Bruce McGorum BSc, BVM&S, PhD, CEIM, DipECEIM, FRCVS

University of Edinburgh
United Kingdom

bruce.mcgorum@ed.ac.uk

MANAGING EQUINE ACUTE LOWER AIRWAY DISEASE

Causes of acute LRT dysfunction

- Severe asthma (heaves, summer pasture asthma)
- Pulmonary oedema
- Bronchopneumonia
- Eosinophilic pneumonia
- Interstitial lung disease
- Near drowning
- Smoke inhalation
- Neoplasia
- Anaphylaxis
- Pulmonary thromboembolism

Assessment of the breathing pattern can yield valuable diagnostic information. Most dyspnoeic horses have an increased rate and depth of breathing. Slow deep breathing is a concern because it suggests severe URT or LRT obstruction, when airflow is so severely restricted that the breathing rate cannot be elevated. Fast shallow breathing occurs with conditions which make breathing painful (eg pleuropneumonia), restrictive or interstitial lung diseases (eg pulmonary oedema) and pleural effusion/ pneumothorax. Horses which have dynamic collapse of intrathoracic airways (eg asthma) have dyspnoea predominantly during expiration, resulting in a prolonged and laboured expiratory phase, and exaggerated expiratory contraction or 'heave' of the abdominal muscles. In contrast, inspiratory dyspnoea results in a prolonged and laboured inspiratory phase and exaggerated activity of the intercostal muscles during inspiration. In both types of dyspnoea, the nostrils are usually dilated throughout the breath cycle.

Investigation may also require endoscopy, cytology and bacteriology of airway secretions, thoracic radiography and ultrasonography, arterial blood gas analysis and/or pulmonary biopsy. Management of acute LRT disease includes removal of inciting causes (eg organic dusts, allergens), bronchodilators, anti-inflammatory drugs, intranasal oxygen, frusemide, antimicrobials and salbutamol.

Asthma; the most common cause of acute LRT dysfunction is readily recognised when severe. However, as a consequence of increased client awareness, cases are increasingly presented early in the disease course, when they have only occasional coughing or mild exercise intolerance, and before they develop overt expiratory dyspnoea. Consequently fewer cases are now diagnosed confidently from history and clinical findings alone, and there is increased reliance on the use of ancillary diagnostic techniques. Controlled exposure to hay/straw may be done to exacerbate clinical signs and thereby aid diagnosis of mild cases. Asthma is typically effectively managed by removal of inciting organic dusts and allergens, administration of bronchodilators and glucocorticoids, and if severe, intranasal oxygen supplementation. Failure of asthmatic horses to respond adequately may indicate 1) incorrect diagnosis – consider other disorders such as equine multinodular pulmonary fibrosis, 2) inadequate dust/allergen control – this is common - visit and assess environment and management, 3) failure of bronchodilator therapy – B_2-agonists provide a clinically evident benefit effect only in some horses - consider assessing the response to atropine – and note that airway obstruction may be due to mechanisms other than bronchoconstriction, such as mucus plugging and airway oedema, or 4) irreversible airway remodelling including bronchiectasis, but significant pulmonary fibrosis and emphysema are not thought to occur.

Interstitial lung diseases are poorly understood and likely under-diagnosed in horses. These disorders reduce lung compliance, causing inspiratory dyspnoea (fast shallow breathing pattern, with increased inspiratory effort and inspiratory duration). Fine crackles, especially at the end of inspiration, and wheezes are common. Breath sounds may be inaudible in severe cases. Weight loss, exercise intolerance and fever are common, while nasal discharge and coughing are usually minimal. Pulmonary hypertension, cor pulmonale, pulmonary fibrosis and cyanosis may result.
Arterial blood gas analysis reveals severe hypoxaemia, reduced oxygen saturation and often hypercapnia. By definition, a diagnosis of acute lung injury (ALI) is based on PaO_2/FiO_2 <300 mmHg and a diagnosis of acute respiratory distress syndrome (ARDS)

based on PaO2/FiO2 <200 mmHg. When horses are breathing room air (assuming FiO_2 = 0.21), ALI and ARDS are diagnosed when PaO_2 <63mmHg and PaO_2 <42, respectively. Ultrasonography may reveal multiple coalescing comet-tail or ring-down artefacts. Endoscopy may reveal frothy or viscous and highly proteinaceous fluid in the airways. Radiography may reveal an interstitial or mixed interstitial and alveolar pattern and nodules/granulomas. Haematology and clinical chemistry are usually indicative of non-specific inflammation. Aggressive treatment must be initiated early in the acute inflammatory phase, to prevent acute respiratory failure and the subsequent development of irreversible pulmonary fibrosis. Putative causal agents should be eliminated. Dexamethasone, pentoxyfylline, NSAIDs, antimicrobials may be indicated. If there is pulmonary oedema, frusemide, salbutamol, colloid support and judicious fluid therapy are indicated. Inhaled frusemide, which attenuates bronchospasm and exerts anti-inflammatory and immunomodulatory effects by decreasing cytokine production, has yielded mixed results based on clinical impression. As bronchoconstriction is typically not an important component of the pathology, bronchodilators are rarely beneficial, although β-adrenergic agonists may aid clearance of oedema. Supplemental oxygen is indicated in the acute stage, but severe hypoxaemia may be minimally responsive to oxygen therapy, indicating significant intra-pulmonary shunting.

Other causes of pulmonary oedema;

- Acute URT obstruction
- Cardiac failure
- Smoke inhalation
- Neoplasia
- Infectious agents
- Toxic reactions
- Immune mediated & anaphylaxis
- Endotoxaemia and systemic inflammatory response syndrome
- Pulmonary embolism
- Volume overload
- Idiosyncratic drug reactions
- Idiopathic

Haemorrhagic pulmonary infarction & necrotising pneumonia; affected horses have acute haemorrhagic pulmonary infarction, necrotising pneumonia, pulmonary thromboembolism and suppurative haemorrhagic pleural effusion. This aetiology of this syndrome is not determined, but many cases followed strenuous exercise and the pathology resembled that of porcine *Actinobacillus pleuropneumoniae* infection. Actinobacillus spp. and *Streptococcus equi* var. *zooepidemicus* were cultured from some affected horses. The syndrome has a poorer prognosis than conventional pleuropneumonia. Management included pleural drainage, pleural lavage, broad spectrum antimicrobials, NSAIDs, oxygen and fluids.

Pulmonary thomboembolism; Massive pulmonary infarction due to thromboembolism appears to be an uncommonly recognised, probably under-diagnosed, but important complication of many serious medical and surgical disorders. It may present as sudden onset severe respiratory distress and tachypnoea, and less commonly with coughing, epistaxis and haemoptysis. This complication has a guarded prognosis, although some affected horses have survived.