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Emergency Surgery Mini Series

Session 2: Emergency surgery of the gastrointestinal tract

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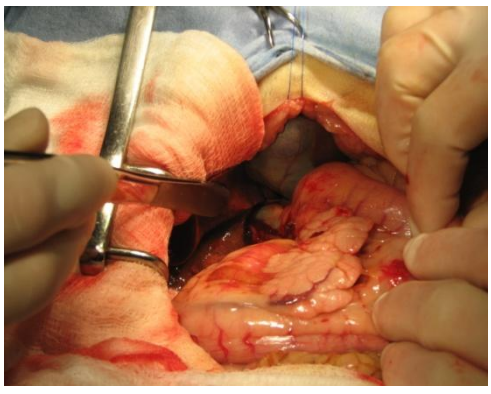

Introduction:

Today's webinar will aim to provide an introduction to some of the common gastrointestinal surgical emergencies seen in veterinary medicine. The majority of the time will be sent addressing pre- and post-operative management of these cases and with good reason. Whilst the surgery itself may be complex, ultimately a good outcome is often determined as much by attention to the non-surgical management as the surgery itself. The importance of early nutrition is stressed throughout the presentation and is one of the keys to success in managing gastrointestinal surgical cases.

Oesophageal foreign bodies:

Oesophageal foreign bodies are more commonly identified in dogs than cats. They most commonly lodge at the thoracic inlet, heart base or caudal oesophagus due to the presence of structures external to the oesophagus which constrict dilation at these points; more than 60% are located between the heart base and the diaphragm. Terrier breeds are over-represented (Hayes 2009, Deroy et al 2015).

Clinical signs vary depending on the degree of the obstruction. Complete obstructions generally lead to acute clinical signs. Signs include regurgitation of food within a few minutes of eating (usually water can pass the obstruction unless it is completely blocking the lumen), retching, gagging, hypersalivation, lethargy and Inappetance. Oesophageal perforation will lead to pneumomediastinum, mediastinitis, pneumothorax and pyothorax and affected animals will demonstrate respiratory signs as will animals which develop aspiration pneumonia secondary to regurgitation.

Diagnosis can usually be made on plain radiographs. Bone foreign bodies are easily identified as they are mineralised. Non mineralised foreign bodies may be identified as an increase in soft tissue opacity and dilation of the oesophagus cranial to the obstruction with air. Radiographs should be examined closely for signs of pneumomediastinum, pneumothorax, pneumonia and pleural effusion. Endoscopy is both diagnostic and therapeutic.

If possible, oesophageal foreign bodies should be removed endoscopically. This is reported to be successful in around 90% of cases (Gianella et al 2009) with a 12.9% complication rate (predominantly perforation). If the foreign body cannot be retrieved per os, it can be advanced in to the stomach and either removed via a gastrotomy or left to dissolve (if bone). If endoscopic retrieval is successful, the oesophagus should be examined following removal of the foreign body to assess the degree of mucosal damage and also identify possible perforation of the wall if necrotic. Any deterioration in oxygen saturation or difficulty ventilating the lungs should raise suspicion of perforation and development of a pneumothorax. If there is significant mucosal damage or questionable tissue viability after PEG removal, a percutaneous endoscopic gastrostomy tube (PEG) should be placed. If retrieval or advancement of the foreign body is not successful, surgical retrieval is required. For obstructions located in the cervical oesophagus, the oesophagus should be approached via a ventral midline approach. Ensuring there is a stomach tube in the lumen aids with identification. A two layer closure using monofilament absorbable suture is recommended. Very caudally located obstructions may be able to be accessed from a gastrotomy. If the obstruction is located within the thoracic oesophagus, an intercostal thoracotomy is required. If surgical retrieval is required, a PEG tube should be considered.

Postoperatively omeprazole or an H2 receptor antagonist should be given and any aspiration pneumonia treated as we discussed in the last webinar. If there is severe oesophageal trauma or oesophageal trauma, food should be withheld by the oral route for 3-7 days If there is minimal mucosal trauma, water and then food can be offered after 12-24 hours. Complications include esophagitis, ischaemic necrosis of the oesophageal wall, broncho-oesophageal fistula formation, stricture and diverticulum formation. The outcome is generally very good except in cases of thoracic

oesophageal perforation where the prognosis is more variable. Gianella et al 2009 reported bone foreign bodies, a bodyweight of less than 10 kg, and oesophageal or gastric FB in place for more than three days were significant risk factors for complications.

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, Weimaraners, 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 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.

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. A right lateral abdominal radiograph can 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.

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 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 and certainly there is an argument that the use of such colloids should be avoided in septic veterinary patients also. 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 hydroxylethyl starches but this is not very common in dogs. Gelatins are currently the colloid of choice in human sepsis. Colloids are usually administered in 5ml/kg boluses up to a maximum of 20ml/kg/24 hours.

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. 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

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.

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 are to decompress and reposition the stomach, assess the viability of the stomach wall and 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 used in a clinical setting. 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 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 can be challenging. If the patient is stable, an oesophagostomy tube should be placed prior to recovery from anaesthesia to ensure the provision of early enteral feeding. Analgesia is essential and as previously discussed; methadone represents the most common choice possibly in conjunction with a lidocaine CRI. Lidocaine may also have a beneficial effect on survival in these patients in terms of reducing post-operative cardiac arrhythmias (Bruchim et al 2012). Maintenance of an adequate circulating volume and plasma protein levels important. 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. I routinely use intravenous omeprazole, maropitant, a metoclopramide CRI +/- intravenous antibiotic therapy depending on the appearance of the gastric wall at surgery. Electrolytes and plasma protein levels should be monitored regularly until within the normal laboratory range. Early feeding encourages peristalsis which improves intestinal blood flow and provides enterocytes with nutrition. Complications of GDV include septic peritonitis secondary to unidentified gastric wall necrosis, ileus, pancreatitis, disseminated intravascular coagulation, cardiac arrhythmias and oesophagitis/gastritis.

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 I still perform in my 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 certainly 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). 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 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 (Norepinepherine) 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 (Epinepherine) 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 but ensure an adequate size of celiotomy incision. A Poole suction tip will aid with evacuating the exudate from the abdominal cavity as it 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.

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.

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. Medical management and 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 usually a decision made on the basis of the ultrasound findings and the blood work although sometimes this is a decision which can only be made at surgery. If this cannot be established, a cholecystoduodenostomy is the procedure of choice. 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.

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 adhesed to the diaphragm, neuromuscular blockade is useful to aid in dissection of any adhesions. 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 and oversewn with suture or ligated with a VA30 stapler. Our 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 therefore keep flushing to a minimum if possible. 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 20% of patients die prior to discharge from the hospital) and this should be discussed with the owner prior to surgery. An oesophagostomy tube should be placed in these patients at the time of surgery.

References:

Bruchim, Y., Itay, S., Shira, B.-H., Kelmer, E., Sigal, Y., Itamar, A. and Gilad, S. (2012), Evaluation of lidocaine treatment on frequency of cardiac arrhythmias, acute kidney injury, and hospitalization time in dogs with gastric dilatation volvulus. Journal of Veterinary Emergency and Critical Care, 22: 419–427

Deroy, C., Corcuff, J. B., Billen, F. and Hamaide, A. (2015), Removal of oesophageal foreign bodies: comparison between oesophagoscopy and oesophagotomy in 39 dogs. J Small Anim Pract, 56: 613–617.

Evans KM, Adams VJ (2010) Mortality and morbidity due to gastric dilatation-volvulus syndrome in pedigree dogs in the UK. J Small Anim Pract. 2010 Jul;51(7):376-81.

Gianella, P., Pfammatter, N. S. and Burgener, I. A. (2009), Oesophageal and gastric endoscopic foreign body removal: complications and follow-up of 102 dogs. Journal of Small Animal Practice, 50: 649–654

Goodrich, Z. J., Powell, L. L. and Hulting, K. J. (2013), Assessment of two methods of gastric decompression for the initial management of gastric dilatation-volvulus. J Small Anim Pract, 54: 75–79

Loy Son, N. K., Singh, A., Amsellem, P., Kilkenny, J., Brisson, B. A., Oblak, M. L. and Ogilvie, A. T. (2016), Long-Term Outcome and Complications Following Prophylactic Laparoscopic-Assisted Gastropexy in Dogs. Veterinary Surgery.

Mackenzie G1, Barnhart M, Kennedy S, DeHoff W, Schertel E. A retrospective study of factors influencing survival following surgery for gastric dilatation-volvulus syndrome in 306 dogs. J Am Anim Hosp Assoc. 2010 Mar-Apr;46(2):97-102.

Pipan M1, Brown DC, Battaglia CL, Otto CM. J Am Vet Med Assoc. 2012 Jun 15;240(12). An Internet-based survey of risk factors for surgical gastric dilatation-volvulus in dogs.

Przywara, J. F., Abel, S. B., Peacock, J. T., & Shott, S. (2014). Occurrence and recurrence of gastric dilatation with or without volvulus after incisional gastropexy. The Canadian Veterinary Journal, 55(10), 981–984.

Snowdon, K. A., Smeak, D. D. and Chiang, S. (2016), Risk Factors for Dehiscence of Stapled Functional End-to-End Intestinal Anastomoses in Dogs: 53 Cases (2001–2012). Veterinary Surgery, 45: 91–99