OXYGEN DELIVERY AND ANAESTHESIA

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Elisa Bortolami, Emma Love look at complications during equine anaesthesia, such as hypoxaemia, as well as discussing causes and treatments of inadequate delivery

ONE of the most common complications during equine anaesthesia is hypoxaemia. This condition may be encountered both in field and hospital anaesthesia, although it is more easily recognised in a hospital setting where monitoring, such as pulse oximetry, blood pressure measurement and blood gas analysis, is available.

In equine anaesthesia, recumbency and commonly used drugs and techniques impair respiratory and cardiovascular function.

Hypoxaemia is defined as insufficient oxygenation of blood to meet metabolic requirement and is somewhat arbitrary when considered as an arterial oxygen partial pressure below 60mmHg to 70mmHg when breathing 100 per cent oxygen. Hypoxia is caused by low oxygen in the tissues and manifests as organ dysfunction and/or cellular damage[1].

Delivery of oxygen to the tissues during anaesthesia is important, as oxygen is the fuel for cellular aerobic metabolism and is required to convert biochemical energy from nutrients to adenosine triphosphate (ATP), which is used in many cellular processes.

The most serious consequence of inadequate tissue oxygenation is the acute cessation of vital organ function and death. A sustained, moderate degree of hypoxaemia during anaesthesia may cause organ dysfunction and play a role in post-anaesthetic complications[1], such as myopathies and fractures.

Horses become hypoxaemic during anaesthesia for several reasons. These include breathing air during anaesthesia “in the field” when anaesthesia is maintained with injectable agents, and, in theatre conditions, inhalant anaesthetics decrease respiratory rate, tidal volume, cardiac output and blood pressure. The peculiar anatomy of the horse’s diaphragm, which is dome-shaped and caudo-cranially slanted, means the weight of the abdominal organs compresses the lungs when the horse is positioned in dorsal recumbency. This is compounded further in cases of abdominal distension. In lateral recumbency, the uppermost lung tissue will compress the dependent lung. Other factors, such as body shape[2],[3], may affect the efficiency of oxygenation, as larger, round-bellied horses are more prone to develop hypoxaemia.

Ventilation-perfusion mismatch (V/Q) is a common consequence of these anatomical and physiological factors under anaesthesia. V/Q mismatch is defined as a regional imbalance between alveolar ventilation and pulmonary capillary blood flow, with areas in the lungs with good ventilation but insufficient perfusion, and areas with insufficient ventilation and good/ excessive perfusion. This causes impairment of gas exchange leading to hypoxaemia.

Physiology

The oxygen cascade describes the process of declining oxygen tension from atmosphere to mitochondria. Inspired oxygen is humidified in the upper airways, reaches the alveoli and then diffuses into the pulmonary capillaries where it is mainly bound to haemoglobin in red blood cells. A very small percentage is dissolved in the plasma. Cardiac output and regional blood flow then determines oxygen transport to the tissues. During these stages of transport, the oxygen tension declines from values of approximately 160mmHg (when breathing air, which is 21 per cent oxygen) to values of only 3mmHg to 4mmHg at the cellular level at a barometric pressure of 760mmHg. The first relevant drop in the partial pressure of oxygen happens in the alveoli (from 160mmHg to 100mmHg at barometric pressure and when the alveolar partial pressure of carbon dioxide is assumed to be 40mmHg), as dictated by the alveolar gas equation according to which $PA_{O2} = Fi_{O2}$ (PB-PH2O) – PAC_{O2}/R, where PA_{O2} is the alveolar oxygen partial pressure, PB is barometric pressure, P_{H2O} is the water vapour pressure, PA_{CO2} is the alveolar partial pressure of carbon dioxide and R is a constant – its normal value being 0.84.

As mentioned, the goal of anaesthesia is the delivery of oxygen to tissues (D_{O2}), which is calculated with the following equation: D_{O2} ml/ min = $COxCa_{O2}$, where CO is the cardiac output and CaO2 is the arterial content of oxygen. The arterial oxygen content can be calculated with the following equation: Ca_{O2} = (1.39x[Hb] xSa_{O2} %)+(PAO2x0.003) where [Hb] is the concentration of haemoglobin, SaO_2 % is the arterial oxygen haemoglobin saturation and PaO2 is the arterial partial pressure of oxygen – that is to say the oxygen dissolved in plasma, which contributes only minimally to CaO_2[5]. Combining these equations, we can understand how tissue hypoxia (inadequate D_{O2}) can exist, in the presence of normal PA_{O2}, if either [Hb] and SaO_2 % or CO are low.

Oxygen delivery is the critical process and it can be improved, ensuring optimisation of all points of the oxygen cascade. Unfortunately, in a clinical setting, tissue oxygenation cannot be measured directly as expensive monitoring devices and/or invasive techniques would be required.

Maintaining tissue oxygen delivery during anaesthesia

The first strategy to maximise oxygenation is to increase the inspired fraction of oxygen (Fi_{O2}). If the PA_{O2} is very low and nitrous oxide is being used, its administration should be stopped to increase the Fi_{O2}. Any leaks from the anaesthetic machine or breathing system should be fixed as these may result in the Fi_{O2} being lower than intended. Unfortunately, in practice, the situation is not that straightforward as most of the time oxygen is the sole carrier gas being used and several studies have demonstrated that when using higher concentration of inhaled oxygen absorption atelectasis and a worsening of V/Q mismatch happens[6],[7],[8], although this is controversial as a more recent study has reported different results[9].

Hypoventilation is conventionally defined as an arterial partial pressure of carbon dioxide (PA_{CO2}) higher than 45mmHg in conscious people. When breathing air, severe hypoventilation has an impact on PA_{O2} and a very high PA_{CO2} contributes to hypoxaemia as dictated by the alveolar gas equation $PA_{O2} = Fi_{O2} (PBP_{H2O}) – PA_{CO2}/R$. When breathing 100 per cent oxygen, hypercapnia affects Pa_{O2} to a minor extent. Moreover, during equine anaesthesia, a certain degree of hypercapnia is usually accepted (permissive hypercapnia), although the maximal acceptable level of hypercapnia has not been unanimously agreed[10].

Mechanical ventilation can improve ventilation and achieve better gas exchange, especially if instituted from the beginning of anaesthesia[10]; the main disadvantage is that it may cause significant depression of the cardiac function by decreasing venous return and, consequently, cardiac output, and indirectly by lowering the arterial partial pressure of CO_2, which provides a sympathetic drive in terms of increased systemic vascular resistance and an increase in heart rate[11],[12]. During intermittent positive pressure ventilation (IPPV), it is also fundamental to optimise inspiratory/expiratory ratio, where increasing inspiratory time may increase alveolar ventilation[10].

Many researchers have focused on minimising V/Q mismatch, which is considered the most important element in equine anaesthesia affecting arterial oxygenation, as has been shown with computerised tomography and morphological analysis studies[13]. According to these researchers, atelectasis seems the most likely cause of shunt and impaired arterial oxygenation.

Strategies aiming at decreasing atelectasis have been developed. The use of positive end expiratory pressure (PEEP) has been evaluated for decades in horses[14]. If PEEP is associated with IPPV, there is an increase in PaO_2, but if high airway pressures are used the consequent negative effects on cardiac output may outweigh the benefits of improving V/Q[15]. Promising results have been achieved in terms of improved arterial oxygenation by applying the so-called “recruitment manoeuvre” associated with IPPV with constant PEEP[16]. A recruitment manoeuvre is

a ventilatory technique that uses a relatively high inspiratory pressure combined with end-expiratory pressure for a determined length of time, usually seconds, and the theory is that it reopens collapsed alveoli. However, cardiovascular compromise may be associated with this manoeuvre, so, although it may result in an increase in PA_{O2}, it may not increase tissue oxygen delivery.

In research settings, the pulsed delivery of nitric oxide during inhalation anaesthesia has been investigated and the results seem to be promising[17],[18]. In these experiments, nitric oxide was added to the inspired gas as a pulse, and increased PA_{O2} and decreased venous admixture were reported[18]. The theory behind it is nitric oxide-induced pulmonary vasodilation redistributes blood flow to well-ventilated areas of the lung, thus decreasing V/Q mismatch.

The administration of aerosolised salbutamol, a ?2-agonist, is apparently an effective technique for improving Pa_{O2} values in some hypoxaemic horses during inhalant anaesthesia, although this drug is not licensed for use in veterinary species[19]. Aerosolised salbutamol is administered via the endotracheal tube and an increase in PA_{O2} may be seen, although the mechanism of action is not fully understood and the effectiveness varies significantly between individual animals. Side effects such as tachycardia and sweating may also be noticed.

Another way to minimise hypoxia is improving the cardiac output by administering fluids and inotropes, when deemed necessary[10]. Cardiac output is not often routinely monitored in the clinical setting as limitations include expensive equipment and training. So at the moment, in clinical practice, blood pressure measurement is considered an indirect indicator of cardiac output, but this has limitations as arterial blood pressure equals cardiac output times peripheral vascular resistances. If blood pressure is within the normal limits, it does not mean the cardiac output is adequate; it may be that peripheral vascular resistances are high and this can impair oxygen delivery to tissues.

Dobutamine is a synthetic catecholamine, which exerts primarily cardiac ?1 receptor agonist action, but also cardiac and vascular ?2 receptor activation may be involved; therefore, it directly affects the contractility of the heart and increases its stroke volume. Dobutamine is not licensed for use in veterinary species. Its administration seems to be an effective treatment for hypotension in anaesthetised horses, not only under experimental conditions, but also in a clinical setting[20]. Dobutamine has been proved to increase hindlimb blood flow in anaesthetised horses due to local vasodilation[21], although the effects on microvascular perfusion are controversial and still to be elucidated[21],[22],[23].

In a study, acepromazine seemed to improve haemodynamic variables and arterial oxygenation during injectable anaesthesia in healthy normovolaemic horses breathing room air spontaneously, after tracheal intubation[24].

The concentration of haemoglobin plays a very important role and hypoxaemia can be caused by

anaemia. If anaemia is detected on a pre-anaesthetic examination it is important to optimise the condition of the horse, as far as possible, before inducing general anaesthesia. The shape and characteristic of the haemoglobin dissociation curve determines the uptake of oxygen in the lungs and its delivery to the tissues. Equine haemoglobin has a greater affinity for oxygen so that horses are more tolerant to low PaO_2 values than people[25].

Administration of oxygen is particularly important in the recovery period. In this phase, there is a transition from maximal respiratory support, especially in a hospital setting, when mechanical ventilation might have been used, to a state where the horse is unconscious and in lateral recumbency. It is then important to administer oxygen – a demand valve can be used to give occasional breaths if there is a delay in return to spontaneous ventilation. Once the endotracheal tube has been removed, the airflow through both nostrils should be checked to ensure it is adequate and the thoracic wall should be watched to look for paradoxical ventilation, which may indicate upper respiratory tract obstruction. The most common cause of this is oedema of the nasal passages and this can often be avoided by careful positioning of the head during anaesthesia. Otherwise, phenylephrine can be instilled in the nostrils before the recovery[10]. Phenylephrine is not licensed for use in veterinary species. Replacing the oral endotracheal tube or inserting a nasopharyngeal tube may be necessary. In horses at high risk of respiratory tract obstruction (surgery entering the nasal cavities) or aspiration of reflux (following colic surgery) the endotracheal tube can be secured in place until the horse is standing.

In summary, it is important to remember that maintaining sufficient oxygen delivery to tissues is the ultimate aim and this depends on arterial oxygen content and blood flow. Techniques used to improve the former often cause a decrease in the latter, but horses seem to be more tolerant to hypoxaemia than to hypovolaemia.

• Some of the drugs mentioned in this article are used under the cascade.

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