage (EIPH)

Exercise-induced pulmonary haemorrhage (EIPH) has been reported to occur in 55–95% of Thoroughbred horses after fast work (Raphel and Soma 1982; Burrell 1985; Roberts and Erickson 1999); however, its occurrence among groups of horses competing in less vigorous activities is much lower.

EIPH originates mainly from the dorsocaudal region of the diaphragmatic lobes of the equine lung as a consequence of strenuous exercise. The mechanism of EIPH is unknown, but one theory is that haemorrhage is the result of stress failure of pulmonary capillaries (the combination of a relatively thin blood gas barrier required to support a high maximal oxygen uptake, and the high pulmonary capillary transmural pressures as a result of both high vascular and very negative alveolar pressures, places a tremendous stress on the pulmonary capillary walls) (West *et al.* 1993). Schroter *et al.* (1998, 1999) suggested an alternative mechanism of pulmonary capillary disruption, due to localised shear stress within the pulmonary tissue resulting from loading (compression) of the chest by the forelimbs. In occasional cases, atrial fibrillation may predispose to EIPH due to increased left atrial pressures leading to increased pulmonary vascular pressures. The role of small airway inflammation in EIPH is uncertain, and the small airway inflammation and subsequent interstitial, vascular and airway remodelling seen in horses with EIPH may be a response to haemorrhage rather than the initiating cause (Step *et al.* 1991), but its presence may well cause further susceptibility to the condition (Pascoe 1997).

Clinical signs include bilateral epistaxis after exercise, coughing and poor exercise performance. In some cases with low volumes of pulmonary haemorrhage, epistaxis may be unilateral. Occasionally, EIPH can be fatal. However, most affected animals show no overt clinical signs, with only 1–10% of horses with evidence of blood in the trachea having epistaxis (Raphel and Soma 1982). Blood is usually best visualised in the trachea 60–90 mins after exercise (Fig 17), but old discoloured blood may be present for a week or more if pulmonary haemorrhage has been severe. A semiquantitative grading system is used widely by racecourse veterinarians in the UK (Table 2).

Tracheal aspirates or bronchoalveolar lavage (BAL) fluid may be red or brown tinged, and following recent EIPH, cytology of these fluids may initially reveal erythrocytes which are either free or within phagocytosing macrophages (Fig 18). Haemosiderophages (Fig 18) may be identified in either Diff

TABLE 2: Grading system for exercise-induced pulmonary haemorrhage

Grade	Endoscopic examination of trachea
1	Flecks of blood only
2	>Flecks<continous stream
3	Bloodstream<half tracheal width
4	Bloodstream>half tracheal width
5	Airways filled with blood

Quik or Leishmann's stained preparations for >150 days after the last episode of EIPH (O'Callaghan *et al.* 1987). Consistent radiographic changes are detected in only 10% of affected horses and are usually present for several days following EIPH. These changes include increased soft tissue density (broncho-interstitial pattern) in the caudodorsal lung fields, and occasionally a (temporary) alveolar pattern if haemorrhage has been severe.

The treatment of horses which have undergone severe haemorrhage includes rest and NSAIDs (Hinchcliff 2001). A graded incremental training regime can be introduced for affected horses, and good air hygiene is indicated to control and aid resolution of small airway inflammation (Hinchcliff 2001). Frusemide is the most widely used pharmacological treatment for EIPH, as some studies have shown it to reduce pulmonary vascular pressures during exercise (Manohar and Goetz 1996). However, there is conflicting evidence as to its efficacy in preventing EIPH (Manohar and Goetz 1996; Lester *et al.* 1999; Kindig *et al.* 2000). The use of frusemide is permitted during racing in the USA, but it is banned for Thoroughbred racing in the UK. The use of nasal strips has also recently been shown to significantly reduce the BAL erythrocyte count post exercise (Kindig *et al.* 2000; McDonough *et al.* 2000), but such devices are also banned for Thoroughbred racing in the UK.

Haemorrhagic pleuropneumonia

Acute necrotising pneumonia with thrombosis has been described as a cause of epistaxis in the horse (Carr *et al.* 1994). It is a rare disorder, usually associated with prior strenuous exercise or transportation. Affected horses present with serosanguinous nasal discharge, tachypnoea, tachycardia, toxic mucous membranes, pyrexia, cough and petechial haemorrhages. On endoscopy, blood can be visualised in the trachea (Fig 19). Horses respond very poorly to medical treatment and, on *post mortem*, lesions are consistent with primary intraparenchymal haemorrhage (Fig 20) with secondary opportunistic infections (Carr *et al.* 1994).

Neoplasia of the lower respiratory tract

Primary and secondary tumours of the lower respiratory tract occur infrequently in the horse and, when they do, rarely present with epistaxis (Mair *et al.* 2004). Inappetance, cough, depression, lethargy, exercise intolerance, weight loss and intermittent pyrexia are more common presenting signs

(Scarratt and Crisman 1998; Mair *et al.* 2004). Primary tumour types are very rare and include pulmonary granular cell tumour, mesothelioma, pulmonary and bronchial carcinoma and adenocarcinoma, bronchogenic squamous cell carcinoma, pulmonary chondrosarcoma and bronchial myxoma (Mair *et al.* 2004). Secondary (metastatic) tumours of the lower respiratory tract are much more common, accounting for over 90% of cases of thoracic neoplasia (Mair and Brown 1993; Sweeney and Gillette 1989). There are many single case studies reporting a wide variety of metastatic thoracic neoplasms; however, renal carcinoma, squamous cell carcinoma, melanoma, fibrosarcoma and haemangiosarcoma appear to be the most common (Mair *et al.* 2004).

Systemic clotting diseases

Epistaxis may be a presenting sign in these relatively rare equine disorders. They include primary or secondary immune-mediated thrombocytopenia, disseminated intravascular coagulation (DIC), haemophilia A, or any other disease process causing thrombocytopenia (platelet count must usually be $<20 \times 10^9$ cells/l before spontaneous haemorrhage occurs) (Sackett *et al.* 1987; Adams 1997).

Summary

Epistaxis is a clinical sign which occurs with a wide range of disorders of the equine upper and lower respiratory tract. The nature of the epistaxis and the presence of concurrent clinical signs may give clues as to the site and cause of haemorrhage. Unilateral epistaxis usually indicates that the lesion is located in the upper respiratory tract, whereas bilateral epistaxis usually originates from the lower respiratory tract or the guttural pouches (if large volumes of haemorrhage are involved).

Endoscopic examination of the respiratory tract often results in a definitive diagnosis, but if not, will usually reveal the anatomical location of the source of haemorrhage. Radiography is often useful for evaluation of the extent and nature of lesions which are not visualised or incompletely visualised using endoscopy. Other imaging modalities can be useful in selected cases.

The most common causes of epistaxis occurring at rest are facial and sinonasal trauma, progressive ethmoidal haematomas and guttural pouch mycosis. In horses which have recently undergone fast exercise, exercise-induced pulmonary haemorrhage is the most likely cause of epistaxis.

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