The respiratory system

Introduction – The significance of respiratory disease in working equids

12.1

Defence mechanisms of the respiratory tract and spread of disease in populations

12.2

Examination procedure

12.3

Clinical signs of respiratory disease

12.4

Disorders of the upper respiratory tract

12.5

Viral respiratory disease

12.6

Bacterial respiratory disease

12.7

Allergic respiratory disease

12.8

Parasitic respiratory disease

12.9

Epistaxis

12.10

Case study – Aspiration pneumonia

12.11

References

12.12
Respiratory tract problems are common in working equids and, even without extensive diagnostic tools such as radiography and endoscopy, it is possible to do a thorough clinical examination and make an informed decision about the most likely cause based on clinical signs.

Respiratory signs can be very subtle – often just a small increase in watery discharge from the nose, a slight cough or sneeze, or the animal being described as less vigorous than normal by the owner.

There are many different levels and types of respiratory disease, and secondary bacterial infection is common.

The primary purpose of the respiratory system is to enable oxygen to enter the body. Within the alveoli of the lungs, oxygen diffuses into the blood which is then pumped around the body to supply cells with oxygen for aerobic respiration. The musculoskeletal system is extremely important to the working equid; effective work is not possible if the blood to the muscles is not well oxygenated (Figure 12.1.1). Therefore, the cost to the animal from respiratory disease is much more than just discomfort and difficulty in breathing (dyspnoea).
Defence mechanisms of the respiratory tract and spread of disease in populations

The working and living conditions of working equids are usually dusty and polluted (Figure 12.2.1), often with many animals working or housed together.

Damaged airway defences and close confinement will increase the rate of transmission of viral and bacterial respiratory infections through the local population.

The respiratory tract has several functions:
- Intake of air for oxygenation of blood
- Thermoregulation by evaporative cooling
- Compensatory mechanisms, e.g. in metabolic acidosis

The respiratory tract does not have a device to scan air quality; all air goes in regardless of quality. Therefore, if an animal is working or living in an area where the air quality is not good (contaminated with dust, smoke and pollution), then the defence mechanisms may become overwhelmed or damaged.

The mucociliary escalator protects the respiratory tract by trapping dust particles or pathogens in a mucous layer which is then moved upwards towards the throat by beating cilia (small hairs). It has been shown that the cilia are paralysed in humans who smoke; it is likely that the same effect occurs in equids that are exposed to pollutants.

Damage caused by dust and pollution leaves the respiratory tract vulnerable to secondary bacterial infection.

Once an infection is established, the pathogens within the respiratory tract will induce an inflammatory response. Subsequently mucous secretion increases which can be observed as a nasal discharge. Sneezing and coughing spreads the secretions thus aiding the dissemination of the virus and bacteria to other hosts.

It is essential to implement thorough disinfection protocols following the treatment of a respiratory case if an infectious cause is suspected. Disinfect hands, clothes, mouth gags, nasogastric tubes and stables after every respiratory case. Wear gloves when examining and treating cases. Isolate the affected animals from the healthy population.

Figure 12.2.1 Dusty working environments increase the risk of respiratory infection in working equids.
Appropriate isolation with no nose-to-nose contact and a separate air space is important to prevent circulation of pathogens. Monitor the remaining population closely for the onset of clinical signs indicating respiratory infection. Treat and isolate any other suspected cases.

It can often be beneficial to plan isolation facilities in advance. A disused shed or stable could be cleared and prepared.

12.3 Examination procedure

As always, it is important to conduct a thorough clinical examination, even if the animal presents with an obvious breathing problem. Many respiratory diseases result in systemic signs, and also many ‘coughs’, which may worry the owner, can reflect environmental conditions or secondary bacterial infection, rather than being the primary cause.

Overall assessment of the respiratory case

Taking a good history. Ask the owner:
- What signs/symptoms has he noticed?
- How long have the signs been present?
- Have the signs been more common at a particular time of day or during a particular activity?
- Are they getting better, worse or staying the same?
- How old is the animal?
- Are there other animals in the area showing similar signs?
- Have any animals been brought in recently or has anyone been to an equine fair?
- Has the animal been treated by anyone else recently (e.g. drenched)?

Detailed examination of the respiratory system

Look at the body and coat condition and hydration status in conjunction with the clinical parameters of temperature, heart rate and respiratory rate.

- Head and neck  Examine the head and neck for signs of asymmetry. Look for swelling of lymph nodes, especially in the submandibular area, as this may indicate strangles. Asymmetry of the face could indicate sinus problems or tumours.
- Mucous membranes  Check the colour (cyanosis, a blue colour, may indicate poor perfusion) and for any haemorrhage.
Nostrils  Feel the air flow to ensure that it is equal on both sides. Listen for abnormal sounds/smell coming from a nostril that could indicate a problem in the nasal cavity or guttural pouches. Assess for nostril flaring. Assess any discharge, noting amount, type and frequency. Serous nasal discharge is common early in the course of a viral respiratory infection, and a mucopurulent discharge is more likely with bacterial infection.

Frontal/maxillary sinuses  Look for swelling, and percuss to determine whether fluid is present/or there are signs of pain. Anatomical knowledge of the borders of the sinuses is essential (Figure 12.5.2). Fluid will result in a dull sound on percussion. This is sometimes easier to hear with a stethoscope, and opening the animal’s mouth can help accentuate the sounds.

Larynx  Palpate for asymmetry or pain. Gently squeeze at the junction of the larynx and trachea; if a cough is elicited it can indicate that the upper airway is inflamed and irritated.

Auscultation  Auscultate both the upper, larynx/trachea (Figure 12.3.1) and lower respiratory tract, lungs (Figure 12.3.2). Not all conditions will produce changes in the lungs.

A quiet environment is required so that even slight changes can be detected; this takes practice and knowledge of what ‘normal’ sounds like.

The normal margins of the lungs

- Basal border  A line from the costochondral junction of the 6th rib, crossing the middle of the 11th/12th rib, to the margin of the ventral back muscles at the 16th inter-costal space
- Dorsal border  From the caudal border of the scapula to the tuber coxae
- Cranial border  Tricipital margin from the caudal border of the scapula to the olecranon

Auscultating many equids’ lungs not only forms part of a good clinical examination but improves the perception of normal lung sounds allowing more accurate determination of abnormal lung sounds.
When training other veterinarians in auscultation skills it can be useful to outline the lung field on the side of the animal (Figure 12.3.3).

An understanding of the anatomy and physiology of the equine respiratory system allows effective interpretation of sounds to place them in the context of the most likely pathological condition.

For example, increased lung sounds in the cranio-ventral region may be indicative of a bacterial pneumonia, whereas abnormal lung sounds in the caudo-dorsal region may occur as the result of allergic conditions.

Percussion of the chest

Chest percussion can detect fluid or pleural pain. This can be done using a thumb and middle finger to flick the chest. Work around the chest in a systematic manner so the entire lung fields are percussed. Dull areas indicate consolidation near the lung surface or the presence of fluid.

Use of a re-breathing bag

A plastic re-breathing bag can be used to increase the rate and depth of breathing by decreasing oxygen availability. This aids auscultation by accentuating subtle lung changes. Areas of pneumonia/lung consolidation may not have air passing through, and so will be silent on auscultation.

However, there are instances where a re-breathing bag should definitely not be used and, at all times, the animal’s welfare is the first priority. Using a re-breathing bag can cause further respiratory distress in an already compromised animal, and taking deeper breaths, with painful conditions such as pleuritis and pleuropneumonia, will only add to the animal’s suffering for the sake of a diagnosis.

In foals the lung sounds can be easily auscultated through the thin thoracic walls so it is unnecessary to use a re-breathing bag.
Another complication when using a re-breathing bag is paroxysmal coughing which can occur in equids with a sensitive airway, e.g. in chronic respiratory allergies.

To use a re-breathing bag: Loosely cover both nostrils with a plastic bag for approximately 30 seconds; the respiratory rate is increased. Auscultate the lungs once the bag is removed and the equid takes several deep breaths. Stop the procedure if at any time the animal becomes distressed.

Peculiarities of the donkey

Please refer to the paper (Thiemann and Bell 2001) for further information.

- Donkeys rarely cough when suffering respiratory disease and may display only subtle clinical signs. Cases are often presented only once the disease is advanced and severe.
- Their lung sounds are generally more audible than horses'.
- Donkeys have narrower airways and a pointed epiglottis, so take care when intubating to avoid damage or haemorrhage. Use an endotracheal tube size 21.6 mm (size 16) or 18.6 mm (size 14); use a naso-gastric tube size 9.5 or 13 mm outer diameter.

Throughout a clinical examination and diagnostic work-up it is essential to consider the welfare of the horse, donkey or mule.

Clinical signs of respiratory disease

Clinical signs of respiratory disease are varied; consider the signs in context with respiratory tract anatomy when making a diagnosis.

Symptoms of respiratory disease can include the following:

- Cough
- Nasal discharge (Figure 12.4.1)
- Trouble breathing (dyspnoea)
- Increased respiratory rate (tachypnoea)
- Respiratory noise (inspiratory or expiratory)
- Abdominal movement when breathing, or a heaves line (Figure 12.4.2)
- Stance with abducted limbs and elongated neck (Figure 12.4.3)

Figure 12.4.1 Nasal discharge.
Pyrexia and other systemic signs
Associated ocular pathology
Poor mucous membrane colour
Poor body condition score in chronic cases

Examples of respiratory disease relevant to working equids
- Infectious disease – viral and bacterial
- Allergic conditions
- Sinusitis
- Guttural pouch pathology
- Aspiration pneumonia/foreign bodies
- Parasitism

12.5 Disorders of the upper respiratory tract

Conditions affecting the nasal passage
There are many conditions affecting the nasal cavity in working equids. Due to the difficulty of definitive diagnosis in the field, *without endoscopy*, in most cases it is important to try to manage the symptoms and remove the underlying cause if possible.

Examples of nasal passage problems
- Trauma – primary or iatrogenic e.g. via passage of a naso-gastric tube or inappropriate drenching
- Infection – sinusitis, ethmoid necrosis
- Foreign body
Nasal septum abnormalities
Neoplasia
Polyps
Congenital malformation

Clinical signs of respiratory disease
- Nasal discharge – in a range of colours and thickness (see Figure 12.5.1)
- Epistaxis (bleeding) (see Section 12.10)
- Foul odour coming from mouth or nostrils
- Alteration/uneven air flow from nostrils
- Respiratory noises

Treatment
- Nasal foreign bodies  Remove with forceps if visible through the nostrils.
- Tumours or polyps  Surgical removal of tumours or nasal polyps requires good access to the nasal cavity, involving bone flaps or trephination. Without the use of radiography and endoscopy the extent of pathology is difficult to determine which makes planning the surgery problematic. A common complication of nasal surgery is profuse bleeding, so this type of surgery is rarely done in the field.
- Bacterial infections  A prolonged course of antibiotics is required as well as rest from work.
Sinusitis

The maxillary and frontal sinuses are connected to the nasal cavity and, as a result, upper respiratory problems can extend into these areas. The last four upper cheek teeth also extend into the maxillary sinus (Figure 12.5.2), so tooth root infections can present as secondary sinusitis.

Clinical signs

- Chronic, persistent unilateral nasal discharge (mucopurulent)
- Discharge – may be bilateral but more commonly only affecting the sinuses on one side
- Difficulty breathing which worsens with exercise
- Concurrent upper respiratory tract infection
- Dull sound on percussion over the sinus area indicating fluid
- Pain on percussion or palpation
- Smell from mouth and difficulty eating (if a tooth root abscess is the primary cause)

Rare events in chronic cases

- Distortion of the facial bones
- Neurological dysfunction from infection extending beyond the frontal sinus

Treatment

Sinusitis can be frustrating to treat given the difficulty of access to the sinuses and low penetration by drugs. Infections often recur, so warn the owner.

- Long-term antibiotics, often weeks of treatment, are required. Penicillin is the most effective medication.

- If a trephine is available, and the attending veterinarian has the relevant experience, fluid can be drained from the sinus. Drainage of the sinus with a catheter through a small trephine hole may be beneficial in the short term. It is essential to study the sinus anatomy in detail to ensure accurate placement of the catheter. Sedate the animal and inject 5 ml of local anaesthetic subcutaneously at the site of trephining. For placement of a lavage catheter a diameter of 7 mm is sufficient. Close the trephine portals with subcutaneous and skin sutures. The catheter can be secured in place if repeated flushing is required. Pain relief post-operatively is essential. Bone flaps are not recommended for field situations.

- A thorough oral examination is essential to determine whether dental problems exist (see Section 10.4).

Please refer to the dentistry chapter (Chapter 10) for more information on the treatment of suspected teeth problems.
Guttural pouch infections

Guttural pouches are air-filled diverticula of the auditory tube.

The pouches are connected to the pharynx through ‘slits’ which open when the animal swallows. Guttural pouches are lined with a thin mucous membrane which offers little protection to the vital structures that cross the dorso-caudal aspect of the pouch. These structures are very vulnerable to damage if the guttural pouches are infected, and this can result in life-threatening pathology (refer to guttural pouch mycosis under epistaxis in Section 12.10 of this chapter).

These vital structures include:
- Cranial nerves – VII, IX, X, XI, and XII (see Section 18.2 for more information on the cranial nerves)
- Cranial sympathetic trunk
- Internal carotid artery

Guttural pouches are prone to pathology as a result of secondary infection extending from the nasopharynx.

Externally, the location of the guttural pouches can be seen in the region of Viborg’s Triangle. See Figure 12.5.3 in which an abscess within the guttural pouch has ruptured through the outer wall.

The landmarks are as follows:
- Rostral border  Vertical border of the ramus of the mandible
- Dorsal border  Tendon of insertion of the sternomandibularis muscle
- Ventral border  Linguofacial vein

A study of guttural pouch anatomy in the donkey, including a diagram of Viborg’s Triangle, can be found by referring to the paper (Alsafy et al. 2008).

Clinical signs
- Nasal discharge (unilateral or bilateral), usually increasing when the animal swallows or lowers its head
- Distension in the region of Viborg’s Triangle (not always present)
- Endoscopy can be used to diagnose guttural pouch infection; however, observation of signs can support an initial suspicion without requiring further diagnostic tools.
Treatment

- Streptococcus spp. (including S. equi) are the most common bacteria to cause guttural pouch infection. If strangles is suspected, isolate the animal and carry out appropriate disinfection as a priority. Treat with penicillin only if systemically ill.
- Guttural pouch lavage can be performed with warm fluids over several days, placement of the catheter may be difficult without use of an endoscope. See other equine anatomy texts for landmarks.
- Severe infections require specialist surgical drainage through Viborg's triangle – care must be taken to avoid vital structures, e.g. vagus and glossopharyngeal nerves and the internal carotid artery.

12.6 Viral respiratory disease

Respiratory viruses affecting equids include equine influenza, equine herpes viruses (EHV) and rhinoviruses. The symptoms are very similar and it is not always necessary to distinguish between them for clinical purposes. Viral respiratory disease is common in younger animals, although all working equids can be susceptible, especially when kept together in groups as respiratory viruses are very contagious.

Respiratory viruses are transmitted by aerosol as well as by direct contact with nasal secretions.

Remember the potential for secondary bacterial infection to occur.

Suspect a viral cause even in cases with a small amount of bilateral watery nasal discharge.

Clinical signs (These vary from mild to severe.)

- Nasal discharge (small amounts, watery, serous)
- Lethargy
- Fever (39–41°C)
- Poor appetite
- Ocular discharge and conjunctivitis
- Abortion and ulceration of the oral mucosa sometimes seen in EHV-1 and EHV-4

Within 24–48 hours

- Development of a harsh, dry cough in the case of influenza
- Harsh lung sounds
- Nasal discharge becomes thicker and more profuse (mucopurulent), with the colour changing to grey or yellow. In severe cases, pneumonia may develop.
Diagnosis

- Suspect a viral pathogen based on clinical signs and the numbers of animals infected.

- Virus isolation is not performed in most cases, as disease is self-limiting.
- In severe outbreaks a definitive diagnosis is necessary (e.g. to initiate a control programme). Check with local laboratories for available testing methods and which samples are required. Serological testing is the most common. A paired sample, at least 14 days apart, is usually necessary. ELISA (Enzyme Linked Immunosorbant Assay) field kits may be available in some countries. Nasal swabs are only useful if PCR (Polymerase Chain Reaction to detect viral DNA) or other virus-isolation technology is available and often require special transportation.

Treatment

- The best treatment is to allow resolution of the infection by the animal’s own immune system. Rest, warmth and good food will usually ensure recovery in 7–10 days if there is no secondary bacterial infection.

- Isolate affected animals.

- As discussed in Section 12.1 ensure that there is no nose-to-nose contact or shared air space with other animals. Do not share tack or equipment with others.
- Symptomatic treatment may help but will not cure the virus.
  
  Administer:
  - NSAIDs for the pyrexia and airway inflammation
  - Mucolytics, e.g. eucalyptus oil, steam
  - Antibiotics if secondary infection is present, e.g. trimethoprim-sulphonamide

Prognosis

Good for a full recovery but acquired immunity is short-lived so recurrence is possible. In the case of equine influenza virus, the virus itself is constantly changing so immunity to one strain does not prevent infection from another. The severity of disease is reduced in vaccinated animals.

Prevention

Several killed/modified live virus vaccines are available. Vaccinations tend to protect against certain virus strains (equine influenza 1, 2 and EHV-1, 4 are most common) so diagnosis and knowledge of the actual virus responsible for outbreaks is required.

Immunity against equine respiratory viruses is short acting so any vaccinated animals will require a regular booster for vaccine efficacy.

Regular vaccination is not always achievable in the working equid situation. However, if there is a good relationship with owners who understand, a reliable vaccine supply and an effective cold chain, regular vaccination may be applicable.
It is better to manage animals so that the immune system is strong enough to fight viruses easily. Immediate isolation of affected animals helps to prevent spread of disease, as does reduced movement and mixing of animals. Ensure segregation of populations during markets or fairs. Ensure adequate rest allowing animals to recover and prevent a prolonged and severe clinical course of the disease.

It has been stated that working equids are more at risk of contracting influenza, increased severity of clinical signs, delayed recovery and secondary bacterial infections due to the additional stresses on their body and weakened immune system (Abd El-rahim and Hussein 2004). Figure 12.6.1 shows a horse continuing to work while infected with influenza.

Equine influenza

- Spread is rapid, morbidity is high. Uncomplicated infections tend to improve in 4–7 days, but a dry cough can persist for several weeks.
- Complications include secondary bacterial infection, cardiomyopathy, and persistent fatigue.

Equine herpesvirus 4

- EHV-4 is a major cause of acute respiratory infection worldwide. Most equids are affected in the first 2 years of life.
- Infections tend to be less severe and have a lower morbidity than equine influenza.
- Latent infection with EHV-4 and EHV-1 is possible, and reactivation can occur in times of stress or corticosteroid administration.

Equine herpesvirus 1

- EHV-1 can cause respiratory disease (as EHV–4). However, it can also cause abortion. Abortion occurs 2 weeks to 3 months after infection, usually between the 7th and 11th month of pregnancy. Future reproduction is unimpaired.
- In some cases a neurological condition is associated with EHV-1 caused by a viral myeloencephalitis. Ataxia with a history of a viral respiratory disease is a common presenting sign. Infrequently EHV-1 can lead to severe neurological signs such as recumbency and eventual death. Loss of bladder function is also a feature and this may persist in the long term.
Notifiable viral respiratory disease

African Horse Sickness

Cause

This disease is caused by an orbivirus of which there are nine serotypes.

The virus is spread by arthropod vectors, usually a Culicoides midge. It is important to realise that African Horse Sickness (AHS) is not contagious between individual equids but will spread in a population from the biting vectors, hence the importance of vector control.

Distribution

AHS is a seasonal disease endemic to Sub-Saharan Africa and parts of southern Africa. There have been historical reports of outbreaks in North Africa, Spain, India and Pakistan; however, there is no ongoing clinical disease in these areas. Consult the OIE WAHID interface for up-to-date information on outbreaks. Horses have the highest susceptibility, followed by mules, with donkeys usually showing mild or subclinical signs (Figure 12.6.2). Wild equine species (i.e. zebras) are resistant but may be involved in transmission because they attract Culicoides midges. AHS mainly occurs in warm, rainy climates/seasons when Culicoides midges are plentiful.

Clinical signs

All breeds of equids are affected (mortality rate 70–90%). There are a number of different manifestations of this disease depending on which form is present.

- Acute (horses) – Lung form
  - Has a short incubation period of usually 3–5 days
  - High fever
  - Laboured breathing/head down, coughing and profuse frothy nasal discharge
  - Mortality rate is high. Up to 95% of horses die within a week, often from ‘drowning’ in their own pulmonary secretions.

- Cardiac or subacute (donkeys)
  - Has an incubation period of 7–14 days
  - Fever followed by swelling over the head, eyelids, lips, cheeks and under the jaw
  - Conjunctival swelling which can be significant, even blocking vision
  - Mortality rate is about 60%, death results from heart failure.

- Mixed
  - A combination of the above two types
  - The incubation period is between 5–7 days and is indicated initially by mild respiratory signs which progress to the typical swellings of the cardiac form.

- Mild or Horse Sickness Fever form
  - Seen in zebras and African donkeys, this form is suspected with mucosal congestion and conjunctivitis (Figure 12.6.2). Animals usually recover.
Diagnosis

A presumptive diagnosis can be made based on clinical signs or post-mortem lesions in areas with a known vector presence. Laboratory virus isolation from infected tissues is available in some countries. Collect samples for serological tests 3 weeks apart to show a rising antibody titre associated with current infection. Serological ELISAs are available in South Africa. Animals often die before mounting an effective immune response.

Treatment

Currently no specific treatment exists; supportive therapy is the only course. Horses with the extreme acute form may have to be euthanased on welfare grounds.

Prevention

AHS is notifiable in many countries; report suspected cases to the relevant governing bodies. AHS is an OIE-listed disease. It is the responsibility of government veterinary services to report outbreaks to the OIE.

It is the responsibility of the veterinarian to know and recognise diseases that are notifiable and to ensure all suspected cases are reported at the earliest opportunity.

Live attenuated vaccines are available commercially in South Africa; these vaccines are polyvalent but may not contain the serotype causing a local outbreak. It is essential that the vaccines are administered annually. Viable vaccines at point of use are dependent on an effective cold chain. Ethiopia has a government subsidised vaccination program following a series of recent outbreaks (Aklilu et al. 2012).

Ensure vector control is maintained in endemic areas by covering animals with rugs or providing fly-proof shelter (especially in the evenings when midges feed) and using repellents such as citronella or other locally available substances that are not harmful to equids.
If vaccines and a virus testing facility are available this is the preferred protocol:

- Eliminate affected animals, by isolation initially and then euthanasia on confirmation of infection.
- Vaccinate non-infected equids with a polyvalent vaccine.
- When the virus has been serotyped re-vaccinate these animals with the homologous vaccine.
- Make housing for equids insect-proof.

For a good review of AHS, including prevention and control, please refer to (Mellor and Hamblin 2004).

Myths

The following will not achieve adequate vector control:

- Smoking drums around stables. This has no effect on midge activity and will have a detrimental effect on the respiratory function of equids and humans in the vicinity.
- Repellents alone. Using repellents is not enough to reduce midge contact; covering or stabling animals during high risks periods is strongly advised.
- Garlic supplements. There is no scientific evidence to support this.
- Moving equids to higher altitude. Midge can tolerate altitude, providing other environmental conditions are adequate.

Bacterial respiratory disease

Bacterial involvement is common in respiratory disease. It can be a primary infection, for example in strangles, or secondary infection to any viral or systemic disease causing the immune system to be compromised, including transport or overcrowding. Many different bacteria may be involved; it is not always necessary to know the primary pathogen.

Suspect bacterial involvement with any bilateral muco-purulent nasal discharge.

Bacterial pneumonia

This is one of the most common causes of disease affecting the lower respiratory tract of equids.

Causes

- Primary bacterial disease, e.g. strangles
- Secondary to upper respiratory tract problems (viral or otherwise)
- Secondary to generalised systemic disease
Inhalation of foreign material (aspiration pneumonia), e.g. food as a result of dysphagia, inappropriate drenching techniques (oral administration of medication, such as paraffin), home remedies (see Section 4.1)

Predisposing factors are those that suppress the pulmonary immunity: strenuous exercise, long-distance transport, and general anaesthesia.

Clinical signs

- Bilateral mucopurulent nasal discharge (Figure 12.7.1)
- Coughing
- Increased respiratory rate and effort
- Reduced exercise tolerance and lethargy
- Pyrexia and poor appetite
- Harsh lung sounds including pleural friction (see earlier in this chapter)
- Dullness of lower lung areas on percussion
- Pale or cyanotic mucous membranes in severe cases

Treatment

- The first line treatment of choice is Trimethoprim-sulphonamide; followed by procaine penicillin and gentamicin if unsuccessful. Use metronidazole if an anaerobic infection is suspected.

Ensure that owners are willing to comply with the necessary prolonged duration of treatment.

- Duration would be for at least 7 days, cessation of treatment is dependent on resolution of clinical signs which may take weeks or even months.
- Rest in a clean, dust-free environment. Ongoing stress whilst an animal has bacterial pneumonia is one of the most frequent causes of severe complications, e.g. pleuropneumonia. Offer a high quality palatable diet.
- Reduce pyrexia with the administration of NSAIDs. It is important NSAIDs are discontinued before stopping the antibiotic therapy – these medications can give a false impression of improvement.
- Corticosteroids may be administered in severe cases including aspiration pneumonia. One or two high doses will reduce inflammation. Always use in conjunction with antibiotics as steroids depress the immune response to infection.

Prognosis

The prognosis depends on the inciting cause: guarded for bacterial pneumonia, poor for inhalation pneumonia, contingent upon the amount of permanent lung damage.
Prevention

- Allow animals with viral respiratory disease to rest and recover properly before starting work again.
- Provide early treatment of secondary bacterial infections.
- Avoid or take extreme care when drenching equids (see Section 4.1 and the case study at the end of this chapter) or treating for oesophageal choke (Section 11.4).

Pleuropneumonia

Pleuropneumonia is defined as infection of the lungs and pleural space. This may occur as an extension of severe pneumonia, if an animal is not rested to allow recovery, or from the rupture of an abscess into the pleural space. Rarely, a penetrating injury to the thorax is the inciting cause of a pleuropneumonia. This condition is often accompanied by a pleural effusion; fluid builds up in the pleural space which is normally under negative pressure.

Clinical signs

- Respiratory distress – due to the pleural effusion
- Acutely painful (pleurodynia) – grunting on inhalation, abducted elbows, guarded behaviour when coughing, or flinching on percussion of the chest
- Nasal discharge – mucopurulent, bilateral. May be foul smelling if anaerobes are involved
- Inappetance
- No lungs sounds heard ventrally on auscultation
- Percussion – dull resonance ventrally

Treatment

- Long-term antibiotic therapy, as for pneumonia. Do not stop antibiotics before a complete recovery is made.
- Include metronidazole in the treatment protocol if anaerobic involvement is suspected. Anaerobic organisms are usually isolated in pleuropneumonia cases of greater than 5 days’ duration, and have been associated with poor prognosis for survival (Raidal 1995).
- Rest and supportive care
- IV fluids
- NSAIDs
- Good nursing care and nutrition are important in the recovery phase (see Chapters 21 and 22).

Prognosis

Guarded; especially if there is foul odour to the breath

Remember: Differential diagnoses for pleural effusion in equids include pleuropneumonia, neoplastic effusion, congestive heart failure, thoracic haemorrhage, chylothorax and pulmonary hydatidosis.
Strangles

Strangles is very contagious and can spread rapidly through a group of equids.

Younger animals are more susceptible, and the disease is often thought to be introduced to a native population by the arrival of new animals which are incubating the disease or have become carriers. Streptococcus equi sub-species equi (S. equi), is currently the most frequently diagnosed infectious disease of horses worldwide, responsible for high morbidity and occasional mortality of infected animals. Refer to the ACVIM Consensus statement for a detailed description of the treatment, control and prevention of strangles (Sweeney et al. 2005).

Transmission

- **Inhalation** of infected droplets in the air
- Direct contact with nasal discharge or pus from discharging abscesses
- **Fomites** on the ground, in feed, water troughs, equipment and on human hands/clothes

The incubation period is between 3 and 10 days.

Clinical signs (acute in onset)

- The first clinical sign is often pyrexia. If an animal has been exposed to a strangles case, monitoring the temperature will give an indication of whether an infection is developing.
- Swelling in the submandibular area, lymphadenopathy (Figure 12.7.2) which may progress to abscession of lymph nodes (Figure 12.7.3). Thick pus is often produced when these rupture.
- Poor appetite and dysphagia (can result in aspiration pneumonia)
- Coughing
- Thick purulent nasal discharge (Figure 12.7.1)
- Increased respiratory rate and effort

Signs that an animal may have had strangles in the past

- Chronic weight loss as a result of ‘bastard strangles’. This happens when abscesses have metastasised to other internal organs.
- Fibrosis in the area of the submandibular lymph nodes
- Chronic infections can develop in the **guttural pouches** and can be present without any clinical signs; animals which have this condition act as carriers, shedding bacteria and infecting others.
BACTERIAL RESPIRATORY DISEASE

Diagnosis

- Look for clinical signs, especially if lymph node abscessation is present and there is history of exposure.
- Bacterial culture of swabs from the nasal discharge or discharging abscesses will show the presence of S. equi.
- A nasopharyngeal swab can be taken, although it is unlikely to yield a positive culture if collected within 24 hours of exposure. An elongated swab is gently inserted into the nasopharynx (via the nostril). This stimulates the animal to swallow which releases any bacteria that are trapped in the guttural pouches.

Treatment

- Complete rest is very important.

- The majority of strangles cases require no treatment with antibiotics; those that are treated are more susceptible to recurrence of infection. Limit the use of medication to those cases where severe systemic signs of infection are present (cost versus benefit): administer procaine penicillin at high doses (20,000 IU/kg) daily.

Antibiotic therapy is indicated in cases with dyspnea, dysphagia, prolonged high fever, and severe lethargy or anorexia.

- Antibiotics, if used for treatment, must be continued for 5–7 days after clinical signs have resolved, as failure to do this may result in recurrence or development of bastard strangles.

- NSAIDs
  - Apply warm compress on abscesses to encourage maturation and rupture.
  - Abscesses may require drainage if they inhibit breathing or mastication. Determine the area where the skin is thinnest. Inject local anaesthetic sub-cutaneously. Clip and disinfect the region. Use a sterile scalpel blade to create an incision of up to 5 cm. Flush with warm saline. Continue flushing daily for 7 days. Remember the purulent discharge is highly contagious, so ensure all equipment and all personnel are disinfected before treating the next case. Drainage of the guttural pouches via Viborg’s triangle is not recommended for field surgery.
  - Give soft, palatable food.

Management of cases

Preventing dissemination of strangles is difficult as the disease is so contagious. Recovered animals shed large numbers of bacteria for 2–3 weeks after resolution of clinical signs, and intermittently for years if a carrier state is generated.

- Isolate affected animals for at least 4 weeks. Do not allow nose-to-nose contact with other equids.
- If bacterial culture is available then three negative nasopharyngeal swabs (at 4–7 day intervals) can be used as a determining factor for ending quarantine. (Intermittent shedding of bacteria occurs so this protocol increases the likelihood of detection.)
Ensure that buckets, feeding sacks, grooming equipment, etc. are not shared with other equids.

Strangles can be transmitted on human hands and clothes; it is important to observe strict hygiene measures, especially as a veterinarian or community animal health worker is likely to be handling many animals after treating an infected case.

Disinfect all instruments and equipment (including ropes and head collars). Drain and disinfect water troughs thoroughly as the bacteria can persist for a long time in this environment.

If draining abscesses, do not let pus collect on the ground where it will be a source of infection for other animals.

Inform owners of the epidemiology of disease and how to avoid spread.

Monitor the temperature of in-contact equids and isolate if the temperature increases and/or if there is sub-mandibular swelling.

**Prognosis**

Good for most cases, but poor if ‘bastard strangles’ develops

**Complications of strangles infection**

**Bastard strangles**

This is the systemic spread, or *metastasis*, of *S. equi* to any other part of the body other than the lymph nodes of the head. A large range of organs can be infected, e.g. lungs, liver, spleen or kidneys. Suspect bastard strangles in any animal that is currently suffering or has recently had strangles and is showing obvious and unusual clinical signs. Signs tend to be non-specific and depend on which organ has been infected. General signs include depression, anorexia, intermittent pyrexia, chronic weight loss and intermittent colic in a case where an abdominal organ is affected.

**Treatment**

This comprises a prolonged parenteral course of penicillin. If a very long period of treatment is required, potentiated trimethoprim-sulphonamide antibiotics given orally are an alternative. NSAIDs are useful to control temperature, colic signs and encourage the horse to eat if inappetant.

**Purpura haemorrhagica**

This is an immune-mediated condition, which is more frequently seen in older horses. It is a vasculitis resulting in obvious subcutaneous oedema, mainly of the head and limbs and petechial haemorrhages of the mucosa. In severe cases the vasculitis can affect other/all internal organs and the peripheral oedema can be so severe it can result in fatal circulatory collapse.

**Treatment**

Corticosteroids are given to suppress the immune response and reduce the inflammation in the blood vessels. Treatment with penicillin, although there are concerns that this may release more of the antigen that initiates the immune-mediated vasculitis, is still currently recommended (Pusterla et al 2003). Supportive care – such as leg wraps, light walking exercise, and palatable food.

**Rhodococcus equi infection of foals (‘rattles’)**

See Section 18.1.
Notifiable bacterial respiratory infections

Glanders (Burkholderia mallei infection)

Distribution

Asia, Africa and the Middle East

Glanders is very contagious to other equids and it can be transmitted to humans (zoonotic).

The lungs, nasal cavity, lymphatic system and skin (a variant condition called farcy) are affected with nodular abscesses. A sticky, purulent discharge is released once these nodules ulcerate. Skin exudates and respiratory secretions contain large numbers of bacteria that are easily spread by direct contact, fomites and environmental contamination.

Clinical signs

- Acute form predominantly affects mules and donkeys in which death may occur within a few days.
- Chronic form predominantly affects horses; the infection may persist for years.

Clinical signs of acute glanders are largely respiratory and more often seen in mules and donkeys.

- High fever
- Depression, anorexia, coughing
- Swollen nostrils with septum ulceration and a thick, mucopurulent haemorrhagic discharge (Figure 12.7.4)
- Multiple nodules and ulcers developing in the respiratory tract
- Swollen/ulcerated mucous membranes; the ulcers healing to form a typical star-shaped scar in those that survive
- Swelling and abscessation followed by rupture of regional lymph nodes
- Lung consolidation and pneumonia – round, firm greyish nodules developing in the lung tissue
- Severe respiratory distress
- Death within a few days

Farcy – cutaneous form of glanders

- Develops secondary to skin injury or respiratory disease
- Superficial and/or deep subcutaneous abscesses form with or without ulceration and inflammation of the local lymph nodes.

Figure 12.7.4 Nasal discharge and abscess seen in a horse with glanders.
Skin nodules can reach up to 2.5 cm diameter and may rupture releasing an infectious purulent discharge.

Nodules develop on the head, neck, thorax, ventral abdomen and legs (Figure 12.7.5).

Lymphatic vessels can become infected and abscessated, resulting in visible ‘farcy pipes’, or develop nodules – ‘farcy buds’ (Figure 12.7.6).

Causes weight loss

- Horses with chronic or latent infections often maintain the disease within the population as they act as reservoirs of infection.

Diagnosis

- Microscopic examination of a gram-stained smear of fresh material may reveal gram negative rods. The bacteria are non-sporolating and non-encapsulated.

- Bacterial culture is not recommended unless sent to a specialist laboratory due to the zoonotic nature of the pathogen. Be aware of the capabilities of the local diagnostic laboratory.

Be aware that handling infected discharge containing the bacteria can result in infection; wear gloves at all times. It is important that laboratory technicians are made aware of the risks when handling infectious material.

Mallein test

- The mallein test relies on a delayed hypersensitivity reaction in animals infected with glanders (Figure 12.7.7). This test is particularly useful in subclinical cases and is used as a prescriptive test for international trade. The protein is commercially available.

- Intrapalpebral injection of mallein protein derived from Burkholderia mallei bacteria

- Equids infected with glanders, and/or farcy, develop marked eyelid swelling, fever and occasionally a purulent ocular
discharge within 24 to 48 hours (Figure 12.7.7). Eyelid swelling is minimal in uninfected animals.

- This is a rapid field test for equids. Prompt confirmation of positive cases is crucial in infectious disease control to allow early intervention to limit spread.

- The mallein test is almost 100% specific although only 76% sensitive (see Section 4.2 for definitions of specific and sensitive) so there are likely to be more false negative results compared to false positives. It is essential to implement the isolation protocols for a suspected glanders case, pending further testing, even if negative in the mallein test.

- If the mallein protein is inadequately purified there are likely to be a greater number of false positives (de Carvalho Filho et al. 2012).

In countries or areas where there are no diagnostic facilities, then a diagnosis has to be made based on clinical signs alone.

Treatment

Euthanasia is recommended as recovered animals remain a source of infection for others, including humans.

OIE lists glanders as a notifiable disease. Follow the prescribed protocols in countries with legislation regarding the control of glanders. It is essential that veterinarians are aware of local control policies.

In India, for example, there is a Glanders and Farcy Act 1899 which outlines that the nearest Government Veterinary Officer (GVO) should be notified of suspected cases. A GVO will carry out the sampling (nasal swab and serum) for diagnosis and send this to the National Research Centre on Equines (NRCE). If glanders is confirmed by laboratory diagnosis the affected equid is euthanased by government authorities and compensation is paid to the owner. The animal is disposed of hygienically, the area is disinfected and animals which have been in contact are assessed.

In countries with no legislation or diagnostic facilities it is essential to implement the following protocol:

- Do not touch the animal with bare hands.
- Use face masks/disposable gloves and isolate the animal immediately.
- Dig a 2-metre-deep hole in the ground, euthanase the animal and bury it; OIE recommends burning the animal prior to burial.
- Clean and disinfect the area where the animal was living and working. Iodine is an effective disinfectant.
- Destroy harness and grooming equipment.
- Monitor any in-contact animals and humans. Early signs of glanders infection would include a high fever and the eruption of nodules.
- Isolate suspect cases.
- Attempt to notify the government and OIE of the case and inform local human health authorities.
Allergic respiratory disease is extremely common in working equids due to continuous exposure to dusty and polluted environments (Figures 12.8.1, 12.1.1 and 12.2.1).

Those at most risk are:

- animals working in urban or peri-urban environments
- brick kiln animals constantly exposed to brick dust
- animals fed dry, dusty food from a sack/nosebag, or stabled next to feed stores (see Figure 12.8.1)
- animals kept on dust, hay or sawdust
- animals suffering smoke irritation from being housed close to cooking fires.

Clinical signs

These vary in severity and may be intermittent. The clinical signs are similar to those seen for infectious disease as the immune system reacts to allergens in a similar way to pathogens.

- A chronic cough. May be more prevalent at certain times of the day when allergen exposure is greatest, e.g. at night, if caused by smoke irritation
- Increased respiratory rate and effort, even at rest
- A ‘heaves’ line which is common in animals with Recurrent Airway Obstruction (RAO) also known as Chronic Obstructive Pulmonary Disease (COPD)
- Flaring of the nostrils
- Lethargy and reduced exercise tolerance
- Harsh lung sounds on auscultation – can be exacerbated with a re-breathing bag
- Possible nasal or ocular discharge
- Usually normal appetite and no fever (unless secondary bacterial infection is present)
- Can present as acute attacks; respiratory distress with an increased respiratory rate and effort occurs suddenly. These can be severe and result in the animal's obvious distress.

Management

Remove the equid from the irritating environment to alleviate the signs of allergic airway disease.
As this is not always practical, long-term management of the disease may include some of the following:

- Change feeding practices to prevent inhalation of dust (e.g. feed pelleted rations rather than a mix). Soak mouldy hay to reduce inhalation of fungal spores (balance this against soaking for too long and losing considerable nutritive value of the feed).
- Improve bedding/resting environment if dust or smoke is excessive.
- Bronchodilators (e.g. clenbuterol, theophylline, aminophylline) may be useful in early stages if available; however, these medications are not as effective in severe cases and tolerance to clenbuterol develops over time and doses need to be increased (Read et al. 2012).
- NSAIDs or antihistamines may help.
- Corticosteroids (e.g. dexamethasone) are effective and cheap; they improve the condition, but long-term use may have side effects (see Section 5.4).
- Administer antibiotics if bacterial infection is present.

**Prognosis**

Without elimination of the allergen the prognosis is poor.
Lungworm – *Dictyocaulus arnfieldi*

Lungworm causes inflammation of the bronchioles in adult equids. Donkeys rarely suffer clinical signs of lungworm, but they act as a reservoir of infection for horses.

**Clinical signs in horses**
- Persistent chronic cough
- Slightly increased respiratory rate and lung sounds
- Non-progressive condition

**Diagnosis**
Patency (larvae in the faeces) is rare in horses. A bronchial lavage can be used to collect larvae for a definitive diagnosis.

Ivermectin or moxidectin are effective. Lungworm can recur if pasture contamination is present. Treat donkeys that are resident with horses, even if they do not show signs, as they can be a source of continued infection.

**Parascaris equorum**

Migrating larvae cause lung damage in foals.

**Clinical signs**
- Frequent coughing
- Greyish-white nasal discharge
- Possible fever
- Other signs of worm infestation, e.g. weight loss, poor coat, colic

**Treatment**

Broad-spectrum anthelmintics are effective.

**Prognosis**
The prognosis is good, but the disease can recur.
Epistaxis is bleeding from the nose (Figure 12.10.1). In many cases the source of the blood can be difficult to determine, especially without specialist equipment such as endoscopy. Blood can discharge from one or both nostrils and can vary from a slow drip or intermittent to profuse and life threatening.

It is important to note the characteristics of the discharge as this can indicate potential causes.

- Overt blood This is often seen with trauma of the nasal passages or lesions within the guttural pouches.
- Sero-sanguinous fluid This is often associated with chronic inflammatory changes and occurs alongside haemorrhage, e.g. abscess, sinusitis, neoplasia.

It is important to note any other associated clinical signs, e.g. fever, depression, inappetance.

Bleeding from the nasal cavity

- Trauma Apply pressure where possible to stop haemorrhage and promote clotting. Careful repair of a damaged nostril is important to prevent deformities which may limit inspiration, particularly if the muscles responsible for dilation are injured.
- Infection resulting in erosion (sinusitis, fungal infections, granulomas) Fungal infections are relatively rare but are more common in tropical/subtropical climates. Clinical signs include mucopurulent malodorous discharge, dyspnoea, abnormal respiratory noise and swelling of the head and lymph nodes of the throat. Culture of nasal discharge can help with diagnosis. In advanced cases, cream-coloured plaque accumulations can progress into ulcerated granulomatous lesions.
- Neoplasia
- Foreign body In addition to epistaxis, this can present as head shyness and obvious pain. A foul-smelling discharge is common. Sedating the animal will allow gentle removal of the foreign body whilst causing the least possible damage to the delicate nasal mucosa and nasal septum.

Bleeding from guttural pouch

Guttural pouch mycosis

This is a fungal infection of the guttural pouches. The guttural pouches contain numerous vital
structures (including the internal carotid artery and the vagus/glossopharyngeal nerves) which run across the caudal border of the guttural pouch, only protected by a thin mucous membrane. Fungal infections over these areas are often erosive and can damage these structures.

Clinical signs

- Intermittent spontaneous epistaxis of fresh arterial blood that can be extremely profuse and life threatening. A smaller episode of haemorrhage may precede the fatal bleed by 24 hours to 3 weeks. It is important to warn owners of the potential severity of even a small amount of blood at the nostrils.
- Dysphagia Difficulty swallowing as a result of damage to the vagus and glossopharyngeal nerves. Equids will cough and may have food/water coming out of their nostrils.
- Horner’s syndrome Characterised by variable degrees of ptosis, prolapse of the third eyelid, enopthalmas or unilateral sweating. Horner’s can be the result of damage to the cranial cervical sympathetic ganglion within the guttural pouch.
- Facial paralysis The facial nerve has been damaged.

Treatment

- In field conditions, treatment is impossible.

If a live threatening epistaxis occurs, or severe dysphagia, advise euthanasia on welfare grounds.

If an equid shows signs of profuse epistaxis of fresh arterial blood there is potential for a fatal bleed. If fungal mycosis is present this event will usually occur within 3 weeks of the initial bleed.

Empyema

This is the term for purulent material within the guttural pouches. It is the result of an infection that either ascends up the auditory tube or through lymphatic spread. The most common cause of this is strangles (see Section 12.7).

Clinical signs

- Intermittent nasal discharge worsening when the head is lowered
- Lymph node swelling and pain in the parotid region
- A sequaleae of strangles is the formation of chondroids within the guttural pouch. These solid balls of inspissated pus are difficult to remove. This can lead to a carrier state with intermittent shedding of bacteria, often without demonstrable signs of illness.

Treatment

- In the field environment treatment is limited, especially for chondroids. These can be removed endoscopically or surgically, neither of which is possible in the field context.
- Encourage lowering of head for drainage, e.g. feed from the floor.
- Treat primary infection.
Bleeding from the lungs
Exercise-induced pulmonary haemorrhage
Epistaxis is associated with strenuous exercise. The pathogenesis is not fully understood.

Clinical signs
Epistaxis during or following strenuous exercise. Poor performance or exercise intolerance, seen as a reduced ability to work. Excessive swallowing and or coughing after exercise as blood is being swallowed.

Treatment
Rest if possible, ideally in a dust-free environment. Advise the owner to do less strenuous/fast work to prevent recurrence. Antibiotics can be helpful if a secondary bacterial infection develops.

Pulmonary abscess. See treatment for pneumonia (see Section 12.7).

Neoplasia in the thoracic cavity is rare in equids and is most commonly due to metastases from distant sites. Weight loss is common and older equids are more often affected. In working equids, diagnosis is usually based on clinical signs alone. The condition is untreatable and, if an animal’s welfare is compromised, advise euthanasia.

Severe bacterial pneumonia or fungal pneumonia (see Section 12.7).

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Case study – Aspiration pneumonia

Area  Pakistan
Attending veterinarian  Dr Mohammad Iqbal Khan

Summary
This is a description of a case of pneumonia. It is assumed that this is a secondary infection as the result of aspiration when the donkey was drenched with local medication. The animal was treated with broad-spectrum antibiotics and anti-inflammatory medication.

History
A 13-year-old entire male donkey was presented to a static clinic with a 2-day history of coughing and nasal discharge (Figure 12.11.1). The donkey had been drenched (oral administration of fluids) with eggs, mustard oil and locally-available herbal masala.
Clinical findings

- Bilateral, muco-purulent nasal discharge (Figure 12.11.1)
- Elevated body temperature (39°C)
- Coughing
- Poor appetite
- Pale mucous membranes
- Watery discharge from the eyes
- Increased respiratory rate and effort
- Harsh lungs sounds on auscultation (Figure 12.11.2)
- Depressed and lethargic/less tolerant of exercise

Diagnosis

Aspiration pneumonia on the basis of history and clinical signs

Treatment (Figure 12.11.3)

- Procaine penicillin twice daily for 7 days
- Dexamethasone injection, single dose on the first day
- Ammonium chloride daily to act as an expectorant and mucolytic
- Pheniramine maleate antihistamine, administered 3 x daily as an oral tablet
Outcome

Although the donkey returned to work in the community he suffered from a low exercise tolerance and reduced work capacity. The owner sold the donkey at a fair.

Discussion

Owners may attempt to treat an animal using an oral drench. This may be carried out to provide the animal with energy, e.g., with milk and eggs. In this case the technique was used to administer a local treatment, herbal masala. These local medicines are also used for human patients and may be provided by a local healer.

If drenching is carried out while the tongue is being held and the head lifted, the equid will be unable to swallow effectively and some of the liquid will pass into the trachea. Thick and oil-based fluids are not suitable for drenching.

Advise owners to feed equids a sufficiently energy-dense fodder to cope with strenuous work instead of drenching with bizarre cocktails to provide extra energy. Provide water in a bucket and allow the equid sufficient time to drink, 4–5 times a day, rather than drenching with water (Figure 12.11.4). If working equids are unwell they should be taken to a veterinarian or trained community animal health worker (CAHW) if available. If drenching is carried out, advise on an appropriate technique so that the equid does not struggle and is able to swallow the liquid (see Section 4.1).

Changing an owner’s attitude and management is important in resolving such problems and preventing re-occurrence. Remember, establishing a good relationship with the owner is crucial (Figure 12.11.5).


World Animal Health Information Database (WAHID) Interface
http://www.oie.int/wahis_2/public/wahid.php/Wahidhome/Home
Further Reading

African Horse Sickness Trust booklet, 2012/13. Available online at www.africanhorsesickness.co.za


