CPD article

Anaesthetic complications and emergencies preparedness part 2: intraoperative and recovery period complications

This is a straightforward reference guide designed to assist in diagnosing and treating anaesthetic complications and emergency situations in the perioperative period. Each complication is presented with its risk factors, signs for recognition and differential diagnoses when indicated. Management is presented in a step-by-step manner. Whenever possible, advice on how to prevent these situations from occurring is also provided. This second part focuses on complications more commonly observed in the intraoperative and postoperative period. It includes common situations, such as spontaneous/seizure-like movement or agitation; rare but life-threatening emergencies, such as malignant hyperthermia; and postoperative conditions less recognised in veterinary medicine, such as nausea and severe pain. The complications are presented in alphabetic order in each section (intraoperative and postoperative periods) for easy, rapid consultation.

https://doi.org/10.12968/coan.2019.0052

Stéphanie Marotto, DVM,HOPia Veterinary Clinic, Guyancourt, France; **Carl A Bradbrook** BVSc CertVA Dipl ECVAA MRCVS, Consultant Anaesthetist, Anderson Moores Veterinary Specialists, The Granary, Bunstead Barns, Poles Lane, Hursley, Winchester, Hampshire SO21 2LL; **Aurora Zoff** DVM Cert(AVP)VA Dipl ACVAA MRCVS, North Downs Specialist Referrals, 3 & 4 The Brewerstreet Dairy Business Park, Brewer St, Bletchingley RH1 4Q. marotto.stephanie@gmail.com

Key words: anaesthesia | cat | complications | dog | management | postoperative

Many complications may arise besides hypothermia, hypotension and hypoventilation in small animal anaesthesia. The aim of this article is to point out some critical situations and emergencies that may be encountered from premedication to recovery, and to provide advice on how to prevent, recognise and manage them. Anaesthetic complications are presented in the time frame in which they are most likely to happen. Part 1 of this 2-part series (Marotto et al, 2018) focused mainly on life-threatening emergencies involving the cardiovascular and respiratory system during the preoperative and intraoperative period, whereas this second part concentrates on other complications of the intraoperative and postoperative period. As the most recent multi-centre enquiry on small animal perioperative mortality (CEPSAF) identified the postoperative period as the one with the highest incidence of mortality (Brodbelt, 2009), a strong focus has been placed on the management of critical events that may happen during this time, such as variations of body temperature or blood pressure, and prolonged recovery. Immediate recognition of failing respiratory and renal systems, and how to deal with these life-threatening complications, is also presented.

Hyperthermia

Hyperthermia is a relatively uncommon complication of anaesthesia, with overall frequency of 1.4% in dogs and cats, according to McMillan and Darcy (2016). Thick-coated dogs are more at risk for this adverse event. A hot surgical environment and/or the use of a rebreathing system are both predisposing factors.

Clinical signs that can raise suspicion of hyperthermia are tachypnoea and hypercapnia. An oesophageal or rectal temperature >38.5°C provides confirmation.

Management

- Switch off any warming device.
- Provide 100% oxygen, increase fresh gas flow and consider hyperventilation
- If hypotension and depth of anaesthesia are not a concern, promote vasodilatation by slowly increasing the concentration of inhalant anaesthetic agent
- If hypotension is not a concern and there are no contraindications, administer acepromazine 0.01 mg/kg intravenously (IV) to promote vasodilatation (Adami et al, 2012)
- Place wrapped ice packs on the main accessible blood vessels

(jugular veins, femoral arteries) — not directly on the skin, as this risks causing skin damage and/or vasoconstriction.

Consider

- Infusing cold IV fluids. Crystalloids at 4 °C are used in humans to rapidly decrease body temperature and induce therapeutic hypothermia. A dose of 18±10 mL/kg of 0.9% saline infused over 10–20 minutes has been described in paediatric patients (Fink et al, 2012). A suggested starting dose in cats is 5 mL/kg and 10 mL/kg in dogs
- If a rebreathing system is being used, changing to a non-rebreathing system, if adequate fresh gas flow can be provided
- Abdominal (if the abdomen is open), gastric, colonic and/or bladder lavage with fluids at room temperature.

Local anaesthetic systemic toxicity (LAST)

Local anaesthetic systemic toxicity is an uncommon event, but has the potential to be fatal. It can occur subsequent to IV administration of any local anaesthetic, as they all have the potential to be neurotoxic and cardiotoxic; lidocaine is the only local anaesthetic that has a relatively wide margin of safety in dogs and is deliberately administered IV, mainly for analgesia and the treatment of ventricular arrhythmias. Overdosing of local anaesthetic while performing epidural, spinal or peripheral nerve blockade is another possible cause of LAST and may lead to clinical signs of intoxication. Furthermore, in cases of hyperkalaemia, hypoxaemia and acidosis, local anaesthetic cardiotoxicity is enhanced and LAST may occur.

Anecdotally, topical preparations containing the local anaesthetic benzocaine have been reported to cause methaemoglobinaemia in dogs. To the authors' knowledge, no such cases have been reported in cats, but it seems sensible to expect that this species may be more prone to develop this complication, as cats are more sensitive to local anaesthetic toxicity than are dogs.

Neurological clinical signs are the first signs to appear in cases of lidocaine systemic toxicity, but they may be masked by general anaesthesia. They include:

- Restlessness/excitation
- Tremors/twitching
- Tonic-clonic seizures
- Respiratory arrest.

When higher toxic doses of lidocaine are reached, cardiovascular compromise arises, with:

- Hypotension
- Cardiac arrhythmias
- Cardiac arrest.

Bupivacaine is much more toxic, and especially cardiotoxic, than lidocaine. Neurological signs such as seizures may not be visible under general anaesthesia or be very short-lived, but cardiac arrhythmias with severe hypotension appear rapidly and are seriously life-threatening.

Management

Prevention is paramount:

- Check drug dose and calculate based on lean body weight; respect local anaesthetic safe doses (*Table 1*)

Table 1. Safe doses of commonly used local anaesthetics

Drug	Maximum safe dose for local block		Maximum safe dose for epidural
	Dog	Cat	Dog and cat
Lidocaine	6–10 mg/kg	3–5 mg/kg	4 mg/kg
Ropivacaine	3 mg/kg	1.5 mg/kg	1 mg/kg
Bupivacaine	2 mg/kg	1–1.5 mg/kg	1 mg/kg
Modified from Rioja Garcia (2015)			

- If mixing different local anaesthetics, use the lower dose of the safe range as a maximum (as additive side effects are possible)
- If injecting in the subarachnoid space instead of the epidural space, the dose must be one tenth of the calculate dose for epidural injection (Rioja Garcia, 2015), or 0.05 mL/kg of bupivacaine 0.5% (Otero and Campoy, 2013)
- Check for the absence of blood before injecting by aspiration when performing a peripheral nerve blockade, or by looking at the spinal needle hub when administering epidural or spinal anaesthesia
- Use aids to locate nerves: ultrasonography and/or electrical stimulation.

In the presence of seizures

- Administer a benzodiazepine (e.g. diazepam 0.2 mg/kg IV), repeat if necessary and/or consider a continuous rate infusion of midazolam. Increasing inhalant concentration or injecting propofol is best avoided, as it could precipitate cardiovascular collapse
- Ensure delivery of 100% oxygen, and ventilate
- Manage hyperthermia if present and consider increasing fluid rate to hasten renal clearance.

If respiratory arrest occurs

- Deliver 100% oxygen
- Intubate the patient's trachea if this has not already been done
- Start controlled ventilation
- Consider increasing fluid rate to hasten renal clearance
- Administer a 20% lipid emulsion bolus and infusion to bind the drug (Weinberg, 2012) (*doses below*).

When facing hypotension:

- Ensure adequate circulating blood volume by administering a crystalloid fluid bolus (e.g. 10–20 mL/kg for dogs and 5–10 mL/kg for cats)
- Treat with vasopressors, avoiding vasopressin, according to the recommendations of the American Society of Regional Anesthesia and Pain Medicine (ASRA) (2018) (*Table 2*)
- Consider a lipid emulsion infusion, especially if hypotension is refractory.

In case of cardiac arrhythmias:

- **Bradycardia:** administer atropine at 0.02–0.04 mg/kg IV. If prolonged anticholinergic activity is needed, consider glycopyrronium 0.01 mg/kg IV as an alternative.

Table 2. Drugs most commonly used as vasopressors

Drug	Administration (IV)	Dose
Ephedrine	Bolus and/or CRI (repeated boluses lead to tachyphylaxis)	**Bolus** 0.02–0.05 mg/kg **CRI** 0.06–0.3 mg/kg/h
Dopamine	CRI	**For vasoconstriction:** 0.2–0.6 mg/kg/h
Noradrenaline	CRI	0.006–0.06 mg/kg/h
Phenylephrine	Bolus and/or CRI	**Bolus** 0.002–0.02 mg/kg **CRI** 0.06–0.18 mg/kg/h

Modified from Duke-Novakovski (2016). CRI: continuous rate infusion; IV: intravenously.

- **Ventricular arrhythmias:** do **not** use lidocaine. According to human guidelines, calcium-channel blockers and β-blockers are best avoided. If available (as the drug is generally not readily available in the injectable form), amiodarone could be considered (1–5 mg/kg slow IV in dogs), but frequent anaphylactic episodes had been reported in dogs (Cober et al, 2009) and the authors do not advise its routine use. To the authors' knowledge, no drugs for managing ventricular arrhythmias in small animals seem to be indicated in this case. Administering one or more boluses of 20% lipid emulsion and considering an infusion seems most appropriate at this stage. (doses below).

If cardiac arrest occurs

- Start cardiopulmonary resuscitation with advanced life support
- If administering adrenaline and considering lipid emulsion use, give less than 0.001 mg/kg IV (recommended dose by ASRA (Rioja Garcia 2015)), because adrenaline impairs effective lipid resuscitation (Hiller et al, 2009).
- Administer lipid emulsion 20% as a slow IV bolus and repeat it or start a CRI if necessary (adequate circulation not restored). Doses in cats are 1–2 mL/kg bolus followed by a CRI at 2–5 mL/kg/h (use for several hours has been described) (O'Brien et al, 2010). Doses in dogs are 4 mL/kg bolus followed by 0.5 mL/kg/min for 10 minutes (Weinberg et al, 2003). According to human guidelines, it seems advisable to use lipid emulsion to treat any life-threatening local anaesthetic adverse effect, from neurological manifestations to cardiovascular instability. Starting doses as a bolus are the same whatever the symptoms. Consider repeating the bolus once or twice if there is persistent cardiovascular collapse; start a CRI at the aforementioned doses if the improvement in clinical signs is not satisfactory. The duration of the infusion will depend on the patient's clinical response; the CRI should be continued until signs of local anaesthetic toxicity have disappeared (Grayling, 2009; Stanford ACAG, 2016).

Malignant hyperthermia (MH)

In small animals, malignant hyperthermia is thought to be a rare genetic condition involving the skeletal muscle and related to an autosomal dominant mutation of the ryanodine receptor RYR1. It seems, nevertheless, that it should be considered more a syndrome than a specific disease (Brunson and Hogan, 2004): other muscle disorders such as canine stress syndrome (also called exercise-induced hyperthermia) and channelopathies share some of the same clinical manifestations (Adami et al, 2012; Thrift et al, 2017).

At the moment, genetic testing for research of RYR1 mutation in dogs is available but screening remains infrequent. Furthermore, muscle biopsies are needed to diagnose muscle disorders, so caution is advised when anaesthetising dogs suspected to be affected by these conditions (Adami et al, 2012). Risk factors that may trigger malignant hyperthermia in a predisposed patient are:

- Preoperative stress
- Inhalant anaesthesia
- Genetic muscle disorders (Adami et al, 2012).

Not all clinical signs may be present, and they may be variable. In humans, they can vary from mild to severe and include (Bellah et al, 1989; Brunson and Hogan, 2004; Thompson et al, 2014):

- Hyperthermia with rapid increase in body temperature over less than 1 hour during general anaesthesia (GA)
- Hypercapnia (rapid increase of end-tidal carbon dioxide ($EtCO_2$))
- Tachycardia (bradycardia is possible)
- Increased blood pressure (hypotension is possible)
- Tachypnoea
- Muscle rigidity (infrequent in dogs) (Adami et al, 2012)
- Cardiac arrhythmias
- Mixed metabolic and respiratory acidosis.

Differential diagnoses when facing these clinical signs under GA may be:

- Inadequate plane of anaesthesia or lack of analgesia
- Overheating
- Seizure-like activity
- Sepsis
- Anaphylactic episode
- Hypertension
- Thyroid storm
- Phaeochromocytoma.

Suspicion of malignant hyperthermia may be supported by observing hyperkalaemia, increased creatinine kinase activity and myoglobinuria. An arterial blood gas will show acidaemia and coagulation status may indicate disseminated intravascular coagulation (DIC).

Management

Malignant hyperthermia is a severe and quickly evolving life-threatening condition. Dantrolene IV is the mainstay treatment in human medicine, but it is very expensive and is not commonly available in veterinary medicine. A dose range of 2–5 mg/kg IV has been reported in the dog by Ramsey et al (2011).

If this drug in not available:

- Stop delivery of the inhalant anaesthetic agent. Changing the anaesthetic machine and/or breathing system is controversial in human medicine (time consuming), but a rebreathing system is definitely best avoided. Maintain anaesthesia with injectable drugs to conclude surgery
- Administer 100% oxygen and high fresh gas flow

- Stop any warming device
- Apply wrapped ice packs on major blood vessels (jugular veins and femoral arteries)
- Infuse cold IV fluids
- Treat hyperkalaemia if electrocardiogram (ECG) changes are observed. It is recommended to protect the myocardium with calcium gluconate 10% (50–100 mg/kg IV over 1–2 minutes) and to administer glucose plus insulin (0.55–1.1 U/kg IV of regular insulin and 1–2 g of glucose per unit insulin).
- Sodium bicarbonate administration could be considered for metabolic acidosis (1–2 mEq/kg IV over 15 minutes) (Saxon et al, 2012). If a blood gas analysis can be performed, administration of sodium bicarbonate should be preferably based on the value of base excess (also referred to as base deficit) and according to the formula: 0.3 x body weight (kg) x base deficit
- The ability to provide manual or mechanical ventilation is recommended.

Consider

- Colon and/or gastric lavage with cool water
- Bladder lavage with cool saline
- Central muscle relaxants (benzodiazepines) to try to counter act muscle rigidity, although there is no evidence of their efficacy.

If recovery from the event is successful, continuing treatment includes (Allman et al, 2009; Stanford ACAG, 2016):

- Continue fluids, to promote diuresis and avoid acute kidney injury because of myoglobinuria
- Based on recommendations in humans, administer dantrolene orally (0.5–2 mg/kg in cats, 1–5 mg/kg in dogs) every 6–8 hours for 24 hours (doses reported by Ramsey et al, 2011).

Seizures and seizure-like muscle contractions

Seizures or seizure-like muscle contractions are rarely seen in anaesthetised patients and are mostly encountered at induction or recovery. According to McMillan and Darcy (2016), seizures or convulsions account for 0.4% of anaesthetic adverse events in dogs and 1% in cats.

Clinical signs that may be observed are those such as muscle twitching or tremors, myoclonus or clonic muscle activity, paddling and dystonia (Lervik et al, 2010; Mitek et al, 2013).

These manifestations are most likely to be seen in cases of:

- Patients with a previous history of seizures
- Insufficient dose of alfaxalone at induction, associated or not associated with poor premedication (Deutsch et al, 2017)
- Repeated boluses or continuous rate administration of alfaxalone (Dehuisser et al, 2017)
- Propofol injection and infusion (Davies, 1991; Mitek et al, 2013; Cattai et al, 2015)
- Ketamine use (Kayama, 1982; Adami et al, 2013)
- Myelography, especially with high-volume contrast injection in the cisterna magna, and in large breeds (da Costa et al, 2011).

Differential diagnoses include:

- Inadequate plane of anaesthesia
- Nociception and inadequate analgesia during general anaesthesia
- Excitement stage/plane of anaesthesia, dysphoria (only before general anaesthesia or during emergence)
- Electrolyte imbalance (hypo/hypernatraemia, hypocalcaemia)
- Local anaesthetic systemic toxicity (fasciculation and true seizures)
- True seizure activity (normally obtunded under general anaesthesia, due to metabolic causes or intracranial disease).
- Air embolism (Mouser and Wilson, 2015).

Management

Prevention of the excitement stage/dysphoria or seizure-triggers is a key point and can be successfully achieved by:

- Not withdrawing anti-seizure drugs on the day of anaesthetic for patients on treatment
- Avoiding stress and minimising physical restraint in patients with suspected intracranial disease
- In animals with previous history of seizure-like activity, avoiding drugs that potentially decrease the seizure threshold, such as lidocaine, or can trigger an epileptic episode, such as ketamine. Note that acepromazine is no longer considered to be contraindicated (Tobias et al, 2006)
- Providing adequate premedication and/or sedation for recovery, especially in anxious or overactive animals (e.g. give before emergence of anaesthesia: medetomidine 0.001–0.002 mg/kg IV, acepromazine 0.005–0.02 mg/kg IV)
- Using a co-induction technique, with the other induction agent injected first, when including a benzodiazepine in the anaesthetic protocol; this decreases the likelihood of paradoxical effects of benzodiazepines (Sànchez et al, 2013)
- If excitement is prolonged and/or intense after benzodiazepine use, consider giving flumazenil IV (benzodiazepine antagonist) (see *Table 3*).

If the excitement stage/dysphoria happens during recovery, it seems advisable to:

- Inject an IV bolus of propofol (1 mg/kg) or alfaxalone (0.5 mg/kg) to gain rapid control of the animal, then administer a sedative such as midazolam or medetomidine, if this has not been done previously. If a sedative has already been injected, consider increasing the dose or adding a different drug, preferring a dose in the lower end of the range, as synergism with the other sedative is likely to occur
- Consider reversing opioids with IV naloxone (see *Table 3*). This is very rarely necessary and care should be taken to ensure adequate postoperative analgesia. A conservative approach consists of administering a small dose of naloxone (0.002–0.01 mg/kg IV), repeating as necessary after a few minutes, until a calm recovery is obtained and the patient is aware.

When facing seizure-like muscle contractions before induction of anaesthesia or during recovery:

- If an IV catheter is not in place, administer a benzodiazepine per rectum (e.g. diazepam 0.5–1.0 mg/kg), or midazolam 0.2 mg/kg intranasally (Charalambous et al, 2017). If an IV catheter is in place, administer a benzodiazepine IV (diazepam or midazolam 0.2–0.3 mg/kg) or directly induce anaesthesia
- Protect the airway/intubate and administer oxygen 100%, ensuring adequate ventilation.

Table 3. Drugs for which a reversal is available and reversal dosage

Drug	Management
Opioids	Naloxone (0.01–0.04 mg/kg IV) administer slowly to effect
α_2-agonists	Atipamezole (1:1 medetomidine volume, 5:1 dose in dogs and 2.5:1 dose in cats), given intramuscularly
Benzodiazepines	Flumazenil 0.05 mg/kg IV
Neuromuscolar blockers (atracurium, vecuronium)	Neostigmine (0.01–0.05 mg/kg) + atropine (0.04 mg/kg) Commercial mix of neostigmine 2.5 mg + glycopyrronium 500 µg/mL (same neostigmine dose as above)
Modified from Malouin Wright (2015)	

If abnormal movement occurs during anaesthesia:

- Ensure correct plane of anaesthesia by checking eye position, mandibular tone and monitoring
- Ensure sufficient analgesia; if in doubt, try giving rescue analgesia (e.g. fentanyl 0.001–0.002 mg/kg IV), repeat if necessary and adapt the analgesic regime (consider a CRI or to increase the rate of an ongoing CRI)
- Increase anaesthetic depth with inhalant, if needed
- Administer a muscle relaxant: a benzodiazepine (midazolam or diazepam, 0.2–0.3 mg/kg IV) or alternatively an α2-agonist (0.001–0.002 mg/kg medetomidine IV)
- Thoracic limb rigidity with or without opisthotonos following alfaxalone or propofol during induction or recovery may require medical intervention. The authors advise proceeding with induction of anaesthesia and/or consider improving muscle relaxation/sedation with an α_2-agonist or a benzodiazepine, depending on the period during anaesthesia (premedication or recovery).

If sustained dystonia occurs:

- Control body temperature to prevent hyperthermia
- Check blood glucose and lactate. Correct hypoglycaemia if present and ensure adequate fluid therapy
- Check for electrolyte imbalances (especially increased potassium, because of possible rhabdomyolysis). Correct these with adequate fluid therapy and in case of clinical hyperkalaemia, protect the myocardium with calcium gluconate 10% (50–100 mg/kg IV over 1–2 minutes) and administer glucose plus insulin (0.55–1.1 U/kg IV of regular insulin and 1–2 g of glucose per unit insulin) to decrease serum potassium
- Continue IV fluid administration to ensure renal perfusion and protection and to accelerate excretion of anaesthetic drugs.

Postanaesthetic oliguria/acute renal failure

Oliguria is defined as a urine production of less than 0.5 mL/kg/h, whereas anuria is the absence of urine production. Postanaesthetic acute renal failure causing oliguria or anuria may be because of:

- Pre-existing chronic kidney disease (consider fluid loading before surgery)
- Nephrotoxins (antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme (ACE) inhibitors), sepsis
- Surgery (compression of the vena cava)
- Computed tomographic contrast administration.

Management

- Monitor urinary output hourly (if urinary catheter in place, or place a urinary catheter)
- Maintain normovolaemia
- Consider crystalloid fluid bolus (5–10 mL/kg/h and assess if any improvement)
- Monitor INs and OUTs (match fluid going in with fluids going out)
- Maintain normal blood pressure
- Check and treat electrolyte disturbances
- Titrate fluid therapy according to losses and urinary output.

Check

- For the correct position of the catheter and the absence of catheter obstruction, as incorrect position or obstruction will not allow urine to flow and will lead to an incorrect conclusion of anuria/oliguria
- Bladder size (palpation/ultrasound guidance), as this will help to confirm if urine is being produced or not, especially in the case of an absent or ineffective urinary catheter
- Haematology and biochemistry, especially for anaemia (compatible with undiagnosed advanced chronic kidney disease) and increase in blood urea nitrogen (BUN) and creatinine values. These will confirm kidney failure and their monitoring will guide fluid therapy and clinical management.

Postoperative hyperthermia in cats

Postoperative hyperthermia in cats (rectal temperature >39.2 °C (Posner et al, 2007)) seems to occur fairly frequently. This condition is unresponsive to administration of NSAIDs (Niedfeldt and Robertson, 2006) and can easily be overlooked, as there are usually no overt clinical signs; therefore postoperative rectal temperature monitoring is required. This adverse event is more likely to present when opioid-based analgesia is used and in the first 5 hours postoperatively (Posner et al, 2010), especially if severe perioperative hypothermia was encountered (rectal temperature ≤34.4 °C) (Posner et al, 2007).

Potential differential diagnoses to consider with an increased postoperative rectal temperature in cats are:

- Iatrogenic overheating
- Seizure-related muscle contractions
- Infection
- Anaphylaxis
- Phaeochromocytoma.

Management

- Supportive treatment for hyperthermia: providing room temperature fluids and placing a wet towel and electric fan in front of the cage door
- As previously stated, NSAIDS are ineffective in this case
- Naloxone 0.01 mg/kg intramuscularly or subcutaneously (SC) is the specific treatment for opioid-induced postoperative

hyperthermia in cats. Return to normal rectal temperature is expected within 30 minutes (Posner et al, 2010). It is possible that this dose of naloxone will reverse the opioid-related analgesia, therefore ensure the cat has other analgesia provided, as required.

Postoperative hypotension

Systolic arterial pressure of less than 110 mmHg and/or mean arterial pressure less than 80 mmHg in a patient that has recovered from anaesthesia could be considered as postoperative hypotension.

Management

- Palpate peripheral and central pulses
- Confirm blood pressure measurement
- Assess heart rhythm by acquiring an ECG trace
- Check for signs of blood loss/haemorrhage (external or internal; abdominal focused assessment for trauma (A-FAST) and/or a thorax focused assessment for trauma (T-FAST) is useful)
- Consider second IV access to:
  - Rapidly correct fluid deficits with crystalloids and/or colloids
  - Infuse vasopressors (see *Table 2*)
- Follow cardio-pulmonary resuscitation guidelines if cardiac arrest confirmed.

Exclude

- Pneumothorax
- Cardiac tamponade
- Anaphylaxis.

(Riley, 2009)

Postoperative nausea/vomiting

Postoperative nausea and vomiting is the second more frequent complaint after pain in human anaesthetic practice (Shaikh et al, 2016). This adverse effect, mostly related to general anaesthesia and the use of some drugs such as opioids, ketamine or inhalant anaesthetics, is likely to be overlooked in veterinary practice, negatively affecting patient comfort and delaying recovery and home return.

Management

- Treat pain
- Assess fluid and electrolyte imbalances and correct them
- Drug therapy: maropitant 1 mg/kg SC or IV once daily, omeprazole 1 mg/kg once to twice daily, metoclopramide (if no gastrointestinal obstruction is suspected) 1–2 mg/kg/day CRI or 0.5 mg/kg bolus SC or IM
- Abdominal ultrasound to assess gastric and intestinal motility/obstruction/emptying
- Consider nasogastric tube to evacuate gastric contents (if gastric emptying is delayed)
- If persistent, administer ondansetron 0.1 mg/kg IV
- Review the patient's analgesia plan, start pain scoring and attempt multimodal analgesia in order to decrease the dose of, or delay the administration of, opioids.

(Riley, 2009)

Prolonged recovery/unarousable dog or cat

When facing a prolonged recovery, rule out:

- Hypothermia
- Metabolic/toxic encephalopathy: hypoglycaemia, sepsis, drug toxicity, liver failure
- Increased intracranial pressure
- Opioid overdose
- Recurarisation, if neuromuscular blockade was used intraoperatively
- If the patient is a dog from a breed predisposed to MDR1 (multidrug resistance) (otherwise called ABCB1) polymorphism (collies, Australian shepherds and others), and morphine, butorphanol and/or acepromazine have been used, consider the possibility that this genetic condition has been undiagnosed (Animal Genetics, N.D.).

Management

- Ensure airway, breathing and circulation are unaffected
- Protect the airway (intubate and ventilate obtunded patients) and support cardiovascular function with inotropes (e.g. dobutamine), vasopressors (e.g. ephedrine, dopamine) or anticholinergics (e.g. glycopyrrolate or atropine) if needed
- Ensure normal body temperature and blood glucose levels
- Continue IV fluid therapy to promote diuresis and hasten renal clearance of drugs
- Check anaesthetic drugs administered (correct dose, drug, route of administration)
- Administer antagonists/reversal agents of α_2-agonists, benzodiazepines and neuromuscular blockers. Consider antagonising opioids if the animal remains unarousable but beware of reversal of analgesia. It is advisable to start with a low dose and to repeat it if necessary (*Table 3*).

Consider

- Continuous ECG and blood pressure monitoring
- Placement of urinary catheter if recumbency is prolonged (more than 8–12 hours)
- Nursing care of the recumbent patient, including changing recumbency side every 4 hours. Lubricate the eyes regularly if the patient remains sedated and is warmed with an air warming blanket.

Respiratory failure and hypoxaemia

Respiratory failure can be considered in case of respiratory distress, inadequate respiratory rate and/or inadequate respiratory depth in a patient that has awakened from anaesthesia. Hypoxaemia is defined as a partial pressure of oxygen (PaO_2) less than 80 mmHg: the pulse oxymeter will indicate a value of haemoglobin saturation with oxygen (SpO_2) less than 90%.

Management

- Measure SpO_2
- Auscultate the lungs
- Inspect the airways for evidence of any obstruction
- Provide 100% oxygen
- Prepare to induce anaesthesia and intubate the trachea

- Obtain chest radiographs
- If there is a history of brain disease, check for signs of increased intracranial pressure with possible compression of the respiratory centre, in the brainstem (assess neurological function, heart rate and blood pressure: obtunded patients with bradycardia and hypertension are showing Cushing's reflex)
- Obtain arterial blood gas to assess the grade of hypoxaemia.

Rule out

- Obstruction of the airways
- Pneumothorax, pleural effusion
- Pulmonary oedema, aspiration pneumonia
- Drugs and doses administered (possible overdose)
- Central nervous system disorder/nerve damage
- Residual neuromuscular blockade.

(Riley, 2009)

Consider

- Pulmonary embolism, especially in animals at increased risk like those with hypercoagulable states or blood stasis (see 'Pulmonary embolism' in Part 1 (Marotto et al, 2018)).

Rough recovery/agitation

Rough recoveries are a fairly common adverse event and risk factors include:

- Anaesthetic drugs: ketamine, benzodiazepines (paradoxical effect), inhalant anaesthetic agents (emergence from anaesthesia too rapidly)
- Opioid-related dysphoria: two nucleotide polymorphism mutations within the opioid receptor have been identified in some dogs and may be associated with dysphoria (Hawley and Wetmore, 2010)
- Discomfort and postoperative pain
- Abrupt termination of drug (e.g. remifentanil, fentanyl) administration
- Geriatric dogs seem clinically more prone (old age is a risk factor in people (O'Keeffe and Chonchubhair, 1994))
- Excitable and anxious dogs, especially of certain breeds
- No research articles appear to be available on the subject but, in the authors' experience, Labrador Retrievers, Staffordshire Terriers, Siberian Huskies and Yorkshire Terriers appear to be clinically predisposed.

Management

- Prevent the patient from injuring themselves or others
- Consider sedation:
  - Dex/medetomidine 0.001–0.002 mg/kg IV
  - If this is not available immediately, or in case of a cardiovascularly unstable patient: alfaxalone 0.3–0.5 mg/kg IV or propofol 0.5–1 mg/kg IV
  - Acepromazine at 0.005–0.02 mg/kg IV may be considered in dogs expected to experience agitation on recovery. It is best administered before emergence from anaesthesia, as its onset is delayed compared to the previous drugs suggested. Alternatively, acepromazine can be administered following an alpha2-agonist to continue the sedative effect.
- Check for bladder distension and express as needed
- Reconsider analgesic plan (too much or too little)
- Consider antagonism of drugs (benzodiazepines, opioids).

Rule out

- **Serotonin syndrome:** restlessness, myoclonus, tremor, shivering, sweating fever and other autonomic system symptoms following use of serotonin enhancing drugs (tramadol, pethidine, trazodone, selective serotonin reuptake inhibitors, monoamino-oxidase inhibitors)
- **Anticholinergic syndrome:** associated with the administration of atropine
  - In this case, agitation is accompanied with mydriasis, tachycardia, peripheral vasodilation. Management is with physostigmine (0.5–2 mg in people, 0.04 mg/kg in horses). Wiese et al (2014) showed improved recovery quality when horses were given physostigmine instead of neostigmine.

(Riley, 2009)

Severe postoperative pain

Risk factors

- Poor intraoperative analgesia
- Abrupt interruption of analgesics such as remifentanil and fentanyl.

Management

- Check the animal does not have a full bladder and is actually uncomfortable because of this; express the bladder if needed
- Check that the animal has not been positioned in a painful body position, particularly when osteoarthritis is present, and ensure it is lying on a comfortable blanket and/or mattress
- Pain score the patient using a validated pain assessment tool. The results will also help to choose the most appropriate analgesic regime. Validated pain scoring systems for postoperative pain include, for example, the Glasgow composite measure pain scale for dogs (the short form version usually gives satisfactory information) and the Glasgow feline acute pain scale or the UNESP-Botucatu Multidimensional Composite Pain Scale for cats
- Increase full mu agonist opioid or start opioid analgesia
- Administer a non-steroidal anti-inflammatory drug if not contraindicated and not previously done
- Give a ketamine bolus (0.3–0.5 mg/kg IV) and start a CRI (e.g. 0.15–0.3 mg/kg/h IV), especially if severe pain is more likely to be because of somatic or neuropathic origin
- Give a lidocaine bolus (2 mg/kg) and start a CRI (0.72–1.8 mg/kg/h), especially if the pain is of visceral origin
- Lidocaine for analgesic purposes is not currently recommended in cats, as this species is very sensitive to its toxic effects
- In dogs, consider paracetamol 10 mg/kg BID to TID if there is no liver or kidney functional impairment. Consider gabapentin (10 mg/kg BID to TID) or pregabalin (2–4 mg/kg TID) oral administration if there is a neuropathic component.
- Consider dex/medetomidine CRI (0.5–2 µg/kg/h).

(Dudgale, 2010; Vettorato and Corletto, 2011)

KEY POINTS

- Knowledge of perioperative complications related to anaesthesia allows for their faster recognition and management.
- Knowledge of risk factors in intra- and postoperative care might prevent the events from occurring.
- Accurate and recorded monitoring, both intra- and postoperatively, will allow for a much prompter recognition of adverse events.

Conclusions

This review, along with part 1 (Marotto et al, 2018), highlights the variety of challenging situations related to anaesthesia that may occur in the perioperative period. Prompt recognition of the complications that may occur in the perioperative period is likely to improve their management and result in successful outcome. This review is designed to provide the general practitioner with a valuable tool to aid safe, everyday anaesthetic practice.

References

Adami C, Axiak S, Raith K, Spadavecchia C. Unusual perianesthetic malignant hyperthermia in a dog. J Am Vet Med Assoc. 2012;240(4):450–453. https://doi.org/10.2460/javma.240.4.450

Adami C, Spadavecchia C, Casoni D. Seizure activity occuring in two dogs after S-ketamine- induction. Schweizer Archiv für Tierheilkunde 155(10):569-572. https://doi.org/10.1024/0036-7281/a000513

Allman KG, McIndoe AK, Wilson IH et al. Emergencies in anaesthesia, 2nd edn. Oxford: Oxford University Press; 2009

American Society of Regional Anesthesia and Pain Medicine (ASRA). Checklist for treatment of local anesthetic systemic toxicity. 2018. https://www.asra.com/content/documents/asra_last_checklist_2018.pdf (accessed 16 January 2020)

Animal Genetics. Multi-drug resistance gene. http://www.animalgenetics.us/Canine/Genetic_Disease/MDR1.asp (accessed 16 January 2020)

Bellah JR, Robertson SA, Buergelt C, McGavin AD. Suspected malignant hyperthermia after halothane anesthesia in a cat. Vet Surg. 1989;18(6):483–488. https://doi.org/10.1111/j.1532-950X.1990.tb01132.x

Brodbelt D. Perioperative mortality in small animal anaesthesia. Vet J. 2009;182(2):152–161. https://doi.org/10.1016/j.tvjl.2008.06.011

Brunson DB, Hogan JH. Malignant hyperthermia: a syndrome not a disease. Vet Clin North Am Small Anim Pract. 2004;34(6):1419–1433. https://doi.org/10.1016/j.cvsm.2004.05.010

Cattai A, Rabozzi R, Natale V, Franci P. The incidence of spontaneous movements (myoclonus) in dogs undergoing total intravenous anaesthesia with propofol. Vet Anaesth Analg. 2015;42(1):93–98. https://doi.org/10.1111/vaa.12160

Charalambous M, Bhatti SFM, Van Ham L et al. Intranasal midazolam versus rectal diazepam for the management of canine status epilepticus: a multicenter randomized parallel-group clinical trial. J Vet Intern Med. 2017;31(4):1149–1158. https://doi.org/10.1111/jvim.14734

Cober RE, Schober KE, Hildebrandt N, Sikorska E, Riesen SC. Adverse effects of intravenous amiodarone in 5 dogs. J Vet Intern Med. 2009;23(3):657–661. https://doi.org/10.1111/j.1939-1676.2009.0314.x

Da Costa RC, Parent JM, Dobson H. Incidence of and risk factors for seizures after myelography performed with iohexol in dogs: 503 cases (2002-2004). J Am Vet Assoc. 2011;238(10):1296–1300. https://doi.org/10.2460/javma.238.10.1296

Davies C. Excitatory phenomena following the use of propofol in dogs. J Vet Anaesth. 1991;18(1):48–51. https://doi.org/10.1111/j.1467-2995.1991.tb00014.x

Dehuisser V, Bosmans T, Kitshoff A et al. Cardiovascular effects, induction and recovery characteristics and alfaxalone dose assessment in alfaxalone *versus* alfaxalone-fentanyl total intravenous anaesthesia in dogs. Vet Anaesth Analg. 2017;44(6):1276–1286. https://doi.org/10.1016/j.vaa.2017.04.006

Deutsch J, Jolliffe C, Archer E, Leece EA. Intramuscular injection of alfaxalone in combination with butorphanol for sedation in cats. Vet Anaesth Analg. 2017;44(4):794–802. https://doi.org/10.1016/j.vaa.2016.05.014

Dudgale A. Veterinary anaesthesia: principles to practice. Oxford: Wiley-Blackwell; 2010:55–56

Duke-Novakovski T. Haemodynamic support during the anaesthetic period. In: Duke-Novakovski T, de Vries M, Seymour C (eds). Manual of canine and feline anaesthesia and analgesia. 3rd edn. Gloucester: British Small Animal Veterinary Association (BSAVA); 2016:231

Fink EL, Kochanek PM, Clark RSB, Bell MJ. Fever control and application of hypothermia using intravenous cold saline. Pediatr Crit Care Med. 2012;13(1):80–84. https://doi.org/10.1097/PCC.0b013e3181fe27c7

Grayling M. Regional anaesthesia. In: Allman KG, McIndoe AK, Wilson IH (eds). Emergencies in anaesthesia. 2nd edn. Oxford: Oxford University Press; 2009:235–239

Hawley AT, Wetmore LA. Identification of single nucleotide polymorphisms within exon 1 of the canine mu-opioid receptor gene. Vet Anaesth Analg. 2010;37(1):79–82. https://doi.org/10.1111/j.1467-2995.2009.00506.x

Hiller DB, Gregorio GD, Ripper R et al. Epinephrine impairs lipid resuscitation from bupivacaine overdose: a threshold effect. Anesthesiology. 2009;11:498–505. https://doi.org/10.1097/ALN.0b013e3181afde0a

Kayama Y. Ketamine and E.E.G. seizure waves: interaction with anti-epileptic drugs. Br. J. Anaesth. 1982; 54(8):879-883. https://doi.org/10.1093/bja/54.8.879

Lervik A, Haga HA, Becker M. Abnormal muscle activity during anaesthesia in a dog: case report. Acta Vet Scand. 2010;52(1):64. https://doi.org/10.1186/1751-0147-52-64

Malouin Wright A. Sedative, muscle relaxant and narcotic overdose. In: Silverstein D, Hopper K (eds). Small animal critical care medicine. 2nd edn. St. Louis (MO): Saunders; 2015:400–407

Marotto S, Bradbrook C, Zoff A. Anaesthetic complications and preparedness part 1: pre- and intraoperative complications. Companion Anim. 2018;23(10):578–584. https://doi.org/10.12968/coan.2018.23.10.578

McMillan M, Darcy H. Adverse event surveillance in small animal anaesthesia: an intervention-based, voluntary reporting audit. Vet Anaesth Analg. 2016;43(2):128–135. https://doi.org/10.1111/vaa.12309

Mitek AE, Clark-Price SC, Boesch JM. Severe propofol-associated dystonia in a dog. Can Vet J. 2013;54(5):471–474

Mouser PJ, Wilson JD. Fatal venous air embolism during anesthesia in an apparently healthy adult Chihuahua. J Am Anim Hosp Assoc. 2015;51(3):176–179. https://doi.org/10.5326/JAAHA-MS-6118

Niedfeldt RL, Robertson SA. Postanesthetic hyperthermia in cats: a retrospective comparison between hydromorphone and buprenorphine. Vet Anaesth Analg. 2006;33(6):381–389. https://doi.org/10.1111/j.1467-2995.2005.00275.x

O'Brien TQ, Clark-Price SC, Evans EE, Di Fazio R, McMichael MA. Infusion of a lipid emulsion to treat lidocaine intoxication in a cat. J Am Vet Med Assoc. 2010;237(12):1455–1458. https://doi.org/10.2460/javma.237.12.1455

O'Keeffe ST, Chonchubhair AN. Postoperative delirium in the elderly. Br J Anaesth. 1994;73(5):673–687

Otero PE, Campoy L. Epidural and spinal anesthesia. In: Campoy L, Read MR (eds). Small animal regional anesthesia and analgesia. Oxford: Wiley-Blackwell; 2013:252

Posner LP, Gleed RD, Erb HN, Ludders JW. Post-anesthetic hyperthermia in cats. Vet Anaesth Analg. 2007;34(1):40–47. https://doi.org/10.1111/j.1467-2995.2006.00287.x

Posner LP, Pavuk AA, Rokshar JL, Carter JE, Levine JF. Effects of opioids and anesthetic drugs on body temperature in cats. Vet Anaesth Analg. 2010;37(1):35–43. https://doi.org/10.1111/j.1467-2995.2009.00508.x

Ramsey I et al. Small Animal Formulary. 7th edn. Gloucester: British Small Animal Veterinary Association (BSAVA); 2011:442

Riley R. Recovery problems. In: Allman KG, McIndoe AK, Wilson IH (eds). Emergencies in anaesthesia. 2nd edn. Oxford: Oxford University Press; 2009:313–352

Rioja Garcia E. Local anesthetics. In: Grimm KA, Lamont LA, Tranquilli WJ, Greene SA, Robertson SA (eds). Veterinary anesthesia and analgesia, the fifth edition of Lumb and Jones. Oxford: Wiley-Blackwell; 2015:344

Sànchez A, Belda E, Escobar M et al. Effects of altering the sequence of midazolam and propofol during co-induction of anaesthesia. Vet Anaesth Analg. 2013;40(4):359–366. https://doi.org/10.1111/vaa.12038

Saxon WD, Drobatz KJ, Macintire DK. Endocrine and metabolic emergencies. In: Macintire DK, Drobatz KJ, Haskins SC, Saxon WD (eds). Manual of small animal emergency and critical care medicine. 2nd edn. Oxford: Wiley-Blackwell; 2012:382

Shaikh SI, Nagarekha D, Hegade G, Marutheesh M. Postoperative nausea and vomiting: A simple yet complex problem. Anesth Essays Res. 2016; 10(3):388–396. https://doi.org/10.4103/0259-1162.1793.10

Stanford Anesthesia Cognitive Aid Group. Emergency Manual, Cognitive aids for perioperative critical events, V3.1. University of Stanford, Stanford Medicine; 2016. http://emergencymanual.stanford.edu (accessed 16 January 2020)

Thompson SM, Burton CA, Armitage-Chan EA. Intra-operative hyperthermia in a cat with a fatal outcome. Vet Anaesth Analg. 2014;41(3):290–296. https://doi.org/10.1111/vaa.12097

Thrift E, Wimpole JA, Child G et al. Exercise-induced hyperthermia syndrome (canine stress syndrome) in four related English springer spaniel. Vet Med. 2017;8:59–68. https://doi.org/10.2147/VMRR.S123836

Tobias KM, Marioni-Henry K, Wagner R. A retrospective study on the use of acepromazine maleate in dogs with seizures. J Am Anim Hosp Assoc. 2006;42(4):283–289. https://doi.org/10.5326/0420283

Vettorato E, Corletto F. Gabapentin as part of multi-modal analgesia in two cats suffering multiple injuries. Vet Anaesth Analg. 2011;38(5):518–520. https://doi.org/10.1111/j.1467-2995.2011.00638.x

Weinberg GL, Ripper R, Feinstein DL, Hoffman W. Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. Reg Anesth Pain Med. 2003;28(3):198–202. https://doi.org/10.1097/00115550-200305000-00005

Weinberg GL. Lipid emulsion infusion: Resuscitation from local anesthetic and other drug overdose. Anesthesiology. 2012; 117(1):180-187. https://doi.org/10.1097/ALN.0b013e31825ad8de.

Wiese AJ, Brosnan RJ, Barter LS. Effects of acetylcholinesterase inhibition on quality of recovery from isoflurane-induced anesthesia in horses. Am J Vet Res. 2014;75(3):223–230. https://doi.org/10.2460/ajvr.75.3.223