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Focus on Emergency Surgery Mini Series

Session Three: A to Z of Emergency Abdominal Surgery

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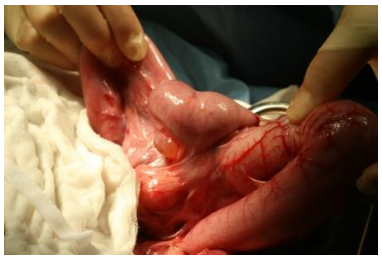


Webinar notes: Emergency surgery of the abdomen

Introduction:

So far in our webinar mini-series we have looked at management of trauma patients and management of respiratory emergencies. In this final webinar, we will look at commonly encountered abdominal emergencies.

B is for biliary surgery:

Within the liver, canaliculi drain bile into interlobular ducts. These converge further into lobar ducts that become known as *hepatic ducts* as they exit the liver parenchyma. Hepatic ducts converge to form the common bile duct, which enters the duodenum at the major duodenal papilla. The point at which the first hepatic duct joins the cystic duct is the point at which the common bile duct commences.

There are several important differences in hepatobiliary and pancreatic ductal anatomy of dogs and cats. In dogs the common bile duct enters the duodenum at the major duodenal papilla adjacent to, but not conjoined with, the pancreatic duct. The accessory pancreatic duct is actually the larger pancreatic excretory duct and enters at the minor duodenal papilla; this smaller, less obvious papilla is located approximately 2 cm aboral to the major duodenal papilla and, in dogs, is the principal conduit for pancreatic secretions.

In cats the common bile duct and pancreatic duct conjoin just before their entry into the duodenum at the major duodenal papilla. In addition, only approximately 20% of cats have a smaller, accessory pancreatic duct that exits at a minor duodenal papilla.

Gall bladder mucoceles

Gall bladder mucoceles are the most common reason for surgical intervention of the biliary tract in dogs. Hypersecretion of mucus (possibly secondary to cholecystitis) leads to an accumulation of thick gelatinous bile within the lumen. This may lead to extrahepatic biliary obstruction or bile peritonitis secondary to gall bladder rupture. The cause of this condition remains largely unknown. Certain breed predispositions are reported; Shetland sheepdogs and border terriers. Hyperadrenocorticism and hypothyroidism have also been reported to be associated with the development of gall bladder mucoceles.

Clinical signs are often non-specific but can include abdominal pain, intermittent pyrexia, vomiting, lethargy and icterus (if concurrent CBD obstruction). Diagnosis is predominantly made by ultrasound. Gall bladder mucoceles have a characteristic “kiwi fruit” appearance on ultrasound. Medical management with resolution is reported in sporadic case reports but this is generally considered to be a surgical disease. Cholecystectomy is the surgical procedure of choice but ONLY if there is patency of the hepatic ducts and common bile duct. This is sometimes a decision made on the basis of the ultrasound findings and the blood work (if the common bile duct is of a normal size and there is no evidence of icterus) although some surgeons will always flush the CBD to confirm patency prior to resection. If flow cannot be established by lavage alone, a stent may need to be placed prior to gall bladder removal.

Biliary surgery can be very difficult. Vitamin K is essential for coagulation but requires bile salts to be absorbed from the gastrointestinal tract. Coagulation times (PT and APTT) should be assessed before surgery but do not guarantee normal coagulation and therefore vitamin K supplementation is recommended before surgery regardless of the numbers. Platelet numbers should also be assessed.

The gall bladder is dissected free from its fossa; a stay suture placed at the apex can be useful in helping to manipulate the gall bladder. If the gall bladder is adhered to the diaphragm, neuromuscular blockade is useful to aid in dissection of any adhesions as can resection of the triangular ligaments. The major issue with this initial dissection is haemorrhage and it is important to stay as close the gall bladder capsule as possible. Using a fine suction tip, sterile cotton buds or a cautery device such as the Endseal or a harmonic scalpel can be very useful. Once dissected free from the liver to the level of the entry of the hepatic ducts from the liver (at which point the cystic duct becomes the common bile duct), the cystic duct should be either ligated (with an encircling ligature) and oversewn with suture or ligated with a VA30 stapler. My clinical experience is that aggressive flushing of the CBD to establish patency (particularly in a retrograde direction from the major duodenal papilla) can lead to significant pancreatitis and cholangiohepatitis. I am therefore more cautious with keeping the volume of flush to the minimum required. If the patency of the CBD has not been established prior to surgery, a cholecystotomy and catheterisation of the cystic duct and CBD can be performed prior to cholecystectomy. This procedure carries a relatively high post-operative morbidity and mortality (up to 15% of patients die prior to discharge from the hospital depending on the paper you read) although with correct post-operative management, this can be reduced. Thus the rate of post-operative complications should be discussed with the owner prior to surgery. An oesophagostomy tube should be placed in these patients at the time of surgery as ileus is common post-surgery.

F is for feeding

Provision of assisted feeding should be considered in all cases where an extended period of inappetence has been experienced and is expected to continue. Protein energy malnutrition can lead to impaired immunity, increased risk of infection, delayed wound healing, weakness and in very extreme cases, organ failure and death. In pigs, villous atrophy has been demonstrated in as a little as 1.5 days when fasted. Starvation, especially when complicated by stress, adversely affects the intestinal epithelial barrier.

Resting energy requirements in the non-traumatized surgical patient can be estimated using the following equation:

$$RER\ (Kcal/day) = 30\ BW\ (kg)+70$$

In severely traumatised patients, requirements may be double this figure. A discussion of the individual components of diet for post-surgical patients is outwith the scope of this lecture but is an important factor to consider.

Enteral nutrition is easier, cheaper and more physiologic than parenteral feeding. As a general rule, food should enter the gastrointestinal tract as close to the oral cavity as possible to allow as many of the normal physiological processes involved in digestion to occur. Various steps can be taken to encourage voluntary feeding such as warming food, owner visits and the use of appetite stimulants.

Options for assisted feeding include naso-oesophageal tubes, oesphagostomy tubes, gastrotomy tubes and jejunostomy tubes. The decision should be made based on the underlying pathology, length of expected tube use, the consistency of the food which you wish to use and co-morbidities which may affect suitability for anaesthesia should all be considered. For many trauma patients, an oesophagostomy tube may be the most sensible compromise.

- **Naso-oesophageal tube**
    - **Pros:** can be placed in the conscious patient
    - **Cons:** small tube size limits diets which can be used. Not always well tolerated by patients. Easily dislodged.

- **Oesophagostomy tube**
    - **Pros:** easy to place, larger tube size allows use of a wider range of diets. Can be removed when no longer required. If stomal inflammation occurs, removal usually resolves this issue without the need for further intervention. Positioning away from the face usually means these tubes are well tolerated.
    - **Cons:** stomal inflammation is relatively common. Should not be used in patients with oesophageal disease.
- **Gastrostomy tube**
    - **Pros:** can be placed whilst already in the abdomen without need for post-operative radiography or need for repositioning. Larger tube size allows for use of normal canned diets (when blended).
    - **Cons:** risk of septic peritonitis if leakage around the stoma. Needs to be maintained ideally for 10 days prior to removal.
- **Jejunostomy tube**
    - **Pros:** allows stomach to be bypassed so can be useful in case where there is severe pancreatitis. Can be placed through a gastrostomy tube in some cases.
    - **Cons:** Challenging to place and maintain. Must be placed as an open surgical procedure. Risk of septic peritonitis if dislodged.

What to feed:

Larger tubes allow the use of specific (which can be blended) whereas smaller tubes such as a NO tube rely on the use of liquid diets such as RCW Convalescence. All feeding tubes should be flushed with water prior to feeding to check positioning and also following feeding to prevent blockage of the food.

Medications can be given via the tube if crushed or if available in a liquid formulation. However, medications which are presented in enteric coated granules such as omeprazole should not be given via the tube as they can cause blockage.

Care of feeding tubes:

The exit point of the feeding tube through the skin should be dressed with a sterile dressing (e.g. Melonin or Primipore) which should be changed twice daily after cleaning of the stoma site. It is not unusual to get peri-stomal inflammation which usually resolves once the tube is removed. Iodine (e.g. Betadine) can be used as a topical cream at the stoma site.

Feeding tubes should be flushed with water twice daily even when not in use. If oesophagostomy or gastrostomy tubes become blocked, Coca-Cola (full fat not diet) can be used to unblock the tube.

G is for Gastric Dilatation and Volvulus (GDV):

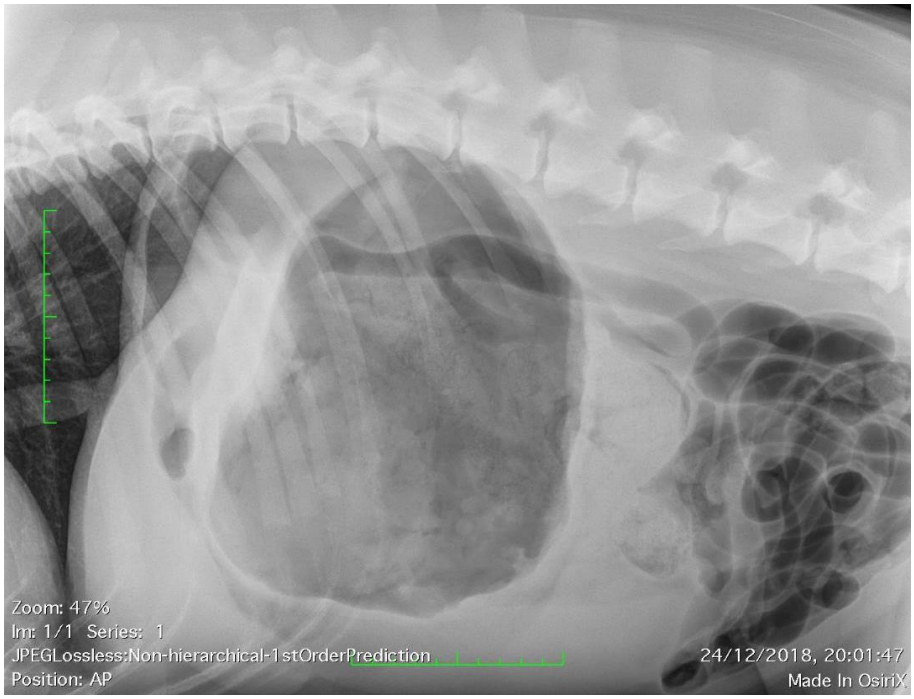
GDV is a life-threatening syndrome where gastric dilation and torsion lead to portal hypertension, systemic hypotension and cardiogenic shock. It is reported to be responsible for 2.5% of deaths in pedigree dogs (Evans and Adams 2010). Reported risk factors include breed and size of dog (Great Danes, Gordon Setters, Irish Setters, Weimaraner, Standard Poodles and Bassett Hounds are over-represented), dogs with a close relative that also had a GDV, a large thoracic depth to width ratio (narrow deep chest), being underweight for the breed standard, increasing age, feeding habits (eating from a raised bowl/once a day/eating rapidly), stress. The effect of exercising immediately after eating is still controversial (Pipan et al 2012).

The exact pathogenesis remains unknown – whether volvulus precedes dilatation or vice versa continues to be debated. As the stomach distends, the pylorus is displaced right to left ventrally across the floor of the abdomen and then cranially to end up positioned on the left, dorsal to the intra-abdominal portion of the oesophagus. Significant gastric distension has effects on multiple other body systems and volvulus prevents release of this gas. Effects on the respiratory system include increased pressure on the diaphragm which reduces the excursion of the diaphragm and reduced functional lung capacity which lead to alveolar hypoventilation, VQ mismatch and hypoxaemia. Increased PCO2 leads to respiratory acidosis. There are also significant effects on the cardiovascular system. Compression of portal vein and vena cava leads to venous stasis, loss of the intestinal mucosal barrier and subsequent bacterial translocation. Reduced venous return leads to decreased cardiac output and hypotension, portal hypertension and therefore decreased clearance of bacteria and endotoxins. Myocardial ischemia occurs due to inadequate coronary blood flow and production of myocardial depressant factor. All of the above leads to metabolic acidosis, hypoglycaemia and endotoxaemia.

Presenting signs are well documented and include hypersalivation, unproductive vomiting or retching, abdominal distension, compensated vs decompensated shock and dyspnoea. In early compensated septic shock, patients are haemodynamically stable. Dogs will often present with a hyperdynamic and hypermetabolic response which manifests as tachycardia, tachypnoea, hyperaemia, reduced CRT and pyrexia. Compensatory mechanisms begin to fail leading to hypoperfusion and hypotension, increased blood lactate and decreased central venous oxygen saturation, maldistribution of blood flow, endothelial dysfunction, activation of leucocytes and the coagulation cascade. Increased capillary permeability leads to interstitial oedema. As septic shock progresses; refractory hypotension develops leading to multi-organ failure and death. These patients are hypothermic, comatose and have poor/absent peripheral and central pulses. Chronic weight loss and intermittent bloating are more commonly seen in chronic partial volvulus.

Diagnosis: a high suspicion of GDV can be identified from the clinical presentation.

- Minimum blood work database at presentation should include PCV/TP, electrolytes, glucose, urea and creatinine +/- lactate +/- acid base measurements if available. Trends in lactate concentrations have been suggested to be predictive of survival but results from various retrospective studies vary. There is considerable overlap between the values of survivors and non-survivors and therefore this **cannot** be used as a rigid “cut off” to predict survival. Ultimately, lactate should be only be used to monitor trends and only if the values have the power to change your treatment plan. Do not repeatedly check lactate and then ignore the results.
- A right lateral abdominal radiograph should be used to assess the position of the pylorus. Classically, patients with GDV demonstrate the reverse “C” sign. The gas dilated pylorus is identified sat dorsal to the gastric lumen; separated by a “shelf” of soft tissue. Pneumoperitoneum is indicative of gastric wall rupture secondary to necrosis.



Stabilisation:

The priority is stabilisation by restoration of circulating volume. Place two large bore peripheral intravenous catheters or a central line (although this usually requires general anaesthesia). Do not use the saphenous vein as fluid will simply pool in the venous circulation caudal to the obstruction caused by the gastric dilation. A continuous recording ECG should be attached to the patient if available and oxygen supplementation provided.

Analgesia is usually indicated and opioids represent the best choice. If an opioid alone is inadequate, other options for analgesia include either a ketamine constant rate infusion (CRI) or a lidocaine CRI. Intravenous paracetamol can also be considered (10mg/kg as a slow intravenous infusion over 15-30 minutes). Non-steroidal anti-inflammatory drugs e.g. meloxicam should be avoided in hypovolaemic, hypotensive patients as there is an increased risk of renal toxicity and gastric ulceration.

ECG abnormalities are seen in 40-70% of GDV cases which are predominantly ventricular in origin. Correction of fluid deficits and electrolyte disturbances and provision of analgesia are necessary prior to surgery. Anti-arrhythmic treatment is indicated when the arrhythmia is seen to affect cardiac output. These include:

- runs of VPCs >20
- ventricular tachycardia >160bpm
- poor peripheral pulses
- multiform VPCs
- R on T

Gastric Decompression:

There are two main options for gastric decompression, **orogastic decompression** using a stomach tube or via **percutaneous decompression** of the gastric lumen through the body wall using a large bore needle. Goodrich et al reviewed 116 cases of GDV undergoing gastric decompression. Decompression was performed via orogastric tubing in 31 dogs, gastric trocarization in 39 dogs and a combination of both in 46 dogs. Tube decompression was successful in 59 (75·5%) dogs and unsuccessful in 18 (23·4%) dogs. Trocarization was successful in 73 (86%) dogs and unsuccessful in 12 (14%) dogs. No evidence of gastric perforation was noted at surgery in dogs undergoing either technique. One dog that underwent trocarization had a splenic laceration identified at surgery that did not require treatment. Oesophageal rupture or aspiration pneumonia was not identified in any dog during hospitalization. No statistical difference was found between the method of gastric decompression and gastric compromise requiring surgical intervention or survival to discharge.

Surgical management:

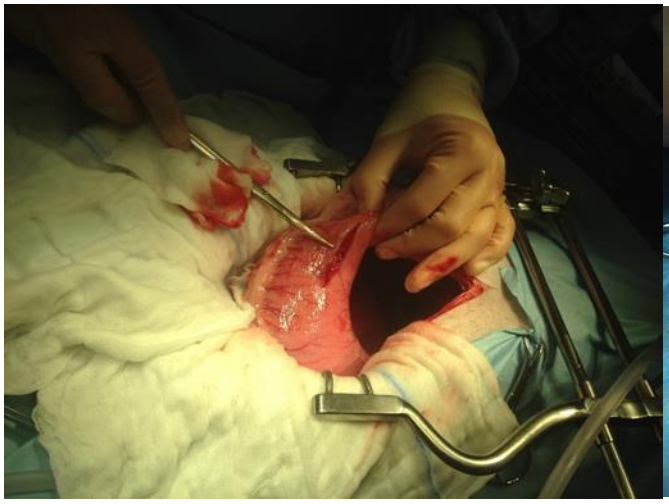
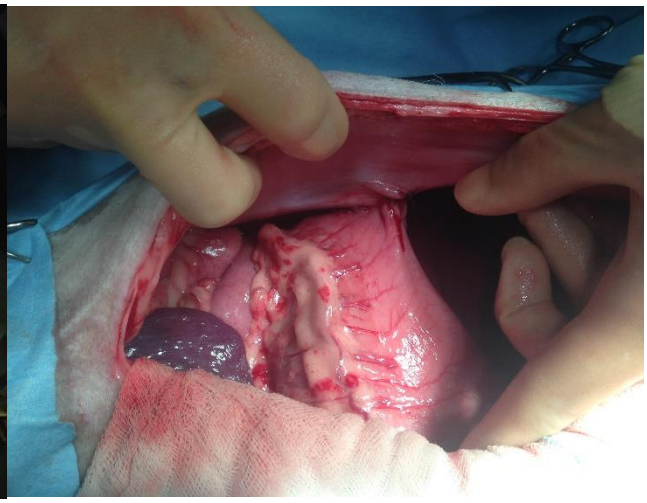
Anaesthesia can be challenging for these cases so be organised! Perform as much of the pre-surgical clip as possible prior to induction of anaesthesia to reduce duration of anaesthesia. The head should be elevated above the thorax until an inflated endotracheal tube is in place as there is a risk of aspiration pneumonia and ensure you have suction ready in case of debris in the pharynx.

Goals of GDV surgery

- **to decompress and reposition the stomach**
- **assess the viability of the stomach wall**
- **create a permanent adhesion between the gastric antrum and the body wall**

Initially, the stomach wall can appear very discoloured due to venous stasis so it important to reposition the stomach and wait for 5-10 minutes to allow blood flow to be restored. Determining tissue viability can be challenging – assess the thickness of the tissue and look for signs of arterial pulses within the gastric vessels. Fluorescein dye has been used experimentally but is not often used in a clinical setting. **Have a good look round!** Ensure you check the gross appearance of the dorsal surface of the stomach as well as the ventral surface.

Any obviously dead tissue (black, dark purple or green) should be either invaginated in to the gastric lumen or removed via partial gastrectomy (the author prefers the latter). The final step in surgical management is the creation of a permanent adhesion between the gastric antrum and the right side of the body wall caudal to the last rib. Numerous methods are described for gastropexy with varying reports of the strength of the adhesion formed by each technique. Ultimately the author believes the simplest effective technique should be pursued and therefore performs incisional gastropexy as it is rapid and effective (Przywara et al 2014). All techniques are well described in all major textbooks. It is, however, worth taking a moment to consider that the position of the pexy can cause significant issues if not correctly chosen. Ensure you do not cause an outflow obstruction in the positioning of your gastropexy. Laparoscopic assisted gastropexy is reported for prophylactic purpose in at risk breeds. Loy Son et al reported this technique in 49 dogs. None of these dogs experienced GDV in the median follow-up time of 698 days.

Post-operative management of GDV patients:

If the patient is stable, an oesophagostomy tube should be placed prior to recovery from anaesthesia to ensure the provision of early enteral feeding. Other feeding tubes (e.g. gastrostomy tube) can be considered although an oesophagostomy tube is the author's site of choice unless there is significant oesophageal disease. This ultimately is personal preference.

Analgesia is essential and as previously discussed; methadone represents a sensible choice and can be used in conjunction with a lidocaine CRI if methadone alone is not effective. Lidocaine may also have a beneficial effect on survival in these patients in terms of reducing post-operative cardiac arrhythmias (Bruchim et al 2012). Epidural anaesthesia can be considered at the end of surgery.

Maintenance of an adequate circulating volume and improving plasma protein levels are both important. The best way to achieve this is through early enteral nutrition. Enteral nutrition provides an energy source for the enterocytes lining the intestinal mucosa and helps to reduce the risk of post-operative ileus. Intravenous fluid therapy is also important but the volume given should be tailored to the individual dog. Consider use of an indwelling urinary catheter to monitor urine output and urine concentration (although acute kidney injury due to endotoxaemia etc may affect USG) and also an "ins and outs" sheet. In the simplest terms, this is a chart which adds up the volume of fluid being given (intravenously, orally etc) and those being lost (urine production, vomiting, diarrhoea, insensible losses etc) and allows the clinician in charge to ensure the patient's needs are being met whilst avoiding over-hydration.

Post-operative medications: The author routinely uses intravenous omeprazole, maropitant, a metoclopramide CRI +/- intravenous antibiotic therapy (depending on the appearance of the gastric wall at surgery and the suspected degree of mucosal compromise).

Electrolytes and plasma protein levels should be monitored regularly until within the normal laboratory range.

Complications of GDV include septic peritonitis secondary to progressive gastric wall necrosis, ileus, pancreatitis, disseminated intravascular coagulation, cardiac arrhythmias and oesophagitis/gastritis.

H is for haemoabdomen:

Patient history: Owners may report previous episodes of acute onset lethargy, inappetance or “off days” which may equate to previous episodes of haemorrhage which were self-limiting. However signs can also be reported to be acute or peracute in onset with collapse being the most commonly reported clinical sign during active haemorrhage.

Presenting signs: Patients may be ambulatory or collapsed/obtunded. Heart rate varies with patient breed and size and therefore it is not possible to give one universal figure which should raise concern. You should also assess non-invasive blood pressure, pulse quality, mucus membrane colour and capillary refill time e.g. a heart rate of 130bpm may be inappropriate in a Great Dane but not in an anxious Jack Russell Terrier. When palpating peripheral pulses start in the most distal location and work your way to a more central location if you cannot palpate a pulse more peripherally. Note not only the presence of the pulse but also the quality of it. Abdominal distension can be difficult to assess depending on the size and body condition score of your patient but abdominal palpation should be performed to look for both the presence of an abdominal mass and also a fluid thrill.

What baseline data do we need?

- Rectal temperature, heart rate and respiratory rate
- Blood pressure (arterial line placement is preferable but can be challenging in the conscious patient. Oscillometric and Doppler blood pressure measurement represent credible alternatives)
- PCV/TP/urea/creatinine and electrolytes as a minimum. Haematology and biochemistry profiles are useful if available rapidly.
- In house blood smear to assess platelet count (>10 platelets per oil immersion field is equivalent to around 150x109/L). Be aware that in house haematology analysers can be inaccurate when it comes to assessing platelet numbers so a manual count is preferable if you are in doubt.

The priority is stabilisation by restoration of circulating volume. Place two large bore peripheral intravenous catheters or a central line (although this usually requires general anaesthesia). Our aim is to restore perfusion and oxygen delivery to organs, restore circulating volume, improve cardiac output and increase oxygen content of blood. A continuous recording ECG should be attached to the patient and oxygen supplementation provided. Analgesia is usually indicated and opioids represent the best choice. If an opioid alone is inadequate, other options for analgesia include either a ketamine constant rate infusion (CRI) or a lidocaine CRI. Intravenous paracetamol can also be considered (10mg/kg as a slow intravenous infusion over 15-30 minutes). Non-steroidal anti-inflammatory drugs e.g. meloxicam should be avoided in hypovolaemic, hypotensive patients as there is an increased risk of renal toxicity and gastric ulceration.

Crystalloids represent the mainstay of initial fluid resuscitation although it should be remembered around 75% of the volume given will be lost to the interstitium within one hour of administration. A balanced electrolyte solution is preferable to 0.9% saline as the latter can lead to metabolic acidosis secondary to elevated chloride levels. Rather than considering “shock-rate” fluids, it is preferable to give intravenous fluids in incremental boluses (10-20ml/kg given over 15 minutes) and monitor heart rate (which should reduce) and blood pressure (hypotension should improve) to assess response. Please note these doses may not be appropriate in patients with concurrent structural cardiac disease. Hypertonic saline can also be used (although the author has limited experience of it) and causes expansion of circulating volume by dehydration of the interstitium. It must therefore be followed by an isotonic solution.

In patients which are unresponsive to crystalloid therapy or where it is anticipated that large volumes of fluid supplementation will be required, colloids should be considered. Options for colloid therapy include hydroxyethyl starches and gelatins. Hydroxyethyl starches come in a variety of molecule sizes and therefore have variable duration of effect. There has been significant research efforts within human medicine over the past few years looking at the morbidity and mortality associated with the use of hydroxyethyl starches in critically ill patients. Recent randomised trials in human medicine have suggested that the use of hydroxyethyl starches in critically ill patients can lead to significant acute kidney injury and mortality; particularly in sepsis. This has led to a move away from the use of such colloids in veterinary medicine also although the same research has not currently been duplicated. At the present time, the use of hydroxyethyl starches in human medicine is limited to management of haemorrhage. Coagulopathy is reported as a side effect of hydroxyethyl starches due to a reduction in the concentration of factor VIII and von-Willebrand factor but this is not frequently recognised clinically. Gelatins are generally smaller sized molecules than those contained in hydroxyethyl starch solutions but are present in larger numbers. As the molecules are smaller, they do not remain in the circulation as long as the hydroxyethyl starch molecules. Increased anaphylaxis is reported in humans treated with gelatins than hydroxyethyl starches but this is not very common in dogs. Colloids are usually administered in 5ml/kg boluses up to a maximum of 50ml/kg/24 hours.

With particular respect to the haemoabdomen patient, the use of blood products is often indicated and replacement of erythrocytes will restore oxygen carrying capacity within the circulation. There is no set cut off for when a transfusion must be carried out. However it should be considered in patients which have lost >20% of circulating blood volume. The decision to transfuse should be based on the availability of blood products, the underlying cause of haemorrhage (and the likelihood of being able to control it), the volume of blood lost and the clinical status of the patient.

Patients should be blood typed and any patient which has previously received a blood transfusion (unless within the last 72 hours) should be also be cross matched. However, in an emergency situation, patients which have never received a transfusion can receive either DEA 1.1 positive or negative without typing if typing is not available. Two main options exist for replacement of erythrocytes: packed red cells and whole blood transfusions. Pros and cons exist for both of these options. Packed cells are convenient and there is now increased availability through the Pet Blood Bank. Packed cells also contain higher number of erythrocytes/ml so will increase PCV more than a whole blood transfusion. However a packed cell transfusion does not replace clotting factors or platelets; both of which are actively depleted in a patient with a haemoabdomen and there is also a cost implication to the use of such products. Where whole blood has been lost and a packed cell transfusion is planned, it is wise to consider combining this with fresh frozen plasma (FFP) to replace clotting factors but this can be expensive in larger patients (ideally a ratio of one bag of FFP is administered per bag of packed cells is recommended). Furthermore this approach will not address thrombocytopaenia. In contrast whole blood provides erythrocytes, platelets and clotting factors although platelet survival in whole blood transfusions is reduced. Furthermore, although whole blood is a “free” resource, collection of whole blood requires access to suitable equipment and also a donor.

Another option for provision of blood products is the use of cell salvage. Such systems are in common use in human medicine for autotransfusion of blood within a body cavity. Cell salvage machines pump the collected blood into a centrifugation bowl where dense erythrocytes are separated from plasma proteins and lighter cellular elements. The erythrocytes are then washed and re-suspended in 0.9% saline. This blood may then be administered to the patient immediately or stored for administration within 6hr of collection. Cell salvage systems are preferable to direct auto-transfusion of blood from the surgical field as the system removes other contaminants from the collected fluid and allows concentration of packed red cells which minimises transfusion volume. The use of such systems in neoplasia is controversial as there is the concern that autotransfusion of neoplastic cells from the ruptured spleen may lead to metastasis.

Research is currently underway looking at the efficacy of leucocyte reduction filters to remove any cells which are not erythrocytes from the collected blood. In vitro studies suggest that leucocyte reduction filters may be effective in removing neoplastic cells from abdominal fluid.

Oxygen supplementation can be provided via a number of routes. Face masks provide a maximal fractional inspired oxygen concentration (FiO2) of 50-60% with oxygen flows of 8-12L/min. More commonly flows of 2-5L/min are used. There is risk of rebreathing and also a risk of hyperthermia. Nasal prongs/catheters achieve a FiO2 of ~50% can be achieved using flows of 2l per 10kg per minute. However this is often not well tolerated in clinical patients and 2-5 l/min is more commonly used. It is important to humidify gas, if possible, to prevent irritation of the nasal mucosa. Oxygen cages and incubators can achieve FiO2 of up to 60% (more commonly 40-50%) but there is again a risk of hyperthermia and patient temperature should be closely monitored.

Differential diagnoses of a haemoabdomen include:

- Coagulopathy
    - Thrombocytopaenia
    - Thrombocytopathia
    - Vitamin K deficiency
    - Hepatopathy
    - Disseminated intravascular coagulation
    - Pharmacologic anticoagulants
    - Haemodilution (dilutional coagulopathy)
- Trauma
    - Acute trauma coagulopathy
    - Active haemorrhage from an intra-abdominal structure
- Iatrogenic e.g. post ovariohysterectomy
- Neoplasia
    - Hepatic (haemangiosarcoma (HSA), hepatic adenoma/carcinoma, biliary adenoma/carcinoma, metastatic disease)
    - Splenic (HSA, leiomyosarcoma, undifferentiated sarcoma, fibrosarcoma, osteosarcoma, liposarcoma, myxosarcoma, mast cell tumour, chondrosarcoma, rhabdomyosarcoma, malignant fibrous histiocytoma, lymphoma, metastatic adenocarcinoma, and myeloproliferative disease)
    - Adrenal (phaeochromocytoma, adrenocortical tumours and metastatic disease)

Various options exist for imaging of the abdomen. Abdominal radiographs are easily achieved but the presence of significant ascites will lead to a generalised increase in soft tissue opacity. Liver and splenic masses may also be identified on abdominal radiography depending on their size. However it is difficult to identify adrenal pathology unless it is significant in size or mineralised. Abdominal ultrasound represents the opportunity to determine the source of the pathology although the presence of free abdominal fluid can make interpretation of the scan challenging. Confirmation of a haemoabdomen can be made by identification of free abdominal fluid and abdominocentesis. This is most accurate and carries the least risk when performed under ultrasound guidance but can also be performed as a “blind” tap if a large volume of fluid is present. Once fluid is obtained, a PCV should be checked on the abdominal fluid and compared to the PCV of the patient. If the PCV of the abdominal fluid is over 10-15%, there is usually a reasonable suspicion of a haemoabdomen. If the systemic PCV and that of the abdominal fluid are comparable, this should be considered pathognomic for a haemoabdomen. CT, where available, is useful as it also provides the opportunity for staging of any suspected neoplasia.

The most commonly recognised causes of haemoabdomen requiring surgical intervention are either iatrogenic haemorrhage secondary to planned surgical intervention or neoplasia. Splenic neoplasia is common in dogs and up to two thirds of splenic lesions in dogs are neoplastic and up to 73% of splenic lesions in cats depending on which source within the literature is cited. Haemangiosarcoma is the most common neoplastic lesion of the spleen in dogs and this is characterised by rapid growth and widespread metastasis due to its vascular endothelial origin (which makes access for haematogenous spread relatively easy). Potential sites for metastasis of this tumour include the liver, lungs and right atrium as well as the lymphatic system. It is unclear if concurrent atrial lesions represent synchronous primary tumours or metastatic disease. Many other tumour types are reported to occur in the spleen, both primary and metastatic, including fibrosarcoma, mast cell tumours, osteosarcoma and malignant fibrous histiocytoma. The most common splenic neoplasms in cats are lymphoma and mast cell tumours.

As the incidence of malignancy is high in patients with a splenic mass, staging should be performed prior to surgery to provide information to the owner regarding prognosis. Imaging of the thorax (via three inflated views of the thorax or CT), abdomen (ultrasound or CT) and echocardiography of the heart to assess the right atrium and aurical appendage is recommended. In the case of adrenal tumours, it is important to check for the presence of vascular invasion in to the phrenicoabdominal vein, renal vein(s) or caudal vena cava. This is best achieved by an experienced ultrasonographer or contrast abdominal CT.

Between 0.6% and 2.6% of all canine and feline neoplasms involve the liver. Tumours of the biliary system are generally less common. Hepatobiliary tumours are of four general types: hepatocellular, cholangiocellular, neuroendocrine, or mesenchymal. Metastatic tumours are more common than primary ones, with the most common types being hematopoietic and lymphoid tumours, followed by epithelial and mesenchymal tumours. Mast cell tumours are also rarely found as primary or secondary tumours in the liver. Hepatocellular carcinomas are the most common primary liver tumours in dogs, representing approximately 50% to 70% of all non-hematopoietic neoplasms. They generally present as one of three forms: massive (61%), nodular (29%), or diffuse (10%).

Once your patient is adequately stabilised (aims here include normalisation of heart rate, blood pressure and other clinical parameters as previously discussed) and a frank discussion has been undertaken with the owner, surgery is usually indicated for patients with a haemoabdomen except in cases attributable to coagulopathy or trauma (haemorrhage in the majority of trauma cases is usually self-limiting although this is NOT a hard and fast rule and patients must be assessed on a case by case basis). It is worth noting an acute coagulopathy is present on admission in 25% to 34% of human trauma patients and is associated with a four-fold increase in mortality. Though a finite definition is not yet established for this syndrome, referred to as acute traumatic coagulopathy (ATC), most human studies use a 50% prolongation in either PT or APTT as diagnostic criteria.

If there is potentially a surgical solution for the haemorrhage issue in question and you feel confident to deal with the potential findings at surgery, surgery should be performed as soon as is feasible. Surgical success can be greatly enhanced by ensuring you have adequate lightening, access and equipment. Access to cautery, suction and a Poole suction tip and an assistant will all make life much easier.

Adrenalectomy can be very challenging for several reasons; firstly and perhaps most importantly because of the anaesthesia. When emergency adrenalectomy is performed because of underlying neoplasia this is complicated by the fact that the tumour is usually not known. Adrenal masses are not uncommon; incidental adrenal gland masses are identified in around 4% of routine abdominal ultrasound scans. In this paper, Cook et al reported that malignancy was more likely in lesions > 20mm in size. Adrenal gland lesions may represent neoplasia, hypertrophy or cystic lesions.

The most common types of adrenal tumour include adenomas, adrenocortical tumours (functional versus non-functional) and tumours arising from the chromaffin cells of the adrenal medulla which are known as pheochromocytomas. Functional adrenocortical tumours can produce cortisol, aldosterone or sex hormones. Cortisol secreting tumours usually lead to signs of hyperadrenocorticism. A combination of an ACTH stimulation test and low dose dexamethasone suppression test is used to confirm this diagnosis. There are no consistent abnormalities seen on routine blood and urine tests in dogs with pheochromocytomas. Measurements of urinary catecholamine concentrations or their metabolites are used to strengthen the diagnosis in patients with clinical signs consistent with such a tumour (systemic hypertension, tachycardia, weakness and excessive panting). Adrenalectomy is made challenging both by the potential effects on wound healing and coagulation as seen in cortisol producing tumours and the significant cardiac arrhythmias seen in pheochromocytomas. Pre-treatment with trilostane and phenoxybenzamine respectively has been shown to reduce intra-operatively mortality. However in cases of haemoabdomen secondary to a ruptured tumour, this is usually not practical. Access to drugs which could be used to treat intra-operative hypertension; phentolamine (alpha-antagonist) and nitroprusside (direct vasodilator), is therefore to be highly recommended for suspected pheochromocytoma patients.

Good access and appropriate retraction are essential for adrenalectomy, therefore an assistant is recommended. Prior to surgery it is important to know the extent of any vascular invasion and have planned appropriately to deal with this. The most common approach is a ventral midline one which allows exploration of the whole abdomen. The left adrenal gland is more easily identified at the craniomedial pole of the kidney. The right adrenal gland is often partially obscured by the vena cava and transection of the hepatorenal ligament can help with visualisation. A flank approach is also reported but is reserved for uncomplicated unilateral masses with no caudal vena caval involvement. Finally laparoscopic adrenalectomy has also been reported; again not appropriate for large masses. Dissection can be performed using a combination of blunt and scissor dissection or using bipolar sealant devices or a harmonic scalpel. The phrenicoabdominal vein should be ligated at the lateral aspect of the gland. There are usually multiple penetrating vessels on the dorsal surface of the gland. After the adrenal gland is freed from its lateral and dorsal attachments, a plane is developed between the renal vessels and the caudal aspect of the gland. As a final step, the phrenicoabdominal vein is ligated at its final entry in to the caudal vena cava. This can be very difficult to identify depending on the size of the tumour.

The reported incidence of caudal vena caval thrombi is variable depending on which paper you are reading. Overall the incidence is between 10-20% and is reported to be more common in pheochromocytomas in some papers (Massari et al, Lang et al, Barrera et al). Caval thrombi vary in size and can extent as far as the right atrium. Thrombi can be removed with a venotomy. Rummel tourniquets (loops of polypropylene passed through a section of soft cut chest drain) are passed around the caudal vena cava cranial and caudal to the tumour and also possibly separately around the renal vessels depending on the size of the tumour. The Rummel tourniquets are gently tightened to occlude blood flow through the caudal vena cava and a longitudinal incision is made adjacent to the entry point of thrombus in to the cava. The thrombus is removed and a Statinsky vascular clamp placed across the incision (but not fully occluding the caval lumen) and blood flow allowed to flow through the caudal vena cava once more. The venotomy incision is sutured using 5/0 polypropylene in a simple continuous pattern. Two separate suture lines may be required and thus use of a suture with a double needle (i.e. one on each end) can be useful. The vascular clamp should be gradually released. There is usually some haemorrhage through the holes where the suture needle has passed through the vessel wall which stops when pressure is applied with a swab over the area. If it does not, more sutures may need to be placed.

Prognosis for adrenal tumours is reasonable even with malignancy in patients who survive the initial post-operative period. Lang et al compared 52 dogs undergoing elective adrenalectomy and 8 dogs undergoing emergency adrenalectomy for acute adrenal haemorrhage.

Perioperative morality rates were 5.7% for dogs that underwent elective adrenalectomy and 50% for dogs that underwent emergency adrenalectomy for acute adrenal haemorrhage. However bear in mind these numbers are small. Median survival time was 492 days for the 53 dogs that survived the perioperative period. As there is a lack of prospective trials, there is some debate over prognostic factors but most papers agree that tumour size/volume, the presence of metastasis and the presence of an extensive caval thrombus all affect longer term prognosis.

Splenectomy is much more commonly performed in primary care practices than adrenalectomy and therefore an extensive discussion of technique is not provided. Broadly speaking there are two main techniques for ligation of the splenic vasculature. The hilar technique involves ligation of the individual splenic vessels close to their entry in to the splenic vasculature. This is a time consuming technique but does mean that there is no risk of disturbing the vascular supply to the left limb of the pancreas which is supplied by the splenic artery on its way to the spleen. In cases where the splenic vasculature can be clearly identified, a more rapid technique is to ligate the splenic artery and vein following bifurcation of the pancreatic vessels and also ligation of the short gastric arteries. Triple ligation is recommended so one ligature is removed with the splenic tissue and two ligatures remain on the tissue which stays in situ. Preservation of the left gastroepiploic and short gastric arteries is not required for preservation of gastric blood flow and therefore this technique may save time. In practice, with neoplastic cases, there is often significant neovascularisation and adhesion of the omentum to the spleen and therefore the hilar technique can, in some patients, be the safer option. The rest of the abdomen should be explored for any evidence of gross metastasis (ensure you check all surfaces of the liver thoroughly and the draining lymph nodes) and biopsies taken as appropriate. Several time saving ligation devices are now commonly available on the veterinary market:

- Harmonic scalpel: cuts via vibration. The high frequency vibration of tissue molecules generates stress and friction in tissue, which generates heat and causes protein denaturation.
- Bipolar sealant devices (e.g. Endseal or Ligasure): electrical current is passed through the tissues, denaturing proteins within vessel walls.
- LDS stapler: places two stainless steel clips to ligate the tissue within the cartridge jaw and the knife blade divides the tissue between the two closed clips

Prognosis varies significantly depending on tumour type and the presence of metastasis. Splenectomy for benign lesions such as a haematoma will of course be curative. Haemangiosarcomas are aggressive tumours and outcome depends on the tumour stage, number of gross lesions and the use of adjunctive chemotherapy. Staging of canine haemangiosarcoma depends on tumour size, presence of nodal metastasis and distant metastasis. Chemotherapy is most effective in patients with microscopic metastasis rather than gross disease. A very broad rule of thumb is a reported MST of 6-9 months with traditional adjunctive chemotherapy in patients with no gross metastasis at the time of surgery. The use of metronomic chemotherapy is also reported but discussion of this is outwith the scope of this webinar.

O is for (gastrointestinal) obstruction

The small intestine is structurally composed of four layers; mucosa, submucosa, muscularis and serosa. The mucosa is folded in to villi that increase the surface area for absorption. Two types of cell make up the mucosa; columnar epithelial cells which function in absorption and mucus producing goblet cells. Villus epithelium is replaced totally every 2-6 days. The submucosa is the supporting skeleton of the intestinal wall and binds the mucosal and muscularis layers. This is the “holding layer” and therefore must be incorporated in any closure. The intestinal muscle layer consists of a relatively thin outer longitudinal layer and a thicker inner circular layer. The serosal layer is composed of the peritoneum.

Obstruction of the gastrointestinal tract leads to excessive fluid secretion, malabsorption of water and solutes, proliferation and translocation of luminal bacteria and electrolyte and acid base disturbances. Loss of alkaline intestinal fluids usually leads to metabolic acidosis BUT high intestinal obstructions (proximal duodenum) result in hypochloremic, hypokalemic metabolic alkalosis and these patients are often much more debilitated then the typical foreign body cases on presentation. The main causes of luminal intestinal obstruction are foreign body, neoplasia, intussusception and intestinal entrapment/torsion. The obstruction may be palpable on clinical examination but radiography (plain or contrast) and ultrasound are commonly used to confirm the presence the presence of a lesion. The classic radiograph sign of a mechanical obstruction is the presence of multiple loops of gas dilated small intestine of varying diameters. Animals with a ratio of greater than 2.0 between the maximum small intestine diameter and the height of the L5 vertebral body have a high likelihood of intestinal obstruction.

The intestinal lumen contains both gram positive and gram-negative bacteria. The use of peri-operative antibiotics is still widely performed but is debated in certain quarters. An intravenous preparation should be used with an appropriate spectrum of activity (we use cefuroxime 22mg/kg IV). It should be given 30 minutes prior to the first incision to ensure adequate tissue concentrations are achieved prior to the start of surgery. The use of post-operative antibiotics is not indicated unless there is compromise of the mucosal barrier or evidence of septic peritonitis.

Intra-luminal intestinal foreign bodies are a common finding and patient stabilisation with respect to minimum database, analgesia and intravenous fluid therapy has already been discussed previously. Linear foreign bodies however do deserve a specific mention as they carry a higher complication rate. One end of the foreign body lodges under tongue or at the pylorus and the other is carried aborad by peristaltic waves. The object then becomes taut and embeds in the mesenteric border of the small intestine. They initially tend to cause partial rather than complete obstruction so clinical signs may initially be vaguer. Diagnostic techniques are similar to those previously described although plication of the intestines can be seen on some plain radiographs. A thorough oral examination should be performed after induction of anaesthesia as the string foreign body can on occasion be identified embedded in the soft tissues under the tongue.

A variety of types of neoplasia are identified in the gastrointestinal tract and staging should be performed whenever neoplasia is suspected. Usual routes of metastasis are either via the haematogenous route (liver/spleen/lungs) or via the lymphatic system.

Intussusception is the invagination of one portion of the gastrointestinal tract in to another. In young animals this is often secondary to either infectious enteritis e.g. parvovirus or a parasite burden. In older animals it is most frequently associated with neoplasia. Clinical signs are those of mechanical bowel obstruction. As the arterial blood supply often remains intact (as it is a higher-pressure system) in the face of compression of the veins, intramural haemorrhage occurs leading to loss of blood in to the intestinal lumen and subsequent haemorrhage diarrhoea. In very severe cases, small intestinal intussusceptions can progress to a point where the intestine protrudes from the anus. A cylindrical “sausage like” mass is often palpable in the abdomen of smaller patients. Radiographic signs are also similar to those described for any intra-luminal obstruction. However, on ultrasound, an intussusception has a very distinct target lesion appearing as a series of concentric rings in the transverse image.

This is a surgical condition. Manual reduction can be attempted if the enteric vessels are patent, the intestinal wall does not appear necrotic and the gross appearance is not suggestive of neoplasia. Intestinal resection anastomosis is required when the lesion cannot be reduced, ischemia of the bowel wall is apparent or neoplasia is suspected. Spontaneous reduction is infrequently reported in young animals. If blood flow is identified within the intussusception, there appears to be a higher chance of manual reduction. If resection anastomosis is performed, the tissue should be submitted for histopathology.

Enteroplication is the placement of sutures through loops of adjacent bowel to create a permanent adhesion and reduce the risk of recurrence of the intussusception. The intestines should be laid in gently curving loops to avoid any sharp bends. Sutures should pass through the submucosa to ensure an adequate adhesion is achieved. Plication remains controversial and some authors have reported an increased risk of post-operative complications with this technique but it is a technique the author still performs in clinical cases.

Surgery for gastrointestinal obstructions often involves either enterotomy or enterectomy although on occasion an intra-luminal foreign body may be able to be milked in to the colon from where it will pass in the faeces. Good visualisation is essential; do not be afraid to make a decent sized celiotomy incision. The use of good lighting, self-retaining abdominal retractors and cautery will all aid with your visualisation. The gastrointestinal tract should be run from one end to the other as there may be more than one obstruction present. Once you have identified the level of the obstruction, isolate the area of the GI tract using moist laparotomy swabs. If the obstruction is within the gastric lumen; place stay sutures of 3/0 polypropylene at either end of the incision to reduce risk of spillage of luminal contents. As discussed previously, the appearance of the tissue may improve once the intra-luminal obstruction has been removed and compression of intra-mural vasculature is removed. If in significant doubt and removal of the affected tissue may risk the development of short bowel syndrome (especially with linear FB) a second exploratory celiotomy can be planned after 24 hours when the demarcation between healthy and unhealthy tissue will be more obvious. Short bowel syndrome has been reported with resection of between 50 and 80% of the small intestine.

Suture placement is key to reducing the risk of dehiscence. Sutures should be placed 3-5mm from the edge of the tissue and around 3mm apart. Minimize how much you handle the edges of your tissue with forceps and use Debakey forceps rather than rat tooth forceps to minimize trauma and subsequent mucosal swelling. The submucosa must be included in a closure and I prefer to use simple interrupted sutures of 4/0 polydioxanone in the small intestine unless the tissue is very thick (in which case I use 3/0 polydioxanone). Simple continuous closure is also widely used. I perform a leak test of any intestinal incisions; more for peace of mind than anything else. This is certainly not a physiological test as the intra-luminal pressures achieved are likely higher than that which the gut would normally be exposed to and this test is not performed by all surgeons. The lumen of the intestine either side of the incision is occluded by an assistant's fingers and 5-10ml of sterile saline introduced in to the lumen using an orange needle (depending on the size of the bowel). The incision is checked visually for any signs of leakage. All intestinal incisions should be omentalised and serosal patching can be used if you have significant concerns about tissue viability.

Two options exist for small intestinal enterectomy; hand sewn and stapled anastomosis. In hand sewn anastomosis, the first suture should be placed as a stay suture at the mesenteric border as this is most common site for leakage. Some surgeons also place a second suture as a stay suture at the anti-mesenteric border. Again, I prefer to use full thickness simple interrupted sutures of 4/0 polydioxanone but two separate simple continuous sutures and the use of skin staples have also been reported. A stapled anastomosis is performed using a linear stapling instrument with 2 interlocking halves called a gastrointestinal anastomosis stapler. These come in a variety of staples sizes and cartridge lengths and apply four staggered rows of B-shaped titanium staples with a cutting blade which divides between the second and third row of staples creating a side to side anastomosis which functions as an end to end anastomosis. A thoracoabdominal stapler is then used to close the end of the new anastomosis site. Reported benefits of a stapled anastomosis include decreased surgical time, reduced inflammation and necrosis and reduced risk of contamination. They do, however, have some cost implications and require some training. Surgical time varies greatly with procedure performed, location of surgery, experience with equipment. In dogs, there was no significant difference in mean bursting strength at 0, 4 or 21 days after surgery when comparing anastomosis closed with skin staples with hand-sewn anastomoses (Coolman et al 2000).

Clinically, anastomotic leakage rates following stapled and sutured GIAs in small animal patients are similar (0-12.5% and 2-11% respectively Tobias 2007). However, a recent paper (Davies et al 2018) reported a lower dehiscence rate in patients with pre-existing septic peritonitis when a stapled technique was used (stapled 9.7%, hand-sewn 28.9%). Snowden et al (2016) reported an 11% dehiscence rate in 53 stapled anastomoses. Ultimately anastomotic method cannot compensate for poor technique or non-viable tissues and the risk factors for dehiscence of the surgical site are similar regardless of technique.

Septic peritonitis:

Septic peritonitis is the development of a life threatening intra-abdominal infection secondary to leakage of ingesta from the lumen of the gastrointestinal tract. Possible aetiologies include ruptured neoplasia, rupture of ischaemic stomach or bowel wall, iatrogenic secondary to surgical intervention and perforating injury e.g. dog bite wound. Many risk factors for dehiscence have been reported but the majority are based on retrospective case series and many contradict each other. Hypoalbuminaemia, intra-operative hypotension, pre-existing peritonitis, a plasma lactate concentration >2.5mmol/L and the location of leak have all been previously reported as risk factors.

Sepsis is important as it remains one of the biggest killers in both human and veterinary medicine. Sepsis leads to activation of the immune system, endothelial damage and coagulopathy, inflammation and vasodilation, increased oxygen demand, decreased oxygen delivery, decreased circulating volume, vasodilation and myocardial depression, metabolic derangements and mitochondrial dysfunction – quite the list! It is therefore not simply a case of adding in antibiotics to your regime and certainly antibiotic therapy will not prevent intestinal wound dehiscence.

Clinical signs of septic peritonitis include depression, anorexia, abdominal pain, vomiting, pyrexia, abdominal distension, tachycardia and collapse. Stabilisation is similar to that discussed earlier with respect to GDV. Hydroxyethyl starches should be avoided and gelatins used in their place. It is important to assess total protein/albumin levels as many of these patients are candidates for fresh frozen plasma transfusions. This aims to replace clotting factors as well as plasma proteins but can prove expensive for larger patients. Abdominal radiography may demonstrate reduced serosal detail and or free gas within the abdominal cavity. However, the quickest way to achieve diagnosis is by looking for free abdominal fluid, performing abdominocentesis and cytology. The presence of intracellular bacteria within leucocytes would be suggestive of septic peritonitis and surgery should be performed as soon as the patient is stable. Septic peritonitis is ultimately a surgical disease.

Anaesthesia for such cases is, as expected, a challenge; complicated by hypovolemia, hypotension, peripheral vasodilation, hypogylcemia/electrolyte and acid base disturbances. Ensure adequate volume status, treat cardiac arrhythmias if appropriate and correct electrolyte status if possible. Vasodilation and loss of vascular tone can be due to systemic inflammation and infection and if hypotension persists, vasopressors may be required. Noradrenaline (Norepinephrine) is used when hypotension persists after volume resuscitation. It increases mean arterial pressure, whilst maintaining cardiac output, heart rate and stroke volume (0.1-2 mcg/kg/min as a constant rate infusion). Adrenaline (Epinephrine) is used when noradrenaline is not effective. Vasopressors should only be used in patients that are supervised at all times and with effective blood pressure monitoring.

Surgical exploration has been described above; ensure an adequate size of celiotomy incision. A Poole suction tip will aid with evacuating the exudate from the abdominal cavity as its multiple holes do not all become blocked by the omentum. If it is possible to debride the edges of the affected tissue and close, this is preferable to performing a resection anastomosis as there is a smaller surface area for further leakage. Once the area has been debrided, closed and omentalised, the abdomen should be thoroughly lavaged. There is much debate about the correct volume but my general rule of thumb is to lavage until the lavage fluid runs clear.

We also routinely place a Jackson-Pratt closed suction abdominal drain and an oesophagostomy tube in these patients. The drain should be placed between the liver and the diaphragm to as this is the most effective location for drainage and there is a reduced risk of blockage by omentum.

Post-operative medication	Post-operative nursing
Analgesia Opioids Lidocaine CRI Paracetamol (NOT cats) NO NSAIDs Proton pump inhibitor Anti-emetics Maropitant Metoclopramide Intravenous antibiotics Intravenous fluids	Rectal temperature TID Body weight BID Fluid “ins and outs” PCV/TP/electrolytes IDUC Cytology can be performed on drain fluid

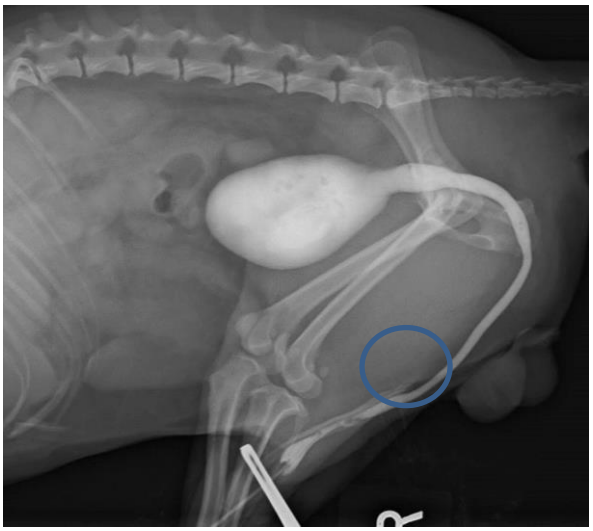
The prognosis for septic peritonitis is variable. Survival rates of between 45 and 85% are reported. Recurrent dehiscence sits at the lower end of that range. Furthermore, finances are often an issue as management of these patients is often very expensive. My personal experience is that aggressive and early management generally has better success rates. Patients where total protein levels start to climb by day three to four post-surgery often seem to have a better chance of discharge from the hospital.

Management of urethral obstructions:

Dogs:

There are a number of possible aetiologies underlying urethral obstruction in dogs and these include: urethral calculi, neoplasia (prostatic/urethral), granuloma, stricture, urethral trauma, prostatic disease and reflex dyssynergia. Of these, urolithiasis is the most common and a number of significant risk factors have been identified depending on the stone type; urinary tract infection (e.g. struvite uroliths), sex (e.g. cysteine uroliths), breed (e.g. calcium oxalate uroliths in Yorkshire Terriers), obesity and urine retention. Struvite and calcium oxalate uroliths remain the most commonly reported although the increase in popularity of the bull breeds (e.g. French Bulldog) has increased the incidence of cysteine urolithiasis. Clinical signs associated with urethral obstruction include polliakuria, unproductive stranguria, haematuria, an inability to produce a normal stream of urine, lethargy, bladder distension, abdominal discomfort and a previous history of urolithiasis or urinary tract infection. Total obstruction results in uremia within two to three days and subsequently death. Upon presentation, intravenous access should be obtained and appropriate fluid therapy and analgesia should be provided. As discussed previously, a NSAID would not be an appropriate choice of analgesic in a case such as this until urine drainage has been established, hydration status corrected and any pre-existing post-renal azotemia has resolved.

Diagnosis is usually made on the basis of contrast radiography. Haematology and biochemistry profiles may be normal unless the obstruction is complete or there is concurrent disease. Ionised calcium measurement should be performed if available as hypercalcemia is a risk factor for formation of calcium oxalate urolithiasis. Urinalysis including sediment examination and urine bacterial culture should also be performed. If a persistent urinary tract infection is suspected and cannot be proven with a routine urine culture, a bladder mucosal biopsy can be considered as this is a more sensitive method for confirmation of infection. The tissue sample should be wrapped in a section of sterile swab which has been moistened with a small volume of sterile saline to prevent the tissue drying out during transit.



Approximately one quarter of plain radiographs produce false negative findings when looking for uroliths and urethral calculi are the most commonly missed stones. Abdominal ultrasound can be used to identify uroliths in the upper urinary tract and bladder but cannot be used to accurately image the whole urethra. Documentation of the presence of bladder wall pathology, however, is very useful.

Contrast radiography (retrograde (vagino)urethrogram) is therefore essential to determine the location and number of urethral stones. Be aware an accurate urolith count is only achieved approximately 50% of the time when radiography is performed. Once the location of the obstruction has been identified, an attempt should be made to retropulse the stone back to the bladder lumen for ease of removal. If the bladder is overly distended, decompressive cystocentesis can be performed prior to retrohydropulsion. This should be performed under general anaesthesia and a combination of warm saline and water-soluble lubricant is the ideal mix.

For retrohydropulsion, a well lubricated rigid dog catheter should be inserted to the level of the obstruction. The size of catheter will vary with patient size but as large a size as possible should be chosen to ensure maximal urethral distension. An assistant should insert a lubricated gloved finger in to the rectum, identify the urethra and occlude the lumen. The surgeon should then distend the urethra with the saline/lubricant mix (compressing the tip of the penis to prevent back flow of the fluid). This creates a fixed section of urethra and distension of this will lift the urethra mucosa from the stone surface. When the assistant can feel distension of the urethral lumen under their finger, they should be instructed to release the luminal compression, allowing an influx of fluid retrograde towards the bladder lumen with a viewing the propelling the stone back to the bladder. This should be repeated multiple times to return all uroliths back to the bladder. A retrograde urethrogram should be performed once a urethral catheter passes easily to the bladder lumen to confirm all stones have been repositioned. Passage of a urethral catheter alone does not prove a completely unobstructed urethral lumen. If the uroliths cannot be repositioned, options for management are either to obtain temporary urinary diversion for 24-48 hours to allow any urethral mucosal swelling to reduce via placement of a temporary cystostomy tube or urethrotomy to remove the stone from the urethral lumen.

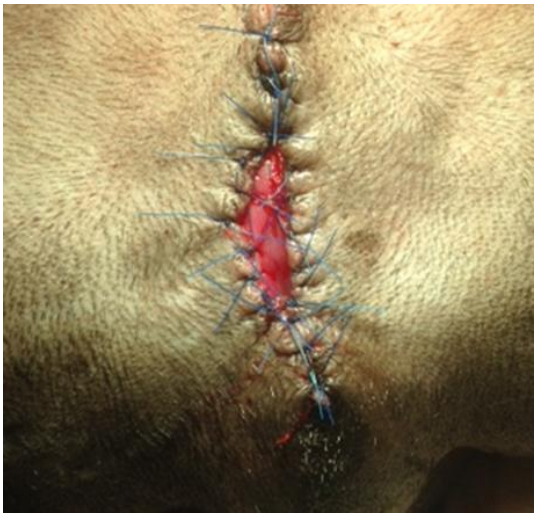
If retrohydropulsion is successful and the uroliths are successfully returned to the bladder, they should be removed via a caudoventral midline celiotomy and cystotomy. Place stay sutures in the apex and lateral aspects of the bladder to minimise handling of the mucosa. If a positive bacterial culture has not been obtained prior to surgery, a small sample of the bladder wall mucosa can be submitted for bacterial culture. Bear in mind that uroliths will likely have fallen back in to the proximal urethra during positioning for surgery. Therefore, it is important to flush the urethra from the tip of the penile urethra advancing the catheter slowly and flushing every few centimetres and repeat this several times.

It is possible to pass a catheter past a urolith under some circumstances – ensure the catheter passes EASILY in both antegrade and retrograde directions prior to closure of your cystotomy.

If uroliths remain lodged in the urethra (most commonly at the caudal end of the os penis) and cannot be moved, an urethrotomy should be performed. This cannot be performed if the obstruction is within the os penis itself unless it is at the caudal end in which case sometimes a small pair of forceps can be passed from an urethrotomy incision caudal to the end of the os penis. Urethrotomy incisions can be performed in the perineal region but be aware the dissection is challenging and there is a significant amount of vascular corpora cavernosa tissue. For a pre-scrotal urethrotomy a 2cm skin incision is made caudal to the os penis and cranial to the scrotum. The retractor penis muscle is identified and retracted laterally. The urethra can be identified as a purple structure 3-4mm wide which is bordered in either side by white corpora cavernosa tissue. Incise over the urolith or catheter if possible (expect profuse haemorrhage which can usually be controlled with direct pressure) and remove the obstruction. Advance your catheter and flush both in both antegrade and retrograde directions. Cystic calculi can then be removed via a cystotomy if present. The author prefers to suture urethrotomy incisions (4/0 or 5/0 poliglecaprone or polydioxanone) in a simple interrupted or simple continuous pattern but second intention healing is also reported. It is useful to suture over a catheter to prevent damage to the contralateral wall of the urethra. More haemorrhage is reported when the urethrotomy incision is not closed. It should not be necessary to leave an indwelling urethral catheter in situ.

Permanent damage to the distal urethra, a persistent luminal obstruction or a history of repeated urethral obstruction due to chronic urolithiasis may lead the surgeon to opt to pursue permanent urinary diversion. In male dogs, urethrostomy can be performed at pre-scrotal, scrotal, perineal and prepubic locations. Scrotal urethrostomy is preferred as the urethra is relatively superficial and wider at this location and less haemorrhage is seen post-operatively. Expect profuse haemorrhage from the urethra upon incision but do not attempt to cauterise this. Firm pressure with a swab can help stem such haemorrhage. Take care not to traumatise the mucosa on the dorsal urethral wall when making your incision as this can lead to further haemorrhage. Ensure precision apposition of the urethral mucosa to the skin as this will reduce persistent haemorrhage post-surgery – start at caudal end as this is place which is most likely to be subject to subcutaneous urine leakage. At the end of surgery, it should be possible to easily advance a catheter from the stoma site to the bladder.

An elliptical incision is made around the base of the scrotum or scrotal remnant and a standard castration and scrotal ablation performed. The retractor penis muscle is identified and freed from its attachments to the urethra then retracted laterally. The tunic of the penis is then tacked to the subcutaneous tissue using two rows of simple continuous suture to reduce dead space. A small incision is made through ventral wall of the urethra on the midline (ideally over a catheter) and the incision is then extended using fine scissors to 2.5-4cm length to ensure adequate stoma size post healing. The urethral mucosa is then apposed directly to skin using fine 4/0 or 5/0 non-absorbable suture (author prefers simple interrupted pattern).



Post-operative management and client education is important following scrotal urethrostomy. A rigid protection collar must be in place at all times. Haemorrhage from surgical site at the end of urination or at excitement should be expected on average for three to seven days post-surgery and owners should be made aware of this as although it is usually self-limiting, it can be very dramatic and sedation can be required. Therefore, it may be worth considering hospitalising patients during this period. The stoma should not be cleaned as this will lead to further haemorrhage. Short term complications include persistent haemorrhage, subcutaneous urine leakage which requires a second surgery and stricture formation. Long term complications include urine scalding of skin (pre-scrotal and pre-pubic locations) and an increased risk of urinary tract infection.

Uroliths should be submitted for analysis to determine the type. This can be done for free via the Hill's service so there really is no excuse not to do it. Spilt feeding is advised to reduce urine pH swings associated with eating and a specific diet should be chosen based on urolith analysis if appropriate. Increase water intake (aim for a USG of less than 1.020). If infection was previously documented, repeat cystocentesis and urine bacterial culture seven days after antibiotic course finishes to confirm resolution of the infection. Advise owners about weight reduction in obese patients and consider neutering in entire male dogs with cysteine uroliths (as androgens are a pre-disposing factor). Increase exercise to ensure more frequent urination and ensure regular urine sediment monitoring – initially monthly. Samples should be examined within one hour of collection (i.e. done in house) and not sent to an external laboratory for analysis as crystals may form during transit.

Cats:

Urethral obstruction in cats is most commonly associated with urethral spasm and feline lower urinary tract disease (FLUTD). A full and frank discussion of FLUTD is beyond the scope of these lectures. Other causes of urethral obstruction in cats include urethral plugs, uroliths, stricture and trauma. Upon presentation, correction of fluid and electrolyte disturbances should be prioritised and you should ensure adequate opioid analgesia (please see above section on urethral trauma). NSAIDs should avoided for obvious reasons until the patient is stabilised.

Attempted urethral catheterisation should be performed under general anaesthesia as this will significantly increase your chances of success. If the bladder is very full, consider cystocentesis prior to catheterisation but ensure the owner gives consent and is aware of the risk of bladder rupture. The site of obstruction is usually the penile urethra as it is the narrowest portion of the feline urethra. It is therefore useful to massage the penis within the prepuce to try and dislodge any urethral plugs. If a catheter will not pass, try to pass a 22G or 24G intravenous catheter first (without the stylet) and flush the urethra with sterile saline.

Urinary catheter selection is very important; rigid Jackson cat catheters lead to significant trauma and should be avoided if at all possible. Ideally a silicone cat catheter is recommended, particularly if you plan to leave the catheter in situ. Once the obstruction has been relieved, a retrograde urethrogram should be performed to identify any other pathology such as stricture or uroliths. The bladder should be lavaged with sterile saline or another such isotonic solution.

The duration of catheterisation remains controversial and a decision must be made on a case by case basis depending on the underlying pathology and the duration and severity of clinical signs. Eisenburg et al suggested a longer duration of catheterisation was positively associated with a reduced risk of repeat obstruction. The IDUC should be sutured in situ (if desired) and connected to a closed drainage system. Leaving an IDUC open to the environment is unacceptable and significantly increases the risk of urinary tract infection. Adequate analgesia should be provided and meloxicam can be introduced at an appropriate juncture depending on renal function. The use of a smooth muscle relaxant such as prazocin is also recommended. Diazepam can cause liver injury in some cats (Beusekom et al 2015) and therefore it's use should be carefully considered. Dantrolene can also be used.

Post-obstruction diuresis is a common finding after relief of a complete urethral obstruction and close monitoring of both electrolytes and fluid requirements is necessary. Cooper et al (2010) reported a technique for managing urethral spasm in male cats without placement of a urethral catheter due to financial constraints. This technique, involving use of sedation, analgesia, a calm environment and decompressive cystocentesis was successful in 11 of 15 cats. The use of intra-luminal lidocaine or atracurium is also reported. In cats with a recurrent history of obstruction or with an obstruction which cannot be relieved, permanent urinary diversion can be achieved via a perineal, transpelvic or pre-pubic urethrostomy. Owners should be made aware that such surgeries do not prevent clinical signs associated with FLUTD, they simply aim to avoid the life-threatening emergency of urethral obstruction.

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