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EMERGENCY MANAGEMENT OF EQUINE ACUTE UPPER AIRWAY OBSTRUCTION

Acute URT obstruction is easily recognised by the characteristic **inspiratory stridor**, prolonged and laboured inspiratory phase, exaggerated intercostal muscle activity, nostril dilation and possible cyanosis. Most URT obstructions result from dynamic inspiratory airway collapse. Airway narrowing increases airflow velocity at the point of obstruction, further decreasing intraluminal pressure and exacerbating the obstruction (Bernoulli principal). Importantly, a moderate reduction in airway diameter can have a marked effect on resistance, because of the following equation;

Resistance = 8 x viscosity of air x length of airway / radius of airway[4]

Causes of acute URT obstruction

- Nostrils - bilateral nostril paralysis (CNVII)
- Nasal cavity – trauma, venous and lymphatic congestion (prolonged low head carriage, prolonged general anaesthesia with low head position, bilateral jugular thrombosis, cranial thoracic mass, Horner's syndrome), oedema (snake and insect bites, allergies, smoke inhalation, purpura haemorrhagica), neoplasia, sinus cysts, nasal surgery
- Pharynx – oedema, retropharyngeal lymph node abscess, cellulitis, neoplasia, HYPP
- Larynx – trauma, oedema, bilateral laryngeal paralysis, arytenoid chondritis, acute epiglottitis, laryngeal surgery, HYPP, hypocalcaemia
- Trachea – trauma, tracheal collapse, masses

General management; Emergency temporary tracheotomy is often required to stabilise the horse prior to investigating the aetiology and managing the underlying cause. Extreme care must be taken to avoid further stress. Sedation should be avoided if safety permits. If the horse collapses, a patent airway may be more readily achieved by insertion of an orotracheal tube than tracheotomy. Insertion of a tracheostomy tube should immediately abolish the stridor and relieve respiratory distress.
If respiratory distress continues, secondary pulmonary oedema/haemorrhage or airway obstruction distal to the tracheostomy should be considered. Airway oedema may warrant administration of dexamethasone (0.1 mg/kg IV) or epinephrine (3-10ml of 1:1000 epinephrine /450 kg IV, IM or SC). Calcium and dextrose may be indicated for hypocalcaemia and hyperkalaemic periodic paresis (HYPP), respectively. Elevation of the head is important to alleviate oedema and congestion.

Bilateral laryngeal paralysis associated with hepatopathy; Hepatopathy is a common cause of bilateral laryngeal paralysis, particularly in ponies. Affected horses are typically presented for investigation of suspected primary URT obstruction because of the inspiratory stridor. Endoscopy reveals total bilateral paralysis, with passive adduction of arytenoids towards the midline during inspiration. Laryngeal dysfunction may be temporary, resolving with restoration of hepatic function. The pathogenesis is unknown, but likely reflects neuromuscular dysfunction. Histopathological lesions not have been identified in laryngeal muscles, recurrent laryngeal nerve or other peripheral nerves of affected horses.

Post-anaesthesia laryngeal paresis; Unilateral or bilateral, total laryngeal paralysis is a rare, but potentially rapidly fatal complication occurring during recovery from general anaesthesia. Proposed predisposing factors include dorsal recumbency with neck hyperextension, dependent head position, pre-existing recurrent laryngeal neuropathy (RLN), large body size, prolonged duration of anaesthesia, hypotension, hypoventilation, hypoxaemia, post-anaesthetic myopathy and laryngeal dysfunction associated with xylazine administration. Excessive extension of the head and neck may cause hypoxic injury to the recurrent laryngeal nerve by occluding the vasa nervorum, or less likely neural stretch injury. Alternatively, nerve injury may reflect compression of the recurrent laryngeal nerve between the endotracheal tube and the vertebrae. Affected horses develop an acute-onset, life-threatening laryngeal obstruction, with inspiratory dyspnoea, stridor and cyanosis. Extreme distress makes them difficult and dangerous to handle, and the condition can be rapidly fatal. Laryngeal obstruction is usually noted immediately following extubation, when the horse is encouraged to stand during recovery, or during vocalization when the horse is walking to the stall following recovery. Treatments include emergency tracheal reintubation via oral or nasal cavities, placement of a temporary tracheostomy tube, supplemental intranasal oxygen, and anti-inflammatory drugs. Pulmonary oedema/haemorrhage may develop

despite prompt restoration of a patent airway. Horses which survive the acute obstruction have variable recovery of laryngeal function; some horses show complete resolution within 24h, while others have permanent laryngeal dysfunction. Neck hyperextension during anaesthesia should be avoided and an emergency tracheostomy kit should be available when recovering horses.

Hyperkalaemic periodic paresis; HYPP is a generalised myasthenic disorder that often presents with episodic URT obstruction. The nature of airway obstruction is variable, with laryngeal spasm or paralysis occurring in approximately 50% of cases. Appropriate medical treatment may ameliorate the severity and frequency of URT dysfunction associated with this disorder.

Smoke inhalation; In the acute phase (first 36h), thermal and chemical injury may cause oedema, congestion and necrosis of the URT and LRT, bronchoconstriction, lung parenchymal injury and degradation of surfactant. URT oedema and necrosis may necessitate emergency tracheotomy. Endoscopy is the most sensitive indicator of URT smoke inhalation injury. Serial blood gas analysis allows monitoring of respiratory function and carboxyhemoglobin concentrations should be determined to detect CO toxicity. Trans-tracheal lavage bacteriology may identify opportunistic pathogens. Intranasal oxygen supplementation facilitates removal of CO and improves pulmonary gas exchange. Consequences of pulmonary injury may be ameliorated using bronchodilators and frusemide. Corticosteroids have been used successfully in horses with smoke inhalation injury to combat inflammatory pulmonary oedema and maintain surfactant production. However, the immunosuppressive effects of corticosteroids may predispose patients to bacterial bronchopneumonia and studies in humans and animals with smoke inhalation have failed to identify therapeutic benefit of corticosteroids. NSAIDs may be an alternative to corticosteroids to reduce pulmonary inflammation. DMSO has proven therapeutic benefit in some smoke inhalation studies. Intravenous fluid therapy and oncotic support are essential components of treatment in human patients with severe pulmonary injury and cardiovascular shock, but must be done judiciously to strike a careful balance between maintaining adequate plasma volume to counteract shock without exacerbating pulmonary oedema. Prophylactic antibiotic therapy is controversial, since it can lead to the selection of resistant bacteria and has not reduced mortality in humans with smoke inhalation. Nonetheless, most equine clinicians consider administration of prophylactic antibiotics standard care in horses with severe pulmonary injury.

Pulmonary oedema after URT obstruction; Severe URT obstruction may induce acute life threatening pulmonary oedema, with or without concomitant pulmonary haemorrhage. This complication may further compromise gas exchange, and must be addressed when treating URT obstructions.