CPD article

Sedation and general anaesthesia of the portosystemic shunt patient

Sedation and general anaesthesia may be required in animals with confirmed or suspected portosystemic shunt for a variety of reasons, not limited to shunt attenuation, such as diagnostic, routine or emergency procedures. Veterinary surgeons should understand normal hepatic functions and processes in order to appreciate the implications of portosystemic shunts associated specifically with sedation and anaesthesia. The pathophysiological and physiological variations, and their effects on anaesthesia and sedation, are discussed, as is management of the peri-anaesthetic period, drug choice for sedation or premedication, induction and maintenance of anaesthesia and analgesia. Patient monitoring and problem solving are also discussed, in relation to situations commonly encountered in portosystemic shunt patients during anaesthesia and sedation.

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The liver is a multifunctional organ and accounts for 1.5–4% of total body mass (Garcia-Pereira, 2015). The liver receives approximately 25% of cardiac output (Self, 2016). The hepatic artery accounts for 30% of hepatic blood flow and 70% of the portal vein flow (Self, 2016). Portal venous blood provides 50-60% of the liver's oxygen supply (Self, 2016). It has a high capacity for autoregulation of blood flow and oxygen delivery (Lautt et al, 1985). A reduction in hepatic portal vein pressure is accompanied by hepatic arteriole dilation (Kock et al, 1972; Lautt et al, 1985), ensuring adequate blood and oxygen delivery (Mathie and Blumgart, 1983; Rocheleau et al, 1999).

Metabolic functions include gluconeogenesis, glycogenolysis, lipid and protein processing (Chambers et al, 2015), accompanied by generation of body heat (Self, 2016). Coagulation and anticoagulation factors are synthesised, activated and cleared, and fat-soluble vitamins, glycogen, minerals and blood are stored (Chambers et al, 2015). The liver also secretes and activates hormones, including antidiuretic hormone and insulin (Chambers et al, 2015). Immune functions include those carried out by Kuppfer cells, specialised macrophages, which phagocytose foreign material and parasites reaching the liver via the hepatic portal vein (Goff, 2015).

Many anaesthetics and analgesics are metabolised by the liver, usually in a two or three phase process. Molecules are rendered water soluble and then excreted in urine or via other routes. For a detailed description of this process readers are directed to other texts (Williams, 1972; Chambers et al, 2015).

What is a portosystemic shunt?

A portosystemic shunt connects the portal circulation to the central circulation, bypassing the liver (Watson, 2014a). The normal liver receives blood leaving the digestive tract via the hepatic portal vein. This blood is rich in the products of digestion and bacterial, viral and parasitic antigens and toxins (Goff, 2015).

In dogs (Watson, 2014b) and cats (Lipscomb et al, 2007), portosystemic shunts may be congenital (70%) or acquired, secondary to portal hypertension (Watson, 1997, 2014a). Extra-hepatic portosystemic shunts are abnormal vessels which connect the portal circulation to the azygous vein or caudal vena cava, and are most common in small breed dogs especially Cairn, Yorkshire and West Highland White Terriers as well as Maltese, Havanese and Miniature Schnauzers (Watson, 1997, 2014b). Intra-hepatic portsystemic shunts are the result of a failure of the closure of the ductus venosus (the foetal vessel which bridges the caudal vena cava and the umbilical vein) and are most commonly seen in larger breed dogs such as Golden Retrievers and Irish Wolfhounds (Watson, 1997). In felines, portosystemic shunts are most common in crossbreeds (Watson, 1997), although purebreeds including Persian, Himalayan and Siamese cats may be over represented (Watson, 2014a).

Pathophysiology and clinical findings

Chronic reduction in portal blood flow results in decreased delivery of oxygen and hepatotrophic factors (Watson, 2014a). In congenital portosystemic shunts, the liver tissue that is present is

usually normal but the organ is small, and lacks the capacity to maintain normal homeostasis and function (Watson, 2014a). The underlying pathology, which leads to the development of acquired portosystemic shunt, including chronic hepatitis, may lead to reduced hepatic mass and function (Watson, 2014b).

Clinical signs of portosystemic shunts are typically of neurological, gastrointestinal or urinary tract in origin (Winkler et al, 2003).

Hepatic encephalopathy is encountered in 80% of congenital portosystemic shunt patients (Watson, 1997; 2014a), and is caused by the shunting of ammonia rich blood into the systemic circulation (Rothuizen, 1993), though other mechanisms have been implicated. Readers are referred to Lidbury et al (2016) for a full review of hepatic encephalopathy. Following attenuation of portosystemic shunt, seizures associated with reduced circulating endogenous benzodiazepines may occur (Hardie et al, 1990; Aronson et al, 1997; Yool and Kirby, 2002). Conflictingly, human studies of hepatic encephalopathy have shown that endogenous benzodiazepines may be a precipitating factor (Grimm et al, 1988; Jones et al, 1993; Baraldi, 2009).

Gastrointestinal and urinary tract signs include vomiting, diarrhoea, urinary calculi, cystitis and polyurina-polydypsia (Winkler et al, 2003; Watson, 2014a, 2014b).

Recognised haematological and biochemical changes consistent with portosystemic shunt include anaemia (alterations in erythrocyte processing and iron metabolism), hypoglycaemia (reduced glycogen storage and abnormal carbohydrate metabolism) and decreased total protein and blood urea (reduced hepatic synthesis) (Watson, 1997; Watson, 2014a). Increased bile acids and increased ammonia may also be recognised (Watson, 2014a).

Patients with congenital portosystemic shunt have been reported to exhibit both a hypo- and hypercoagulable state (Kummeling et al, 2006; Toulza et al, 2006; Gow et al, 2012; Kelley et al, 2013). As such, the coagulation status of portosystemic shunt patients is ambiguous, but clinically, bleeding disorders are uncommon (Niles et al, 2001; Kummeling et al, 2006).

Animals presenting with these signs and laboratory findings should be considered to be at risk of having an un-diagnosed portosystemic shunt, and further investigation may be warranted.

Implications of portosystemic shunt specific to anaesthesia

Many anaesthetic and analgesic drugs undergo hepatic metabolism. A reduced rate of metabolism results in a prolonged and potentially unpredictable period of action. (Waterman and Kalthum, 1990; KuKanich et al, 2012; Dancker et al, 2020).

Hypoalbuminaemia may lead to a greater plasma concentration of un-bound, active drugs (Nicholson et al, 2000; Kiamanesh et al, 2013). Many drugs used in sedation and anaesthesia are highly protein bound, including propofol, (Cockshott et al,1992), non-steroidal anti-inflammatory drugs (NSAIDs) (Papich and Messenger, 2015) and opioids (Trivedi et al, 2011).

Albumin is the main determinant of plasma oncotic pressure, and hypoalbuminaemic patients (<2.0 g/dL) (Nicholson et al, 2000) with polyurina-polydypsia may be predisposed to perioperative volume depletion and hypotension. This may be compounded by elevated circulating bile acids (Self, 2016), nitric oxide and inflammatory mediators potentiating vasodilation and hypotension (Watson, 1997, 2014a). A smaller liver mass represents a smaller capacity for erythrocyte storage and smaller reservoir for systemic release during haemorrhage (Self, 2016).

Small, thin portosystemic shunt patients with reduced hepatic heat generation may be at greater risk of hypothermia (Grimm, 2015; Mosing, 2016). Consequences of hypothermia include a greater risk of drug overdose, drug-induced apnoea (Regan and Eger, 1967), prolongation of the recovery period (Pottie et al, 2007), increased wound infection (Kurz et al, 1996), delayed healing and perioperative shivering with patient discomfort and increased oxygen demand (Mosing, 2016).

Hypoventilation may occur in all anaesthetised patients due to drug-induced muscle relaxation and respiratory depression (Mosing, 2016). Hypoxaemia leads to decrease in hepatic blood flow (Self, 2016), further increasing the unpredictability of the metabolism and the animal's response to drugs (Dugdale, 2010).

In young portosystemic shunt patients, additional considerations regarding paediatric physiology must be considered, such as sympathetic immaturity, a dependence upon heart rate to maintain cardiac output, high metabolic rate and body surface area to volume ratio (Rigotti and Brearley, 2016). In normal dogs, the liver may not mature until 5 months of age (Rigotti and Brearley, 2016), so young animals may have an even further reduced capacity for drug metabolism as a result of their small, immature liver.

Hypoglycaemia may lead to neurological complications including lethargy, weakness (Egger, 2016), seizures and further delays to recovery (Watson, 1997; Rigotti and Brearley, 2016). It may also contribute to hypotension and bradycardia (Frier et al, 2011), and can potentiate hypothermia. A failure to recognise and manage hypoglycaemia complicates attempts to manage other problems encountered during sedation or anaesthesia.

Pre-anaesthetic patient management

Clinical examination should include accurate determination of body mass and assessment of the patient's circulating volume status. Haematology, biochemistry, electrolyte and clotting profiles should be evaluated where possible. It may not be necessary to evaluate clotting profiles before non-invasive diagnostic procedures.

Tacky mucous membranes, tachycardia, low arterial blood pressure and elevated plasma lactate levels may be indicators of inadequate circulating cardiovascular volume (Aukbrually, 2016). The use of 0.9% NaCl as a resuscitation or replacement fluid in these cases is inappropriate, as the high concentration of chloride ions relative to that of blood may contribute to metabolic acidosis (Aukbrually, 2016). Hartmann's solution contains lactate, the metabolism of which uses H^+ ions, sparing bicarbonate, leaving it available to act as a buffer within the plasma (Aukbrually, 2016). Plasma-Lyte®, if available, is similar in composition to Hartmann's solution and has the closest electrolyte composition, and osmolality, to that of human plasma (Weinberg et al, 2016). Lactate is replaced with acetate (which requires no hepatic biotransformation) as a bicarbonate precursor, so Plasma-Lyte® has been recommended as a replacement fluid in cases of severe

hepatic dysfunction (Aukbrually, 2016). In acutely hypovolaemic animals, administration of boluses of 3-5 ml/kg of replacement solution, over 10–15 minutes, followed by reassessment of volume status, should be considered. Care should be taken to monitor animals for evidence of fluid overload. Intra-operative fluid therapy should be planned, with the aim of maintaining a normal fluid balance. Previously, high rates (10 ml/kg/hr) of intra-operative fluid administration have been advocated (Davis et al, 2013). However, the positive fluid balance in these cases has been shown to lead to increased mortality and morbidity, so more conservative rates of 3 ml/kg/hr for cats and 5 ml/kg/hr for dogs are now advocated (Davis et al, 2013). Water should not be withheld before anaesthesia or sedation.

It is essential that hepatic encephalopathy is managed before anaesthesia and sedation, and readers are directed elsewhere for a full discussion of this matter (Lidbury et al, 2016).

Previously well managed hepatic encephalopathy may be exacerbated by drugs such as opioids, as well as inflammation (Lidbury et al, 2016), leading to decreased levels of consciousness and increased blood brain barrier permeability (Lidbury et al, 2016), necessitating the administration of reduced drug doses. Severe hepatic encephalopathy may result in cerebral oedema and brainstem herniation (Lidbury et al, 2016). Readers are directed to other texts discussing the management of raised intracranial pressure (Raisis and Musk, 2014).

Dogs treated prophylactically with levetiracetam (median dose 60 mg/kg/day orally) before portosystemic shunt attenuation were demonstrated to be less likely to develop post-operative seizures than control groups (Fryer et al, 2011), and the authors' institution adopts this approach. Levetiracetam is chemically distinct from other anti-epileptic drugs, and its mechanism of action is unique (Packer et al, 2015). It does not undergo hepatic metabolism (Patsalos et al, 2004) and is increasingly used as by anaesthetists as an anti-epileptic drug in the perioperative period in humans. Anti-epileptic drugs should not be stopped before anaesthesia, sedation or surgery (Posner, 2016). Treatment of seizure activity with phenobarbital may lead to synergistic effects with anaesthetic drugs, including propofol and alfaxalone, necessitating further reduction of doses (Posner, 2016). This is especially important during the first seven days of treatment, before the induction of hepatic enzymes (Posner, 2016).

Controversies exist surrounding pre-anaesthetic fasting, and in human paediatric anaesthesia long fasts are no longer advocated (Frykholm et al, 2018). Pre-anaesthetic fasting aims to increase the pH and reduce the volume of stomach contents, decreasing the risk of regurgitation and aspiration (Savvas et al, 2016). Prolonged fasting of portosystemic shunt patients may worsen anticipated hypoglycaemia. In healthy, adult dogs evidence surrounding pre-anaesthetic fasting is conflicting. In one study, a small meal 3 hours before surgery reduced the incidence of canine gastro-oesophageal reflux (Savvas et al, 2016), whereas another (Viskjer and Sjöström, 2017) showed a greater risk of gastro-oesophageal reflux following feeding 3 hours before anaesthesia compared to 18 hours before. There are no data surrounding perioperative fasting of cats (Robertson et al, 2018). At the authors' institution, paediatric patients are fed a small meal of wet food 3–4 hours before anaesthesia and blood glucose is monitored throughout the perioperative period. With any perioperative feeding regime, there still exists the risk of gastro-oesophageal reflux, regurgitation and aspiration.

Sedation and pre-medication

Opioids alone often provide adequate sedation or premedication for portosystemic shunt patients (Self, 2016). For patients undergoing surgery, 0.2–0.3 mg/kg methadone provides efficacious analgesia (Self, 2016). Sedation and analgesia may be more profound and prolonged than in healthy animals, therefore repeat or post-operative dosing should be based on assessment and pain scoring (Waterman and Kalthum, 1990). Pethidine is a full mu-agonist, licensed in the UK for use in dogs, typically at 3–5 mg/kg intramuscularly (IM). Its chemical structure is similar to that of atropine and clinically it exhibits anticholinergic effects (Latta et al, 2002). Its use has been recommended in portosystemic shunt patients (Dugdale, 2010). Pethidine may also be used to treat post-anaesthetic shivering in hypothermic patients (Mosing, 2016). The accumulation of the active metabolite of pethidine (norpethidine) has been associated with seizure activity, so repeated doses should be avoided (Golder et al, 2010).

Alpha-2 receptor agonists such as medetomidine and dexmedetomidine are reliable sedatives. Dexmedetomidine is the active enantiomer of medetomidine. Theoretically, administration of dexmedetomidine rather than medetomidine may lead to a reduced requirement for metabolism, as only the active enantiomer needs to be metabolised, which may result in predictable sedation and improved analgesia (Murell, 2016). Dexmedetomidine may provide analgesia for a longer duration than medetomidine, though its cardiovascular effects are similar, (Murrell and Hellebrekers, 2005) and clinically there may be minimal difference between the two drugs (Murrell, 2016). When sedation with an opioid alone is inadequate, a low dose of medetomidine 0.5–3 mcg/kg intravenously (IV) or IM, or dexmedetomidine 0.5–1.5 mcg/kg IV or IM, may be administered. The use of alpha-2 receptor agonists in premedication protocols enables a reduction in the dose of induction and maintenance agents and contributes to analgesia (Bloor et al, 1992; Lemke, 2004; Murell and Hellebrekers, 2005). Cardiovascular effects of alpha-2 receptor agonists are vasoconstriction, bradycardia and reduced cardiac output (Bloor et al, 1992; Lemke, 2004; Murrell and Hellebrekers, 2005), with maximal haemodynamic effects of medetomidine at 5mcg/kg IV (Pypendop and Verstegen, 1998). Reduced cardiac output and bradycardia may contribute to hypotension, particularly in very young animals. In the face of profound responses to medetomidine or dexmedetomidine, atipamezole may be used to partially antagonise cardiovascular effects, but this may also reverse potentially advantageous sedation and analgesia (Lemke, 2004). If bradycardia is considered to be the cause of reduced cardiac output, the administration an anticholinergic (glycopyrrolate, 2-10 mcg/kg, IV or IM) or in a life-threatening emergency, atropine (0.01–0.03 mg/kg IV) may be considered. This will lead to an increase in cardiac output through increased heart rate, thus avoiding antagonism of the analgesic effects of medetomidine or dexmedetomidine (Lemke,

2004). However, because of the uncertainties regarding the role of endogenous benzodiazepines in hepatic encephalopathy-related seizures, their use is not recommended in portosystemic shunt patients (Self, 2016).

Acepromazine has a long duration of action (4–8 hrs), undergoes extensive hepatic metabolism and has no reversal agent (Murrell, 2016). Acepromazine should not be used in portosystemic shunt patients as it may result in an excessively profound, long duration of action and prolonged hypotension (Murrell, 2016).

Induction of general anaesthesia

Induction of general anaesthesia should be preceded by placement of an indwelling intravenous cannula and preoxygenation. Propofol (Kraus et al, 2000) and alfaxalone (Berry, 2015) both undergo hepatic metabolism, though additional extra-hepatic extraction of propofol by the lung has been demonstrated (Matot et al, 1993). Propofol is highly protein bound (96–98%), which may lead to a greater plasma concentration of un-bound, active drug in hypoalbuminaemic patients (Nicholson et al, 2000; Kiamanesh et al, 2013), whereas alfaxalone is 30–50% protein bound (Maddison et al, 2008). Side effects of both include dose dependent post-induction apnoea (Muir and Gadawski, 1998; Muir et al, 2008; 2009), and hypotension caused by vasodilation (Goodchild and Serrao, 1989; Muir et al, 2008, 2009). These side-effects may be minimised by adequate pre-medication and slow administration. Alfaxalone may preserve or elevate heart rate, and so may help to maintain cardiac output in young animals (Muir et al, 2008, 2009). Propofol has been shown to cause oxidative damage to feline red blood cells, and significantly increased recovery times following use for three or more consecutive days in cats (Andress et al, 1995). Repeated propofol administration on successive days and in anaemic feline patients should be avoided. Small, sub-anaesthetic doses of alfaxalone (IV or IM) (Kim et al, 2015), or propofol (IV) (Glowaski and Wetmore, 1999), may be administered to provide adequate clinical restraint for quick procedures such as radiography or fine needle aspirate acquisition.

Ketamine, used as a co-induction agent (0.5–2 mg/kg IV) provides a dose sparing effect for other induction agents (Muñoz et al, 2017) and analgesic properties (Berry, 2015). In the case of portosystemic shunt patients, its increased duration of action may provide analgesia throughout surgical procedures. Ketamine's stimulation of the sympathetic nervous system may also help to maintain heart rate, cardiac output and vascular tone and subsequently, blood pressure (Haskins et al, 1985).

Maintenance of general anaesthesia

Maintenance of anaesthesia with a volatile anaesthetic agent reduces the risk of accumulation of drugs that may occur, should total IV anaesthesia be employed. Less than 0.2% of isoflurane undergoes biotransformation (Holaday et al, 1975), while 3% of sevoflurane undergoes hepatic metabolism (Egger, 1994). Isoflurane and sevoflurane both lead to dose dependent hypotension via vasodilation but at clinically relevant doses, hepatic blood flow may not be affected (Bernard et al, 1992; Frink et al, 1992). As such, there is little evidence to support the use of one agent over the other (Self, 2016). Neither agent possesses analgesic properties and so the increased delivery of volatile agent to produce adequate depth of anaesthesia in the face of noxious stimulation is no substitute for adequate analgesia.

Analgesia

Multimodal analgesia is the use of drugs with varying mechanisms of action on pain pathways, to maximise analgesia and minimise unwanted side effects (Kerr, 2016). This is particularly important in portosystemic shunt patients where a multimodal approach will reduce the likelihood of side effects, such as hypotension and hypoventilation, caused by excessive administration of induction or volatile anaesthetic agents. The serum concentration may be increased, and excretion impaired, of hepatically metabolised drugs including NSAIDs, (KuKanich et al, 2012; Ramsey, 2014) opioids (Waterman and Kalthum, 1990), local anaesthetics (Ramsey, 2014), alpha-2 receptor agonists, ketamine and paracetamol. This should not serve as a reason to withhold analgesia in portosystemic shunt patients.

The inclusion of opioids in premedication protocols will provide intra- and postoperative analgesia. Repeat administration in hospitalised patients should be based upon objective pain scoring scales, with a set intervention score rather than pre-specified dosing intervals. The Glasgow Composite Modified Pain Scale (Holton et al, 2001), with an intervention score of 5/24 or 4/20 in dogs and 5/20 cats, is favoured by the authors in these cases. Morphine epidural anaesthesia has been shown to provide safe and efficacious analgesia during recovery in dogs, following extra-hepatic portosystemic shunt attenuation (Dancker et al, 2020), and should be considered in portosystemic shunt cases with normal clotting profiles. Remifentanil (5–40 mcg/kg/hr IV) (Self, 2016), if available, may offer an ideal choice for intra-operative analgesia, as it is metabolised by plasma esterases, meaning the liver is not involved in its elimination and it does not accumulate after IV infusion (Allweiler et al, 2007). On account of its rapid elimination half-life of approximately six minutes (Hoke et al, 1997), remifentanil must be administered as an infusion and not bolus doses.

There is little data to indicate that animals with hepatic disease are at increased risk of NSAID hepatotoxicity, though NSAID administration may be associated with increased risk of gastrointestinal ulceration in hepatic animals (KuKanich et al, 2012). In portosystemic shunt patients with normal clotting profiles, and no other contraindications to their administration, NSAIDs may be administered at the lowest label doses with an increased interval between the administration of doses (Ramsey, 2014) (every 36 rather than 24 hours, if no alternatives exist). No precise dose adjustments have been determined, and NSAID use in portosystemic shunt patients should therefore be cautious, with owner consent (KuKanich et al, 2012) following a full discussion regarding the risks of administration.

Locoregional techniques may provide a useful alternative to systemically administered analgesic medications in portosystemic shunt patients (Dancker et al, 2020). Readers are directed elsewhere

for a full discussion regarding implementation of these modalities (Campoy et al, 2015; Duke-Novakovski, 2016). Commonly used veterinary local anaesthetics, other than procaine, are metabolised in the liver (Duke-Novakovski, 2016), so consideration should be given to the administered dose (Ramsey, 2014). Caution should be taken following the administration of local anaesthetics to monitor and manage side effects, such as hypotension caused by regional vasodilation, which may result from their use (Duke-Novakovski, 2016).

Paracetamol is a commonly used analgesic for post-operative pain in canine patients (Mburu et al, 1988), at doses of 10–20 mg/kg IV in healthy dogs (Serrano-Rodríguez et al, 2019). There is a paucity of evidence regarding the use of paracetamol in animals with hepatic abnormalities. A recent study in human medicine concluded paracetamol to be a safe first line analgesic for almost all liver disease patients (Hayward et al, 2016). At the authors' institution, paracetamol is administered to portosystemic shunt patients at 10 mg/kg every 12 rather than 8 hours. However, its use is strictly contraindicated in feline patients.

Monitoring of patients under sedation or general anaesthesia

The most important resources when monitoring patients under sedation or anaesthesia are the sense of the anaesthetist via 'hands-on' monitoring to include palpebral reflexes, eye position and jaw tone as well as pulse rate and quality, respiratory rate and depth and effort. Blood pressure should be monitored by oscillometric or doppler techniques during sedation and anaesthesia or invasively via arterial cannula and pressure transducer, if available, during general anaesthesia and surgery. In adult patients, mean arterial blood pressure should be maintained >80 mmHg or if using the Doppler technique systolic arterial blood pressure >100 mmHg (Haskins, 2015). Paediatric patients tend to have lower arterial blood pressures than adults and in these patients a mean arterial blood pressure of approximately 55mmHg is adequate (Brierley et al, 2009). Pulse oximetery provides a useful method of assessing the oxygenation status of arterial blood and the saturation of arterial haemoglobin with oxygen should be >95%. Electrocardiographic monitoring enables the early detection and management of cardiac arrhythmias of haemodynamic significance. Capnography provides a method of continuous monitoring of the respiratory system in relation to expired carbon dioxide. A normal partial pressure of end tidal carbon dioxide is 35–45 mmHg (Dugdale, 2007). Many anaesthetists will tolerate a partial pressure of expired carbon dioxide of up to 60mmHg as the sympathetic stimulation caused by mild hypercapnoea may provide beneficial sympathetic nervous system stimulation (Dugdale, 2007).

Problem solving

Close monitoring of patients will often allow problems to be managed before they become serious.

Alpha-2 receptor agonists and opioids may contribute to hypotension and hypoventilation. Sedation and cardiovascular effects of alpha-2 receptor agonists may be reversed by atipamezole (administered at five times the previously administered dose of medetomidine, or ten times that of dexmedetomidine IM). This will also result in the reversal of their analgesic properties (Lemke, 2004). Small doses (0.001–0.002 mg/kg IV) of naloxone may be used to reverse opioid associated thermoregulatory and central nervous system depression, leaving some residual analgesia (Bedneski, 2015)

Hypotension

Assess the patient's depth of anaesthesia. If possible, reduce volatile anaesthetic agent administration to minimise dose-related vasodilation (Bernard et al, 1992; Frink et al, 1992). If the depth of anaesthesia is inadequate following reduced volatile anaesthetic agent administration, ensure that adequate analgesia has been employed. Consider the administration of a fluid bolus (3–5 ml/kg of balanced poly-ionic solution over 10 minutes). The administration of anticholinergics should be considered if bradycardia is deemed to be the cause of reduced cardiac output and hypotension. If these measures fail, dopamine (5–15 mcg/kg/minute) or noradrenaline (6–12 mcg/kg/hr) infusions should be instituted. Ideally, when vasopressors are employed, blood pressure monitoring should be invasive via arterial cannulation and pressure transducer. If invasive blood pressure monitoring is not available, then the use of vasopressor or inotropes should be cautious and accompanied by frequent monitoring of blood pressure with a device validated for use in animals. Blood pressure should be continually monitored into recovery.

Hypoventilation

Most drugs administered during anaesthesia and sedation lead to respiratory depression. This should be monitored and controlled by reducing volatile anaesthetic agent administration, while ensuring adequate analgesia. If the partial pressure of end tidal carbon dioxide exceeds 60 mmHg under general anaesthesia, consider intermittent positive pressure ventilation (Dugdale, 2007). Oxygen supplementation via mask should be provided for sedated animals.

Hypothermia

Hypothermia is best prevented, so it is important to ensure adequate ambient temperature (Sessler, 1997). This can be done by minimising clipping and wetting during patient preparation and using heat pads, blankets or insulation to prevent heat loss (Armstrong et al, 2005). Caution should be taken with all methods of active warming, as they present a a significant risk of burning anaesthetised and heavily sedated animals who can't move away from the heat (Oncken et al, 2001).

Hypoglycaemia

Hypoglycaemia happens when blood glucose drops to less than 3.33 mmol/L (Nelson, 2014). To combat this, blood glucose should be monitored every 30–60 minutes throughout the peri-operative period and supplemented if necessary, until the animal is eating normally. Offer the animal food as soon as possible postoperatively. Adding 2.5 ml of 50% glucose solution to 450 ml of Hartmann's solution will produce a 2.5% glucose solution, which can be administered at once-twice maintenance rate intraoperatively. Acute hypoglycaemia may be treated with 0.5–1 ml/kg of 50% glucose solution, diluted 1:1 with 0.9% saline.

KEY POINTS

- In cases of portosystemic shunt of any aetiology, blood flows directly from the portal vein to the systemic circulation, bypassing the liver.
- In congenital portosystemic shunts, reduction in portal blood flow leads to a small liver, which is usually healthy but lacks the capacity to maintain normal function.
- Portosystemic shunt patients are at increased risk of hypoalbuminaemia and, under general anaesthesia or sedation, hypotension, hypoventilation, hypothermia and hypoglycaemia are associated with prolonged recovery.
- Careful attention should be payed to pre-anaesthetic assessment and stabilisation.
- Owing to reduced hepatic capacity for drug metabolism, many drugs used in anaesthesia and sedation may exhibit unpredictably profound effects, including unexpectedly deep sedation, alterations in onset of action and unpredictably extended durations of action. This necessitates careful consideration of drug choice, dose and dosing intervals, based on monitoring of the animal's responses and pain scoring.
- An appreciation of the anticipated complications and their causes during anaesthesia of portosystemic shunt patients enables their effective and timely management.

The recovery period is an extremely important part of the anaesthesia process, and the risk of complications or death during this period should not be underestimated (Brodbelt et al, 2008). Following sedation or anaesthesia, and upon entering the recovery period, animals should not be left unattended or unmonitored. If the recovery period will not be monitored by the attending anaesthetist, a formal handover of the animal and their care plan should take place with the recovery ward personnel. The recovery plan should include monitoring of vital parameters to ensure adequate ventilation and airway patency, circulatory parameters including blood pressure, body temperature, and level of consciousness and activity (Mosing, 2016). Blood glucose should be monitored and supplemented as above until the animal is eating normally. Frequent pain scoring (every 2–4 hours) should be implemented and a pain management plan formulated. Recovery ward personnel should be briefed to contact the attending veterinary surgeon with concerns regarding a prolonged recovery, or abnormalities in physiological parameters. A prolonged recovery may be a result of a reduced metabolism of drugs, hypoglycaemia and hypothermia (Waterman and Kalthum, 1990; Armstrong et al, 2005; Pottie et al, 2007; Mosing, 2016). Antagonism of drugs should be considered as above.

Conflicts of interest

The authors declare that there are no conflicts of interest

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