Emergency anaesthesia

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Patient's that require 'emergency anaesthesia' usually have a soft tissue rather than an orthopaedic complaint and are mostly abdominal emergencies. The most common types of life-saving surgery and thus, emergency anaesthesia, are for a gastric dilation +/- volvulus or a haemoabdomen.

Gastric dilation + volvulus (GDV)

The early warning signs of GDV are increased anxiety and restlessness in the patient, but these clinical signs are subtle enough that they may be missed or misinterpreted by owners. These signs quickly progress to hypersalivation, vomiting and/or retching, and the presence of a distended abdomen. At this time, venous return to the heart decreases, and the caudal vena cava and portal veins become obstructed due to reduced blood flow to the stomach and surrounding organs. As the GDV progresses; depression, weakness and lethargy take hold, tidal volume and pulmonary function decrease due to gastric distention into the thoracic space, myocardial and tissue hypoxia increase leading to tissue ischaemia, lactate production increases creating a metabolic acidosis and ultimately severe obstructive shock ensues due to inadequate tissue perfusion.

GDV is always considered an emergency and mortality rates are extremely high. Between 10-27% of patients die due to extensive damage to the stomach and surrounding organs, resulting in euthanasia in the perioperative period. The high mortality rates are also associated with cardiac arrhythmias pre, peri or postoperatively, reperfusion injury, hypo or hyperthermia, hypotension and the development of postoperative renal failure, and the length of time from onset of clinical signs to presentation and surgery.

Preparing the GDV patient

On immediate presentation, two large bore IV catheters should be placed in the cephalic veins to start IV fluid therapy. Shock doses of 80-90ml/kg/hr may be used, however volume resuscitation "endpoints" must be kept in mind, so this is best achieved by giving a bolus of 10-20ml/kg, then reassessing perfusion parameters, and repeating the bolus if required. Delivering goal directed boluses rather than the "shock rates" will help to reduce hypervolaemia and tissue oedema.

Gastric decompression by passing a stomach tube should be initiated as soon as possible. If the tube will not pass it should not be forced further down past the obstruction, as this may rupture the stomach. An alternative is performing centesis on the enlarged stomach the same way we do thoracocentesis. The abdomen is prepped, an IV catheter is aseptically placed into the stomach, and a three way tap, extension set and 60ml syringe is attached to the catheter to remove gas/fluid. The catheter can be left in situ during the surgical prep period to relieve gases as they build up.

Blood should be collected to perform a minimum of a packed cell volume (PCV), total plasma protein (TPP), creatinine, electrolytes and acid base levels. Measuring lactate levels is helpful as high lactate levels on presentation that fail to decrease with fluid resuscitation is a predictor for poor outcomes.

A pre anaesthetic assessment and physical examination is required to accurately determine an overall health status at the time of the GDV and should be performed during the initial stabilisation period. It may be difficult to perform a complete physical examination in some of these patients, but the cardiopulmonary systems should be focused on. Oxygen support can be provided via a mask and wherever possible an ECG should be placed to monitor for cardiac arrhythmias. If arrhythmias are auscultated (as they frequently are with GDV patients) these should be identified and treated prior to induction of anaesthesia. Their origin is usually ventricular and a lignocaine bolus of 1-2mg/kg can be given slowly IV, followed by a constant rate infusion (CRI) at 25-100µg/kg/min. Even if no cardiac arrhythmias are detected lignocaine should be considered. Its use during anaesthesia decreases anaesthetic requirements by up to 43%, it has anti-inflammatory effects and may reduce the occurrence of reperfusion injury.

Anaesthetising the GDV patient

Pre oxygenation immediately prior to induction should be provided via a mask. An ECG, blood pressure monitor, and pulse oximeter may be placed well before the induction of anaesthesia. We consider these patients unfasted as they have a “full stomach”, therefore care must be taken to prevent regurgitation and aspiration during the induction and maintenance periods by using a rapid induction technique, maintaining a “head up” position during induction, intubating the trachea with a cuffed endotracheal tube and inflating the cuff while the patient is still in the “head up” position.

Induction of anaesthesia is via either an opioid-benzodiazepine combination in the depressed and moribund patient or may include propofol or alfaxalone in low doses if they are clinically more stable. Maintenance of anaesthesia is achieved with isoflurane, or the combination of isoflurane as well as fentanyl or remifentanil and lignocaine delivered as CRI’s. Once anaesthetised with a protected airway, a stomach tube can remain in situ within the oesophagus to help protect from gastric reflux being aspirated. Most GDV patients require positive pressure ventilation (PPV) either manually or via a mechanical ventilator, as the enlarged stomach greatly reduces both the tidal volume and functional residual capacity of the lungs.

An arterial catheter should be placed wherever possible for direct blood pressure monitoring and arterial blood gas analysis. If direct monitoring is unavailable, indirect methods such as Oscillometric or the ultrasonic Doppler must be utilised and recorded frequently as hypotension is expected due to marked decreases in venous return. Hypotension should be treated by simple methods (decreasing the anaesthetic), as well as including either a vasopressor (noradrenaline), or inotropy agents (dopamine or dobutamine) in the form of a CRI.

As the surgery progresses, an ischaemic reperfusion injury is likely at the time of de-rotation or decompression of the stomach (or may even occur after restoration of fluid volume). The likelihood of a reperfusion injury is directed correlated to the length of time the gut has been ischaemic for, and sometimes a reperfusion injury does not present if the ischaemic event has not been long enough. A reperfusion injury may initially present as cardiac arrhythmias but may progress to signs of disseminated intravascular coagulation (DIC), systemic inflammatory response syndrome (SIRS) or multiple organ dysfunction syndrome (MODS). Both lignocaine and ketamine can reduce ischemic reperfusion injury markers and may be either added to or continued in recovery. Ketamine reduces the adhesion of neutrophils and the production of inflammatory cytokines, while lignocaine prevents the formation of free radicals, stops neutrophil formation, and act as a sodium channel blocker.

It is necessary to continue monitoring of the GDV patient into the recovery period, and they warrant the continued use of monitoring equipment, in particular an ECG, blood pressure monitoring and pulse oximetry. They are expected to recover slower, will most likely be

hypothermic to some degree, and are often painful. Supplemental oxygen can be provided via a mask or nasal prongs in the initial recovery period. Shivering and rewarming is “metabolically expensive”, with oxygen demand increasing by around 400% to perform this task, highlighting the necessity for thermal support during the anaesthesia maintenance phase as well as into the recovery. Cardiac arrhythmias may continue for over 24 hours post operatively. Analgesia should be provided in the form of μ agonist opioids, with a fentanyl lignocaine ketamine (FLK) or methadone lignocaine ketamine (MLK) CRI a good selection. Monitoring should be performed continuously until vital parameters are close to normal limits, however it may take many hours for this to occur. The GDV patient is likely to require several days of intensive nursing support post-surgery, with the first 24 hours being the most critical.

Haemoabdomen

A ‘haemoabdomen’ is free blood in the peritoneal cavity and may be ‘non-traumatic’ or ‘traumatic’ in origin. Over 80% of non-traumatic haemoabdomen cases in dogs are caused by malignant masses of the spleen, with three-quarters of these masses being haemangiosarcoma. In cats a non-traumatic haemoabdomen is less likely, however has been documented. Almost half of feline cases are neoplastic masses within the abdomen, with 60% of these being haemangiosarcoma, most commonly from the spleen. Neoplastic masses may also come from the liver, mesentery, and adrenal glands. Additional causes of a non-traumatic haemoabdomen include coagulation defects caused by an anticoagulant (rodenticide toxicity), splenic and liver lobe torsion and “other” abdominal organ torsion (for example, a GDV). Non-traumatic haemoabdomen patients are generally older, with a median age of 10 years in dogs. Labrador Retrievers, German Shepherds and Golden Retrievers have a higher incidence than other breeds.

The most likely causes of a traumatic haemoabdomen in both dogs and cats is blunt force trauma - caused by being ‘hit by a car’, or from a penetrating injury from sticks, gun shots or bite wounds. These patients often have multiple injuries to many body sites including pneumothorax, haemothorax, pulmonary contusions, fractures, and other soft tissue injuries. A traumatic haemoabdomen is also a possibility after a biopsy, or when a surgery has been performed and prior haemostasis techniques have failed.

Internal haemorrhaging may be a gradual process or be quite sudden and has the potential to become life threatening very quickly. Patients usually present to the veterinary hospital in a depressed state, with signs of ‘early’ decompensated shock, tachycardia, pale pink mucous membranes, slow capillary refill time, hypotension, weak pulses, and mild hypothermia. Owners may report a history of acute collapse, with additional clinical signs of blood loss including weakness, lethargy, anorexia, and abdominal distention with pain on palpation. If the signs of early decompensated shock are not noticed or perhaps not acted on swiftly enough, patients may present in a state of ‘late’ decompensated shock. They show signs of pronounced hypovolaemic shock including marked depression or a moribund demeanour, hypotension, pale mucous membranes with no capillary refill time, bradycardia, and hypothermia.

Preparing the haemoabdomen patient

On presentation, oxygen support should be provided, and an ECG should be placed to monitor for cardiac arrhythmias. A PCV and TPP can be collected, with an understanding that these values may not have decreased significantly yet in an acute bleed. Splenic contraction may elevate PCV 10-20% but will not influence TPP levels. Clotting ability should also be assessed by performing a prothrombin time (PT) and partial thromboplastin time (PTT).

Fluid resuscitation must commence, and this will require two large bore IV catheters to be placed. Goal directed volume resuscitation is sensible, especially as large volume resuscitation

is associated with dilution of clotting factors, elevations in blood pressure and disruption of clot formation, potentially worsening haemorrhage. Types of fluid used differ depending on the level of estimated blood loss. Balanced isotonic crystalloids, colloids or hypertonic saline may be appropriate. If the PCV is lower than 20% the addition of blood products may be necessary, although many institutions prefer the source of bleeding to be under control before transfusing. Volume resuscitation endpoints include a decrease in heart rate, and an increase in blood pressure to low normal values.

A pre anaesthetic assessment and physical examination should be performed on the haemoabdomen patient during the initial stabilisation period.

Anaesthetising the haemoabdomen patient

Pre oxygenation should be performed immediately prior to induction, and an ECG, blood pressure monitor, and pulse oximeter should all be preplaced. We do consider these patients to be unfasted, therefore a rapid induction technique, with a 'head up' position during induction is sensible. Induction of anaesthesia is via either an opioid-benzodiazepine combination or may include propofol or alfaxalone in low doses if they are clinically more stable. Maintenance of anaesthesia is achieved with low concentrations of isoflurane, or the combination of isoflurane as well as fentanyl (or remifentanil) for analgesia and lignocaine for cardiac arrhythmias. An arterial catheter should be placed wherever possible for direct blood pressure monitoring and arterial blood gas analysis. If direct monitoring is unavailable, indirect methods such as Oscillometric or the ultrasonic Doppler must be utilised. Hypotension should be treated; however, care must be taken to not elevate blood pressure too high. 'Permissive mild hypotension' is sensible, with the goal of not increasing systolic blood pressure over ~90mmHg or a mean blood pressure over ~60mmHg until haemorrhage has ceased or is under control.

An autotransfusion of the patient's own blood may be started. This involves collecting blood out of the abdomen aseptically and delivering it back to the patient via a blood transfusion set or a blood filter. Anticoagulant is often not required, as free blood that has pooled in a serosa lined cavity usually does not clot, and it is defibrinated after around an hour. It is a massive benefit to use the patient's own blood, as it is immediately available, prewarmed, and does not carry the risk of a transfusion reaction. This has been shown in humans as well as in dogs, with no reports of increased rectal temperature, urticaria, erythema, vomiting, laboured breathing, or any other reactions associated with administering autotransfusions in any of the patients presented in the literature.

Historically, it was considered contraindicated to perform an autotransfusion in patients with suspected metastatic cancer, however, it is unknown whether delivering an autotransfusion of the haemorrhaged blood from a ruptured cancerous mass will in any way contribute meaningfully to the actual spread of cancer in dogs. Human research has failed to demonstrate a worse outcome or increased metastatic rate associated with delivering an autotransfusion to oncology patients, and more importantly, there was no association between the presence of circulating cancer cells and a worse (poorer) outcome.

Monitoring the continued fluid resuscitation and additional blood loss in theatre is extremely important. This may be difficult when irrigation fluids are used concurrently. Methods of estimating blood loss include weighing bloody swabs, lap sponges and towels, as well as monitoring the volume of lavage fluids used to estimate blood versus fluids inside suction canisters. Additional PCV/TPP may be collected throughout surgery, although haemodilution and the use of colloids may significantly alter these values.

Once haemorrhage has stopped (and if an autotransfusion has not been started), a blood transfusion may be started if facilities allow. If it is the dog's first transfusion it will not matter which blood type is used as they will not have sufficient antibodies against it, however if the patient has had a transfusion before, or is feline, a major crossmatch should be performed to determine the patient's blood type *before* transfusing can commence. Whole blood is preferred, and should be started at a rate no greater than 1ml/kg/hr. If no signs of transfusion reaction occur this may be doubled after 10-15 minutes to 2ml/kg/hr, and continually increased every 10 minutes to 4, 6, 8 and up to a maximum of 10ml/kg/hr. Signs of transfusion reaction in the anaesthetised patient include an increasing heart rate not in response to surgical stimulation, decreasing blood pressure and signs of oedema particularly around the face and eyes of the patient.

Recovery considerations are like the GDV patient, with the addition of transfusion management. Overall prognosis for the haemoabdomen patient is poor if it was caused by neoplasia, however many patients that are stabilised successfully survive to hospital discharge.

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