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REVIEW ARTICLE

Review of hypoxaemia in anaesthetized horses: predisposing factors, consequences and management

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Abstract

Objective To discuss how hypoxaemia might be harmful and why horses are particularly predisposed to developing it, to review the strategies that are used to manage hypoxaemia in anaesthetized horses, and to describe how successful these strategies are and the adverse effects associated with them.

Databases used Google Scholar and PubMed, using the search terms horse, pony, exercise, anaesthesia, hypoxaemia, oxygen, mortality, morbidity and ventilation perfusion mismatch.

Conclusions Although there is no evidence that hypoxaemia is associated with increased morbidity and mortality in anaesthetized horses, most anaesthetists would agree that it is important to recognise and prevent or treat it. Favourable anatomical and physiological adaptations of a horse for exercise adversely affect gas exchange once the animal is recumbent. Hypoxaemia is recognised more frequently in horses than in other domestic species during general anaesthesia, although its incidence in healthy horses remains unreported. Management of hypoxaemia in anaesthetized horses is challenging and often unsuccessful. Positive pressure ventilation strategies to address alveolar atelectasis in humans have been modified for implementation in recumbent anaesthetized horses, but are often accompanied by unpredictable and unacceptable cardiopulmonary adverse effects, and some strategies are difficult or impossible to achieve in adult horses. Furthermore, anticipated beneficial effects of these techniques are inconsistent. Increasing the inspired fraction of oxygen during anaesthesia is often unsuccessful since much of the impairment in gas exchange is a direct result of shunt. Alternative approaches to the problem involve manipulation of pulmonary blood away from atelectatic regions of the lung to better ventilated areas. However, further work is essential, with particular focus on survival associated with general anaesthesia in horses, before any technique can be accepted into widespread clinical use.

Keywords anaesthesia, horses, hypoxaemia, management.

Introduction

For equine anaesthetists, minimising patient mortality and morbidity remains a priority, and it is important to identify the causes to reduce the risk associated with general anaesthesia. In healthy horses, mortality varies between reports but can be as high as 0.9% (Young & Taylor 1993; Johnston et al. 2002; Bidwell et al. 2007; Dugdale et al. 2016). In animals with systemic disease, death rates are much higher (Pascoe et al. 1983; Johnston et al. 2002). General risk factors include duration of anaesthesia with cumulative effects of hypotension, hypoxaemia and acid-base derangements (Johnston et al. 2002).

Since impaired oxygenation can be detrimental, evaluation of the available evidence is warranted. In horses, general anaesthesia is frequently accompanied by impairment of pulmonary function and resultant low arterial oxygen tension or partial pressure (PaO_2), which is challenging to treat (Nyman & Hedenstierna 1989; Nyman et al. 2012), although its incidence in the general horse

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population is unreported. In horses anaesthetized for colic surgery, the incidence of hypoxaemia [using a definition of PaO_2 less than 80 mmHg (10.7 kPa)] has been reported to be as high as 13% (Pascoe et al. 1983).

Currently, evidence that hypoxaemia [defined as a PaO_2 of <60 mmHg (8.0 kPa)] occurring under general anaesthesia is harmful is sparse. The purpose of this review is to present the available evidence describing the consequences of hypoxaemia, to summarise why horses are predisposed to developing hypoxaemia and to appraise treatment strategies used to manage hypoxaemia in horses during general anaesthesia. Databases searched were Google Scholar and PubMed using the following search terms: horse, pony, exercise, anaesthesia, hypoxaemia, oxygen, mortality, morbidity and ventilation perfusion mismatch.

Physiological effects of hypoxaemia

Circulatory effects of experimentally-induced hypoxaemia in anaesthetized horses have been described (Steffey et al. 1992; Whitehair et al. 1996). During halothane anaesthesia, heart rate and cardiac output increase, but total peripheral resistance, arterial blood pressure and oxygen delivery decrease, regardless of the ventilatory mode (Steffey et al. 1992). During periods of hypoxaemia, circulatory function is worse during mechanical ventilation (Steffey et al. 1992), especially when halothane is used compared with isoflurane (Whitehair et al. 1996), and since global oxygen delivery is a function of cardiac output and oxygen content, a reduction in tissue oxygenation may occur.

Coronary blood flow increases by up to 35% in human volunteers subjected to arterial haemoglobin oxygen saturations of 70–75% (Grubbström et al. 1993). When the reserve is insufficient to meet demand, lactate is produced within the myocardium, which adversely affects metabolic, mechanical and electrical activities resulting in a fall in contractility and, therefore, output from the heart (Allen & Orchard 1987). It is logical to assume that similar effects occur within the equine myocardium during periods of acute hypoxaemia, although the coronary reserve of the horse is unknown.

Brain function depends on a continuous supply of oxygen, as neurons do not have the ability to store it for later use. Brain injury in the face of hypoxia occurs because of acidosis as a result of accumulation of lactic acid, intracellular accumulation of calcium, neurotoxic effects of excitatory neurotransmitters released in response to hypoxia and formation of reactive oxygen species (ROS) following reoxygenation (Hopkins & Bigler 2001). Humans exposed to hypoxic environments experience loss of coordination, blurred vision, weakness and dizziness, which mimics mild brain injury, and the metabolic demand of neuronal tissue can increase by up to 15% during tasks that require cognitive function (Turner et al. 2015). Altered cognition in horses recovering from anaesthesia is likely to impact upon recovery quality, although this has not been documented or investigated.

In humans, surfactant production within the lung is reduced or altered during periods of hypoxaemia (Vaporidi et al. 2005), and may contribute to the development of acute respiratory distress syndrome (ARDS). The effect of hypoxaemia on surfactant in the equine lung is not known.

Hypoxaemia reduces calcium reuptake and release in the sarcoplasmic reticulum of skeletal muscle, decreasing cross-bridge activation and force output, possibly through lactate and hydrogen ion accumulation, or free radical production (Romer et al. 2006). Muscle oxygenation is reduced during experimentally-induced hypoxaemia in anaesthetized horses (Portier et al. 2009), and hypoxaemia-induced muscle injury is worse when halothane is used to anaesthetize horses compared with isoflurane (Whitehair et al. 1996). These effects may have a significant impact on the ability of a horse to stand following anaesthesia.

Adequate delivery of oxygen to a wound is essential for optimal healing and resistance to infection in humans (Gottrup 2004). A low PaO_2 [<80 mmHg (10.7 kPa)] contributes to the development of surgical site infection in horses undergoing exploratory laparotomy (Costa-Farré et al. 2014). While wound breakdown and infection do not have direct relevance to anaesthetic risk, it may be linked to morbidity associated with perioperative hypoxaemia during general anaesthesia.

It might be interpreted from the information presented in the previous discussion that these deleterious effects of hypoxaemia on the brain, cardiovascular and pulmonary systems, muscle, and on wound healing and surgical site infection are partly responsible for the high morbidity and mortality associated with equine anaesthesia. However, the affinity of haemoglobin for oxygen is greater in horses compared with that in humans, making direct comparisons problematic (Clerbaux et al. 1993). Clearly,

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