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REVIEW ARTICLE

Respiratory emergencies in adult horses

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Summary

Although respiratory distress is encountered infrequently in equine practice, it can be serious and potentially fatal, requiring immediate and decisive intervention. History and physical examination can often give sufficient clues to identify the cause of respiratory distress and initiate emergency treatment. Ultrasonography can also be invaluable when assessing distress caused by lower respiratory tract issues. Upper respiratory tract obstruction, tension pneumothorax and occasionally pulmonary oedema can be rapidly fatal and require immediate intervention by emergency tracheotomy, thoracocentesis and evacuation of pleural air or administration of furosemide and oxygen, if available. Acute severe asthma and substantial pleural effusion are often not an immediate threat to life but nonetheless require fast intervention including administration of bronchodilators and corticosteroids or pleurocentesis, respectively.

KEYWORDS

horse, pleural effusion, pleuropneumonia, pneumothorax, pulmonary oedema, severe asthma, tracheostomy

INTRODUCTION

While respiratory emergencies are not particularly common in horses, they can be frightening and extremely distressing for horse, owner and attending veterinarian alike. Accurately diagnosing the underlying problem, understanding the underlying pathophysiology and initiating effective treatment quickly can be lifesaving. Causes of respiratory distress in horses and a flow chart of the diagnostic approach are summarised in Figures 1 and 2. Diagnostics, the underlying pathophysiology and management are discussed in the article below. Drugs commonly used in the treatment of respiratory distress are listed in Table 1.

Diagnostic tools for respiratory distress

The combination of history and physical examination findings frequently points towards a certain diagnosis and might be enough to initiate emergency treatment.

Externally visible swelling of the head and throat area points towards upper airway tract obstruction. Breathing is often associated with a characteristic high-pitched inspiratory noise and laboured inspiratory breathing pattern. Airflow is palpably reduced at the level of the nostrils which can be appreciated by holding both hands in front of the nostrils, feeling for airflow. Bilateral nasal discharge suggests guttural pouch or lower airway disease. Profuse mucopurulent discharge can be associated with strangles (upper airway tract obstruction), severe asthma or bacterial pneumonia or pleuropneumonia. Malodourous red-brownish discharge can be seen with severe necrotising pneumonia and frothy, white discharge or sputum can occur with severe pulmonary oedema. Auscultation of the lungs is neither sensitive nor specific but can give an indication whether lower respiratory tract disease is likely and what might be expected. Unfortunately, the absence of abnormal lung sounds does not rule out significant lung disease. In contrast, the presence of abnormal lung sounds is strongly suggestive of lower respiratory tract disease. In an acute asthma attack, widespread wheezes and crackles might be audible. The complete absence of any breathing sounds over the

dorsal or ventral lung field with a clear horizontal demarcation line might indicate the presence of a pneumothorax or pleural effusion, respectively. Occasionally, focal complete consolidation of the lung parenchyma can also result in the focal absence of normal lung sounds but this is difficult to appreciate in overconditioned horses.

Diagnostic imaging including ultrasonography, radiography and airway endoscopy help to refine an initial clinical impression and determine the extent of the lesion or disease process. However, they should not delay emergency treatment, particularly in cases of upper respiratory tract obstruction and suspicion of a tension

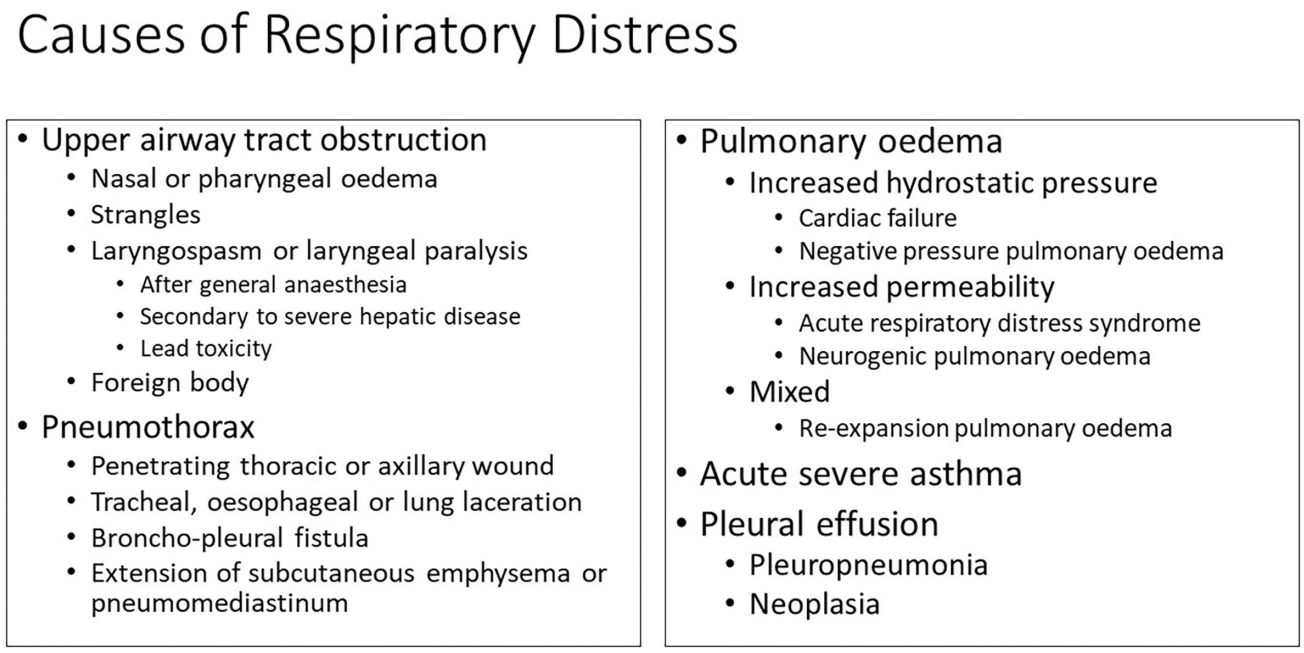

FIGURE 1 Common causes of respiratory distress in adult horses.

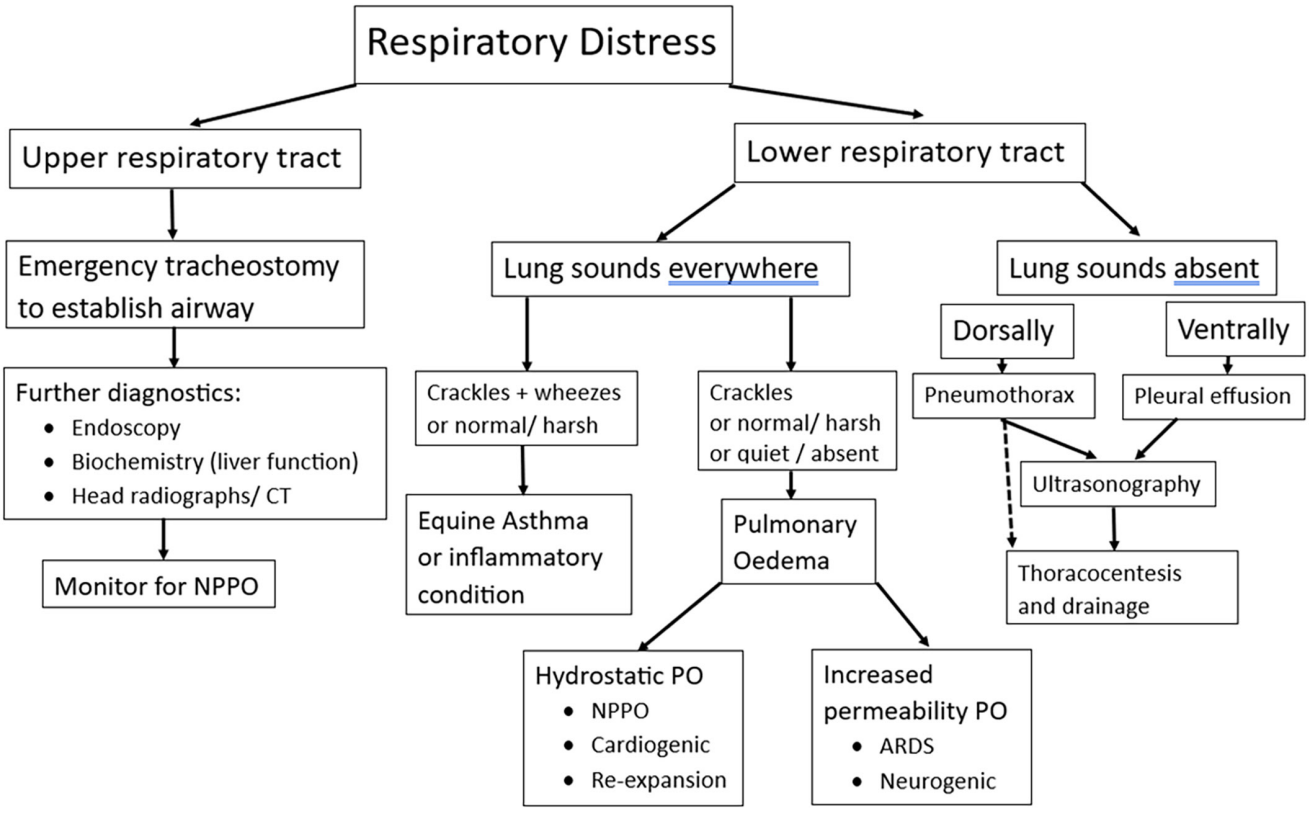

FIGURE 2 Diagnostic approach to respiratory distress in horses; NPPO, negative pressure pulmonary oedema; PO, pulmonary oedema.

TABLE 1 Drugs commonly used in the treatment of respiratory distress.

		Drug	Dose	Notes
Upper respiratory tract obstruction	Tracheostomy tube placement	Sedation: xylazine	0.2–0.4 mg/kg i.v. (1.0–2.0 mL for 500 kg horse)	Start with low dose of short acting α-agonist to establish response
Severe equine asthma[a]	Bronchodilators			
	Systemic	N-butylscopolammonium bromide (Buscopan®)	0.3 mg/kg i.v. once	
		Atropine	0.02 mg/kg i.v. once	Higher risk of systemic side effects than Buscopan®
		Clenbuterol	0.75–0.8 μg/kg i.v. q 12 h	Anticholinergics might be preferable
	Inhaled	Albuterol/salbuterol	1–2 μg/kg q 1–4 h 100 μg/actuation: 5–10	Onset of action 5 min; duration of action 30–60 min
		Salmeterol	0.25–1.0 μg/kg q 6–8 h 25 μg/actuation: 5–20	Onset of action 30 min; peak activity 3 h; duration of action 8 h
		Ipratropium bromide	0.8–5.5 μg/kg q 6–8 h	Onset of action 30–60 min; duration of action 4–6 h
	Corticosteroids			Full effect can take up to 7 days to establish
	Systemic injection recommended in horses with distress	Dexamethasone	0.05–0.1 mg/kg i.v./i.m./po q 24 h	Injectable route preferable
		Prednisolone	1.0 mg/kg po s.i.d.	Possibly less effective than dexamethasone
	Inhaled corticosteroids less effective in crisis			
Hydrostatic pulmonary oedema[a]	Diuretics	Furosemide	1–2 mg/kg i.v. q 4–6 h	Most effective in cardiogenic pulmonary oedema; not effective once hydrostatic pressure has returned to normal
Increased permeability pulmonary oedema[a]	Treat underlying disease			
	Corticosteroids for ARDS	Prednisolone sodium succinate or methylprednisolone	0.1–0.2 mg/kg i.v. divided in 2–4 doses	Based on anecdotal reports in foals and weanlings
Pneumothorax and pleural effusion[a]	Thoracocentesis and drainage			
	Treat underlying condition			

[a]Intranasal oxygen at 10–30 L/min via uni- or bilateral intranasal cannulas is useful if available.

pneumothorax. Endoscopy is invaluable for assessment of the upper respiratory tract, identifying the site and cause of obstruction or detecting inhaled foreign bodies. This should be done after an airway has been established by tracheostomy to avoid collapse or death during the procedure. Thoracic ultrasonography is a rapid assessment tool to confirm the presence and characteristics of pleural effusion, pneumothorax and pulmonary consolidation or abscessation. Pulmonary oedema might be suspected if multiple coalescing comet tails are visualised. Thoracic radiographs are useful to rule out radiographically visible lesions or obtain a better impression of size and location of a lesion but are rarely necessary or indicated in the first emergency assessment. Thoracic ultrasonography is more sensitive than radiography in detecting pleural effusion, pulmonary consolidation and atelectasis (Reef et al., 1991) and it is often quicker in an emergency. Exceptions are cases where widespread subcutaneous emphysema makes ultrasonography difficult and radiography becomes an important tool. A pneumothorax can be readily identified on thoracic radiographs focusing on the caudodorsal aspect of the lung and detection of diaphragmatic hernias, a rare cause of respiratory difficulties, is often easiest using this modality. However, obtaining good quality radiographs with a hand-held x-ray generator can be challenging in adult horses and access to a ceiling-mounted x-ray system is advantageous. In foals and miniature horses, use of computed tomography has been advocated for enhanced visualisation of intrathoracic structures including the lung parenchyma (Lascola et al., 2013, 2016; Schliewert et al., 2015). The need for general anaesthesia in often severely compromised patients will likely limit its usefulness in emergency cases, even if the animal is of an appropriate size.

Arterial blood gas analysis remains the most widely used tool to assess respiratory function and acid base status in the clinical setting to determine the need for oxygen treatment and identify hypoventilation. Analysis should ideally be performed within 10 min of obtaining the sample and any free air must be expelled and syringes sealed airtight. Blood collected in glass syringes kept on ice or in propylene syringes designed for blood gas analysis kept at room temperature can be stored up to 2 h before without significantly altering results (Deane et al., 2004; Kennedy et al., 2012; Picandet et al., 2007). An arterial sample can usually be obtained from the transverse facial artery in a standing horse.

How urgent is it? Pathophysiology of respiratory distress

The most severe and rapidly fatal cause of respiratory distress is complete obstruction of the upper airways as this prevents any inhalation of air. The only treatment in these situations is an immediate emergency tracheotomy. Intranasal oxygen is not effective as the oxygen cannot be inhaled into the lungs. The situation can be compounded by the development of negative pressure pulmonary oedema and clinicians need to watch out for worsening of the condition once an airway has been established which could indicate progressive pulmonary oedema.

A rapidly developing tension pneumothorax can also lead to fast deterioration and death. The rising intrathoracic pressure results in collapse of the lungs, compression of the caudal vena cava with decreasing venous return to the heart and increased pulmonary vascular resistance, further worsening cardiac output and ultimately leading to cardiopulmonary collapse and death. Immediate decompression of the pneumothorax is, therefore, lifesaving. Oxygen therapy is useful but only in conjunction with decompression.

Pulmonary oedema can cause acute, severe and potentially fatal respiratory distress by sudden flooding of the alveoli and small airways. The flooding can be caused by either an increase in hydrostatic pressure or increased vascular permeability, or both. In cases of hydrostatic pressure pulmonary oedema, the alveoli fill with a protein-poor transudate (cardiac failure, negative pressure pulmonary oedema) once the lymphatic drainage capacity is overwhelmed. Pulmonary epithelium and endothelium often remain intact and quick reversal is possible if the underlying cause is addressed. Increased permeability pulmonary oedema is caused by a sudden increase in endothelial and/or epithelial permeability, often due to damage or destruction of the endothelial and epithelial barrier (acute respiratory distress syndrome (ARDS), neurogenic pulmonary oedema). This results in filling of the alveoli with protein-rich inflammatory exudate, blood and debris. Reversal is much more difficult and slower, due to the tissue destruction.

Removal of the underlying cause (treating cardiac failure; establishing an airway; treating underlying disease) and oxygen insufflation is the mainstem of treatment in either case of pulmonary oedema. Furosemide is indicated in cardiac failure to lower the vascular hydrostatic pressure but is not effective if hydrostatic pressure is already normal, which is often the case in negative pressure pulmonary oedema, or if increased permeability is the underlying cause.

Acute asthma caused distress by bronchoconstriction and bronchodilatory therapy is, therefore, paramount. Although clinical distress can be severe, hypoxaemia is often only moderate and fatalities are extremely rare in horses. Additional oxygen therapy is very useful, if available, and recommended in people alongside bronchodilation to avoid worsening of ventilation-perfusion mismatch (see below).

Pleural effusion secondary to pleuropneumonia or neoplasia cause respiratory distress through a combination of systemic inflammation and hypoxaemia, secondary to filling of alveoli and small airways with exudate in mild cases and destruction of pulmonary parenchyma in severe cases. Pleural effusion exerting pressure onto the lung tissue and causing alveolar collapse and atelectasis can exacerbate the situation. However, similar to asthma, sudden death due to hypoxia or cardiovascular collapse is rare with the exception of the most severe cases and even emergency treatment with oxygen and thoracic drainage would probably not have resulted in survival in these horses.

Emergency treatment

Upper respiratory tract obstruction

Partial or complete upper airway tract obstruction can occur due to a variety of reasons ranging from severe nasal or pharyngeal oedema and soft tissue swelling, strangles, arytenoid chondritis, obstruction by a foreign body to bilateral laryngeal spasm or paralysis. The latter can occur following general anaesthesia, with severe hepatic disease, lead or other toxicities or for unknown reasons (Abrahamsen et al., 1990; Allen, 2010; Dixon et al., 2001; Hughes et al., 2009). Irrespective of the underlying cause, the clinical presentation is often similar as outlined above and an emergency tracheotomy is usually the only effective treatment option. If the horse is stable, it should be sedated. Starting with a low dose of a short acting sedative such as xylazine (0.2–0.4 mg/kg; 100–200 mg or 1.0–2.0 mL for a 500 kg horse) might be advisable to avoid possible collapse. If the horse is stable and not sufficiently sedated, higher doses or a longer acting drug, such as detomidine with or without butorphanol, can be administered. An area of skin is clipped, aseptically prepared and infiltrated with a local anaesthetic between the upper and middle third of the neck where the trachea can be palpated. An approximately 10 cm long incision is made vertically on midline, through the skin, subcutaneous tissues and cutaneous colli muscle and then extended between the paired sternothyrohyoideous muscles (Figure 3a). Once the trachea is exposed, a horizontal incision is made through the annular ligament between two adjacent tracheal rings, avoiding the tracheal cartilages and extended to the left and right (Figure 3b). The incision should not exceed more than third of the tracheal circumference. If available, a self-retaining metal tracheostomy tube or short cuffed silicon tube can then be inserted and secured in place (Figure 3c). Alternatively, a piece of nasogastric tube or large plastic syringe casing can be used (Prange, 2019). The relief of respiratory distress is almost instantaneous and respiratory rate and effort should improve within 1–2 h. Persistent or worsening distress despite resolution of an upper respiratory tract obstruction could indicate a secondary problem such as negative pressure pulmonary oedema (see below).

Pneumothorax

Pneumothorax in horses often arises from blunt or penetrating thoracic trauma or injury to air-containing structures (trachea, oesophagus, lung parenchyma). A recent case report describes haemopneumothorax after a bronchoalveolar lavage procedure (Kopec et al., 2024). It can also be secondary to pleuropneumonia causing pulmonary necrosis or formation of bronchopleural fistulas. Another cause is tracking of air from subcutaneous emphysema, after airway surgery or a pneumomediastinum into the pleural space as it might occur following a deep axillary chest wounds or rupture of the retropharyngeal recess. Pneumothorax can be unilateral or bilateral in horses suggesting that some have a complete and others an incomplete mediastinum (Boy & Sweeney, 2000). A tension pneumothorax describes a situation where air enters the pleural space with each inspiration but cannot escape with expiration, leading to progressive accumulation of air and increasing intrathoracic pressures. This, in turn, results in progressive pulmonary collapse, compression of the vena cava causing decreased venous return and decreasing cardiac output which ultimately results in cardiovascular collapse (Tran et al., 2021). Clinical signs are variable depending on the speed of onset, extent and concurrent disease processes. Auscultation can be helpful in identifying a pneumothorax, but has a low sensitivity, being diagnostic in less than 40% of cases in one study (Boy & Sweeney, 2000). Ideally, ultrasonography or radiographs are used to confirm a diagnosis but obtaining radiographs might be difficult in an emergency situation and ultrasonography can be hampered by the presence of subcutaneous emphysema. If the combination of history and clinical findings strongly suggests a pneumothorax, aspiration of air from the thoracic cavity via a needle thoracocentesis can be

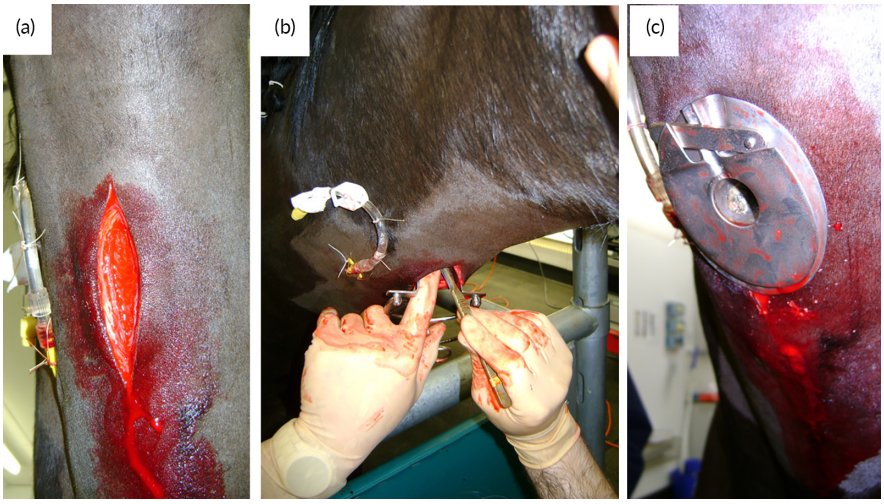

FIGURE 3 To perform a tracheotomy, the area is clipped, sterilely prepared and infiltrated with local anaesthetic before a longitudinal incision is made (a). A second incision is then made horizontally between the tracheal rings (b) to allow insertion of a tracheostomy tube (c).

attempted as diagnostic and therapeutic procedure. Needle thoracocentesis should be performed immediately if there is a suspicion of a tension pneumothorax with signs of cardiovascular compromise.

With a penetrating wound or deep stab wound, the first step of treatment is preventing further entry of air into the pleural space. This can be achieved by suturing or packing of the wound. Covering the wound or wrapping the patient in cling film can work well as an emergency measure if transport to a tertiary care facility is required. An equine compression suit has also been used to reduce air aspiration with good success in the management of a deep axillary wound (Tallon et al., 2023). A mild pneumothorax might not require removal of the free air within the pleural space. However, if signs of respiratory distress are present, emergency thoracocentesis is indicated.

After clipping and aseptically preparing a 5 × 5 cm large square in a dorsal aspect of the 11th through 15th intercostal space, ventral to the transverse processes, the area is infiltrated with a local anaesthetic and a small stab incision is made cranial to the rib using a scalpel blade. A small-bore tube (blunt-tipped teat cannula, 10–14-gauge intravenous catheter or 16-French or 24-French trocar catheter) is introduced into the skin incision and advanced into the pleural cavity. The catheter stylet or trocar is removed once the pleural cavity is accessed. It is helpful to connect the end of the catheter via an extension set to a 60 mL syringe that an assistant holds under negative pressure. Once the plural cavity is entered, air can be aspirated confirming the correct location and presence of a pneumothorax. Air is then removed by repeated aspiration with a 3-way stopcock and large syringe or by attaching a suction unit, limiting the setting to <−15 mmHg (<−20cmH_2O) to minimise the risk of re-expansion pulmonary oedema (Khanoria et al., 2021). In people, oxygen therapy is used to combat hypoxaemia and in an attempt to increase the speed of air re-absorption from the pleural space. Free pleural air is gradually reabsorbed into pulmonary lymphatics and capillaries. Approximately 1.25–2.2% of the volume of a spontaneous pneumothorax can be reabsorbed over a 24-h period in people and resolution of a moderate to severe pneumothorax might take several weeks if there is no ongoing leak (MacDuff et al., 2010). The administration of oxygen leads to a partial replacement of nitrogen by oxygen in the arterial blood and alveolar space, thereby increasing the absorption gradient between blood and air in the pleural cavity and quicker air absorption (Grasmuk-Siegl & Valipour, 2023). The technique is effective in animal models but its clinical usefulness has been questioned (Grasmuk-Siegl & Valipour, 2023; Zierold et al., 2000).

Pulmonary oedema

Pulmonary oedema can be caused by an increased hydrostatic pressure in the pulmonary vasculature, an increased permeability of the alveolar epi- and endothelium or a combination of both.

Cardiogenic pulmonary oedema is the best described example of hydrostatic pressure pulmonary oedema secondary to left-sided or generalised heart failure. While not occurring frequently in horses, any respiratory distress associated with an unexplained tachycardia, presence of a grade 3/6 or louder murmur or arrhythmia could indicate the presence of cardiogenic pulmonary oedema. Treatment with furosemide (1–2 mg/kg i.v. q4–6 h) while monitoring hydration status might provide transient relief until a full cardiac assessment can be carried out. Anecdotally, hypertonic saline has been used in the treatment of pulmonary oedema either with or without addition of furosemide. There is limited evidence to suggest that hypertonic saline is beneficial for treatment of pulmonary oedema regardless of the underlying pathophysiology in any species. Early work in dogs with experimentally induced hydrostatic oedema showed no benefit when using hypertonic saline with or without furosemide over furosemide alone in the clearance of pulmonary oedema despite achieving greater diuresis (Wickerts et al., 1992). This highlights the fact that increased diuresis decreases the hydrostatic drive for the development of pulmonary oedema but does not increase clearance of already accumulated pulmonary fluid. If feasible, intranasal oxygen at 10–20 L/min is indicated in hypoxaemic patients (PaO_2 < 65–60 mmHg).

Negative pressure or post-obstructive pulmonary oedema is less well described but not uncommon. It occurs secondary to a transient acute upper airway tract obstruction when the horse tries to inhale against the increased resistance of a partially or completely closed upper airway. The period of airway obstruction can be very short, only lasting minutes, for example with temporary occlusion of the nostrils or laryngospasm in horses recovering from anaesthesia or secondary to hepatic failure (Ball & Trim, 1996; Borer, 2005; Veres-Nyeki et al., 2011). Forceful inspiration against a closed epiglottis decreases intrathoracic pressure dramatically, thereby increasing the transpulmonary hydrostatic pressure and increasing venous return to the right side of the heart and the lungs. Both mechanisms result in fluid extravasation from the capillaries into the interstitium and ultimately alveoli (Unger & Martin, 2023). Rarely, a similar situation might be encountered after sudden relief of chronic upper airway tract obstruction, but this has not yet been reported in horses (Unger & Martin, 2023). Treatment is focussed on establishing a patent airway if upper airway tract obstruction persists. Furosemide and intranasal oxygen are frequently used; however, as the increase in hydrostatic pressure triggering pulmonary oedema resolves quickly once an airway is established, the need for diuretics has been questioned. Diuretics decrease the hydrostatic pressure, and therefore driving force, for development of pulmonary oedema but do not aid in clearing already existing pulmonary oedema. As the alveolar epithelium usually remains intact, inhaled or systemic beta-adrenergic drugs such as albuterol or clenbuterol might increase the speed of fluid clearance from the alveoli (Frank et al., 2000; Zanza et al., 2023). Providing the upper airway tract obstruction can be resolved, most horses improve rapidly over 12–48 h and recover completely.

Other, rarer forms of pulmonary oedema include *re-expansion pulmonary oedema*, *neurogenic pulmonary oedema* and *ARDS*. *Re-expansion pulmonary oedema* can occur when previously atelectic lung is rapidly re-inflated, for example with rapid drainage of pleural fluid or evacuation of pleural air in a pneumothorax. The exact

pathophysiology is incompletely understood but increased capillary leakage due to trauma to the pulmonary capillaries, changes in hydrostatic forces and ischaemia-reperfusion injury have all been suggested. In horses, only a few cases have been reported. Two horses experienced pulmonary oedema in recovery after general anaesthesia and the authors suspected that re-inflation of the dependent, atelectic lung and intraoperative hypoxaemia might have contributed (Ball & Trim, 1996; Day et al., 1993). A third case developed pulmonary oedema after tissue plasminogen activator instillation into the pleural space of a horse with pleuropneumonia to dissolve restrictive fibrinous pleural adhesions. This might have triggered rapid expansion of the previously collapsed and adhered lung (McGorum et al., 2018). *Neurogenic pulmonary oedema* is a syndrome that shares many features with ARDS and both are examples of permeability pulmonary oedema. Neurogenic pulmonary oedema affects approximately 1% of human patients with traumatic brain injury and has been reported in small animals, but not horses. Again, the pathophysiology is incompletely understood: localised brain ischaemia is thought to activate trigger zones, leading to a massive increase in α-adrenergic discharge. Sudden pulmonary vasoconstriction and increased permeability then result in extravasation of protein-rich fluid and intra-alveolar haemorrhage. *Acute respiratory distress syndrome* is a syndrome of severe pulmonary dysfunction, caused by a widespread uncontrolled inflammatory reaction in the lungs triggered by intra- or extrapulmonary disease. In horses, it is best described in foals secondary to viral or bacterial pneumonia (Dunkel et al., 2005; Lakritz et al., 1993) while reports in adult horses remain sparse. It has been reported following near drowning, smoke inhalation, influenza and experimentally after injection of perilla mint ketones and gram-negative sepsis (Austin et al., 1988; Begg et al., 2011; Humber, 1988; Kemper et al., 1993; Schmidbauer et al., 2004; Sembrat et al., 1978). Cases require intensive treatment of the underlying cause and secondary respiratory failure including intranasal oxygen and possibly systemic corticosteroids.

Acute severe equine asthma

Equine asthma is characterised by the triad of pulmonary inflammation, bronchoconstriction and increased mucus production. During an acute asthma attack, the clinical signs of respiratory distress are largely secondary to severe bronchoconstriction. Emergency treatment, therefore, aims to relieve the immediate bronchoconstriction while longer term management changes and anti-inflammatory therapy decrease pulmonary inflammation which in turn will decrease the drive for bronchoconstriction and mucus accumulation. As immediate rescue medication, systemic or inhaled fast-acting bronchodilators in combination with corticosteroids and removal of offending antigens are indicated. Albuterol/salbutamol 540 mcg/6 puffs have a rapid onset of action within 5 min but the effect only lasts 30–60 min. Salmeterol has a slower onset of action of up to 60 min but remains effective for approximately 6 h. The expected clinical effect of both drugs is difficult to predict as many studies focus on improvement of pulmonary function, rather than clinical scores (Calzetta et al., 2020) and most study subjects are not in respiratory distress. Salmeterol achieved a decrease in clinical score from 6 to 4 at 30 min (with 2 being normal and 8 the worst score with severe nostril flaring and abdominal breathing effort) (Henrikson & Rush, 2001), highlighting that a temporary partial improvement, but not return to normal limits, might be a realistic expectation. Atropine (0.02 mg/kg i.v.) has long been considered one of the most potent bronchodilators in horses. It has been used as rescue medication and for diagnostic purposes but due to its systemic side effects including tachycardia, arrhythmia, pupillary dilation and colic, its use is limited to these indications (de Lagarde et al., 2014). N-butylscopolammonium bromide (Buscopan®) 0.3 mg/kg i.v. causes an improvement of some measures of respiratory function within 2 min of administration comparable to atropine but this is short-lived (30–60 min). The improvement might be difficult to detect when relying on clinical parameters such as respiratory rate, effort and nostril flare alone to judge the response (Couetil et al., 2012; de Lagarde et al., 2014). Arterial hypoxaemia does not improve with either drug (de Lagarde et al., 2014). An underwhelming immediate response is likely due to persistent mucus accumulation, inflammation and airway smooth muscle remodelling, highlighting the need for concurrent anti-inflammatory treatment (de Lagarde et al., 2014). While a positive response to trial treatment with a bronchodilator confirms the presence of bronchoconstriction, a negative or equivocal immediate response does, therefore, not rule out asthma. Systemic corticosteroids, particularly dexamethasone at 0.05–0.1 mg/kg q 24 h i.v. or i.m., are still the most effective drug in improving lung function, even if environmental management remains suboptimal. However, the effects are not immediate, and it takes 3–7 days until the full treatment impact can be appreciated (Leclere et al., 2010; Robinson et al., 2002). Inhaled fluticasone and ciclesonide are less effective than systemic dexamethasone in the treatment of acute asthma exacerbation (Lavoie et al., 2019; Robinson et al., 2009) and systemic corticosteroids are recommended in the initial treatment of acute severe asthma. In people, administration of intranasal oxygen is recommended alongside bronchodilators. During bronchoconstriction, poorly ventilated areas of the lung undergo hypoxic vasoconstriction. When administering inhaled β_2 agonists, systemic absorption via the bronchial tree, the oropharynx, and possibly, the upper gastrointestinal tract can reverse this protective vasoconstriction, potentially worsening ventilation: perfusion mismatch and hypoxaemia (Bush & Griffiths, 2017). However, this is not a frequently recognised problem in horses and should not prevent veterinarians from using bronchodilators if no oxygen is available.

Pleural effusion

Pleural effusion is a less common cause of acute respiratory distress as accumulation of a significant volume is required before respiratory compromise is noticed. However, horses with infectious pleuropneumonia induced by long-distance travelling often experience

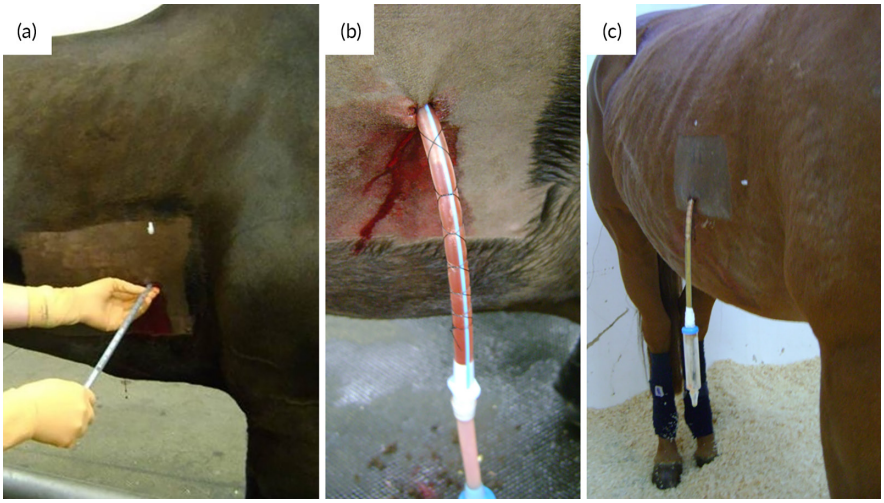

FIGURE 4 To drain a pleural effusion, a thoracic drain is inserted through a skin and muscle incision at the cranial edge of the rib (a) into the pleural space and secured with a purse-string suture and roman sandal pattern (b) before a Heimlich valve is attached (c) to prevent aspiration of air.

marked systemic inflammation with tachycardia and tachypnoea and the overall picture is very much that of acute distress. In countries where long-distance travel is common, such as the USA and Australia, pleuropneumonia is the most common cause of pleural effusion while in the United Kingdom, almost 40% of cases are secondary to neoplasia (Johns et al., 2017). Haemothorax is very rare but can occur after trauma, rodenticide or warfarin toxicity or, very rarely, systemic phenylephrine administration for the treatment of nephrosplenic entrapments or haemangiosarcoma (Frederick et al., 2010). Although a diagnosis can sometimes be made by auscultation alone, ultrasonography is essential to determine the extent and character of the effusion, assess concurrent pulmonary damage and choose the best location for thoracocentesis. There are no strict guidelines when drainage of pleural fluid is necessary to alleviate distress. Thoracocentesis might be indicated for diagnostic and therapeutic purposes if the fluid volume is greater than 3 cm in depth (Davis, 2018). It is prudent to place an intravenous catheter prior to the procedure and consider administration of IV fluids if drainage of a large volume is anticipated as rapid drainage can cause hypotension and re-expansion pulmonary oedema in people. Both complications appear to be rare in horses despite the fact that sometimes very large volumes, up to 30–96 L, are recovered (Arroyo et al., 2017; Camacho-Luna et al., 2021; Johns et al., 2017). To the author's knowledge, only one case of re-expansion pulmonary oedema has been reported in horses and this was in association with tissue plasminogen activator administration (McGorum et al., 2018). Large volume thoracic effusions are more common in cases of thoracic neoplasia than infectious pleuropneumonia and the volume and character of the fluid can be helpful in distinguishing both (Johns et al., 2017). The tube should be placed on the side with the larger effusion first, as ventral as possible and at the cranial edge of the rib while still avoiding the heart, diaphragm and lateral thoracic vein. Frequently, this is just above the costochondral junction of the seventh or eighth intercostal space (one hand's width caudal to the olecranon and one hand's width above the lateral thoracic vein). If extensive fibrin accumulation is present, aiming for a large fluid pocket might achieve the best drainage. After clipping and sterilely preparing, the site is infiltrated with 10–20 mL of a local anaesthetic before a stab incision is made with a scalpel blade through the skin and muscle. A 16 – 32 Fr blunt trocar is then pushed through the intercostal muscles which might take considerable force (Figure 4a). Holding the trocar firmly close to the tip avoids accidentally puncturing the lung. Once in the thoracic cavity a sudden release of resistance is often felt and fluid can be seen exiting between stylet and trocar. The trocar is then removed, a Heimlich valve or punctured condom secured at the end to avoid air aspiration and the tube is secured in place with a purse-string and Roman sandal suture (Figure 4b,c). Additional treatment with antimicrobials, anti-inflammatories and intranasal oxygen is also required.

CONCLUSIONS

History and physical examination can often give sufficient clues to identify the cause of respiratory distress and initiate emergency treatment. Upper respiratory tract obstruction, tension pneumothorax and occasionally pulmonary oedema can be rapidly fatal and require immediate intervention.

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No conflicts of interest have been declared.

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