Emergency Surgery Mini Series

Session 2: Surgical Emergencies of the Gastrointestinal System

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TABLE OF CONTENTS

The Acute Abdomen

Assessment, Stabilisation, Exploratory Laparotomy & Biopsy Techniques

The 'Acute Abdomen' case is a patient that presents with an abdominal disorder that has led to profound systemic signs (such as hypovolemic or septic shock, sepsis and/or severe abdominal pain).

The approach to the management of these patients involves the rapid assessment of the likely cause and of the degree of systemic problems that the patient is experiencing. A plan for rapid stabilisation coupled with focused diagnostic techniques is implemented and a decision on the requirement for surgical intervention is then made.

Possible causes of Acute Abdomen include:

- Gastrointestinal foreign body

- Gastric dilatation-volvulus

- Intussusception

- Perforation of the Gastrointestinal tract

- Uncontrolled abdominal haemorrhage

- Pancreatitis

- Septic peritonitis

- Abdominal abscess – pancreatic, splenic, prostatic, renal, hepatic

- Penetrating trauma

- Abdominal ruptures/hernia with incarceration and possible strangulation of contents

- Abdominal neoplasia

- Urethral obstruction and Bladder rupture

- Pyelonephritis

- Hepatitis/Cholecystitis/Cholelithiasis

- Musculoskeletal pain – fracture, etc

Exploratory laparotomy

The term laparotomy technically only refers to a surgical incision through the flank, but in common usage has come to mean any surgical approach into the abdominal cavity. The scientifically correct term for a surgical incision into the abdominal cavity is a coeliotomy. A laparotomy, most commonly a ventral midline laparotomy, is obviously indicated as a prelude to the surgical manipulation of the abdominal organs as with an enterotomy or ovariohysterectomy. The exploratory laparotomy is however indicated as a diagnostic procedure when the history, clinical signs and diagnostic tests indicate an abdominal problem,

but the exact diagnosis is unknown. This may be performed as an emergency procedure or electively for more chronic conditions.

There are therefore a range of possible indications, but the correct timing of this diagnostic step within the diagnostic plan for a problem will depend on a number of factors. The most important is probably the presenting clinical signs and status of the patient, but other factors such as the access to appropriate diagnostic tests/imaging, the clinical skills and experience of the responsible clinician, the postoperative care facilities available, and the financial status of the client all figure in deciding to proceed. Appropriate diagnostic tests to determine medically-manageable from surgically-managed disease are obviously important to avoid unnecessary surgery. Unfortunately there are many instances of dogs and cats operated on needlessly due to a failure to perform or interpret diagnostic tests or due to an inadequately performed procedure. This can lead to the unfortunate situation of a clinically worse and painful patient and a financially embarrassed client.

Preoperative considerations
Physical examination:
A complete and thorough clinical examination is essential if there is a high index of suspicion for an abdominal disorder. The signs of abdominal disease are however often vague and can be confusing. The animal with severe abdominal pain may be interpreted by its owner to have difficulty breathing. Noticeable and palpable abdominal effusions generally involve quite large volumes of fluid and in some conditions this may take a long time to collect – determining the nature of the ffusion is paramount in assessing the patient. Dullness, depression and anorexia may be the only initial signs such as with bile peritonitis which may not become more clinically obvious for up to a week. Palpating for abdominal pain may be very difficult in the obese or excited animal whereas may not elicit any response in the severely depressed animal.

Stabilisation:
Patient stabilisation is the first priority. Most patients with an "acute abdomen" have an inadequate intravascular volume due to hypovolaemia (due to loss of fluid/blood into the abdomen +/- fluid loss from inappetance, vomiting and diarrhoea) and possibly maldistribution (traumatic, septic or cardiogenic in origin).

In assessing these patients awareness of the three stages of shock – Compensatory, Early Decompensatory and Decompensatory is necessary.

Compensatory stage:

Mild to moderate loss of intravascular volume leads to sympathetic and neurohormonal responses that increase intravascular volume and cardiac output. A hypermetabolic hyperdynamic state occurs.

Clinical signs:

- Mild increases in HR, RR; Normal mentation and BP
- 'Brick' red mucous membranes with capillary refill time <1sec
- SIGNS OFTEN OVERLOOKED – adequate fluid therapy required to reverse

Early Decompensatory stage:

Compensatory stage cannot be maintained indefinitely if there is a continued loss of intravascular volume/lack of support. Redistribution of blood flow occurs to the heart and brain (priority organs). Corresponding decrease in blood flow to the rest of the organs leading to decreased tissue oxygenation which causes lactic acidosis and tissue hypoxia (and further local effects).

Clinical signs

- Tachycardia, Tachypnoea
- Pale MM, poor CRT, weak pulse, poor mentation
- Usually hypothermic and hypotensive
- AGGRESSIVE fluid therapy now required

Decompensatory (terminal) stage:

Prolonged tissue hypoxia/acidosis leads to autoregulatory escape leading to massive vasodilation in all organs. Complete circulatory collapse ensues as blood pools in the peripherary and there is a failure to meet the heart and brain requirements leading to death.

Clinical signs

- Low heart rate
- Absent capillary refill time. Pale or cyanotic MM. Profound hypothermia
- Severe hypotension
- Successful treatment is usually no longer possible.

Initial treatment priorities are to gain good vascular access, start appropriate fluid therapy, supplement with oxygen and assess the response.

Unfortunately many cases only present in early decompensatory shock or the initial signs of compensatory shock are missed. Aggressive fluid therapy with 'shock' doses of lactated Ringers solution (90ml/kg/hour in dogs) is therefore indicated. Although the 90/ml/kg/hr level of fluids is well known this should be given in an incremental manner rather than over a full hour. I usually start at this rate of fluids and then reassess the dog's parameters and requirement for continuing at this rate after 10-15 minutes. If still necessary I will continue this fluid level and reassess again after a further 15 minutes. Catheters in at least both cephalic veins or a central jugular catheter (if not placed preoperatively, definitely place a central line once anaesthetised) will be needed to allow administration at this rate. Care must be taken in cats to avoid fluid overload (shock dose of crystalloids 55ml/kg/hr but again give this in boluses at this rate for 10-15mins as required).

Crystalloids are relatively cheap and can be rapidly delivered, but have the downside that approx 75-85% will move out of the circulation and into the interstitial space within an hour of administration. It therefore is difficult to maintain the intravascular volume and tissue perfusion in these patients with crystalloids alone. Also the movement to the interstitial space can lead to interstitial oedema (especially serious in the lung and brain). They should not be used therefore as the sole source of intravascular fluid support for most patients Synthetic colloids (such as hetastarch) have the major advantage of being retained within the intravascular space for 12-48 hrs (plasma half-life of 25.5hrs). They can therefore be useful as the initial and continued (postoperative) means of supporting the intravascular space. Hetastarch may be administered in 5-10ml/kg boluses over 10-15 minutes (they can be administered more rapidly in the dog). The dosage over 24 hours is 10-20ml/kg/24hrs in the dog (this can be increased to higher levels in the dog, but a clear understanding of possible complications and close monitoring is essential) and 10-15ml/kg/24hrs in the cat. In the dog, constant rate infusions of 20ml/kg/day can then be useful if continued colloid support is required.

If required, blood or plasma transfusions can be considered to correct anaemia or severe hypoproteinaemia. As regards hypoproteinemia, large volumes of plasma are required to increase the serum albumin as 22.5ml/kg of plasma is required to raise the albumin by 5g/l. The half-life of this albumin in the circulation is just 16 hrs, so that its use to support intravascular colloidal osmotic pressure and volume expansion is limited (synthetic colloids such as hetastarch are more effective at this).

Another alternative is the use of the haemoglobin based oxygen carrying solution Oxyglobin (5-10ml/kg/hr) and this has been shown to be beneficial in the resuscitation of the patient in hypovolemic shock.

Oxygen is also provided as this can greatly aid tissue oxygenation in the dog in hypovolemic shock. Even small increases in the amount of inspired oxygen can lead to major improvements especially in the patient with impaired ventilation (as with GDV).

The requirement for preoperative stabilisation of the patient will depend on the type and intensity of the underlying condition. In most cases provision of 1-3 hours of preoperative intravenous fluids will better prepare the patient for the anaesthetic and surgery but this may need to be lengthened or curtailed depending on the underlying problem, status of the patient and the urgency of surgery. The goal is the partial correction of fluid and electrolyte imbalances over the preoperative and anaesthetic period with complete correction in the postoperative period. Depending on the underlying problem and the severity of the derangements there is generally always a requirement for crystalloids, with colloids (and occasionally whole blood or oxyglobin) necessary in more critical patients.

Diagnostic investigation:
Laboratory tests: A baseline of PCV/total protein, BUN, creatinine, and urine specific gravity is essential, to give an accurate picture of the current clinical status of the patient and to allow comparison to a pre-op baseline postoperatively. A complete haematological and biochemistry panel is ideal particularly if there are concerns of concurrent/underlying conditions but may depend on the urgency of the procedure and the availability of a quick laboratory service.

Diagnostic imaging:
- Radiography: A good quality radiograph obtained with the patient in lateral recumbency allows evaluation of most abdominal organs, with an orthogonal ventrodorsal radiograph to further assess the location and significance of the lesion identified. Taking both right and left lateral radiographs may allow better determination of the lesion due to displacement of the gastric/intestinal gas. Obvious abnormalities may clearly support a decision for exploratory surgery. Repeating radiographs a few hours later may give further information on the progression of a lesion (for example to see if an intestinal foreign body is causing an obstruction or is passing through the intestinal tract). Further imaging such as radiographic contrast studies or ultrasonography is indicated in the absence of obvious abnormalities. Survey chest radiographs to assess for lung metastases is appropriate with abdominal masses prior to exploratory laparotomy.

- Ultrasonography: Abdominal ultrasonography has largely superceded gastrointestinal contrast techniques and is an extremely valuable adjunct to taking plain abdominal radiographs. This does however require considerable experience and appropriate transducers to perform a complete assessment as it is very easy to misinterpret,

misdiagnose or overlook ultrasonographic images if you are inexperienced with normal findings. In experienced hands it allows accurate detection and assessment of abdominal lesions. This then allows targeted collection of aspirates or Tru-cut biopsy samples and small volumes of free abdominal fluid.

Abdominocentesis:

Obtaining a sample of free abdominal fluid may give a rapid indication for the requirement of exploratory laparotomy. For example evidence of degenerate neutrophils and intracellular bacteria gives a diagnosis of septic peritonitis. With most peritoneal diseases, copious free abdominal fluid usually collects and collection of a sample using a sterile four quadrant tap technique is usually easy and straightforward. Avoiding the cranial abdominal midline is important to avoid the falciform fat. It has been shown that at least 25ml/kg of free fluid is required for this technique to be successful, so a negative tap does not rule out a peritoneal effusion. Diagnostic peritoneal lavage (DPL) can improve the diagnostic yield. This is performed by inserting a DPL catheter sterilely, infusing 22ml/kg of warmed Hartmann's solution under gravity, rolling the patient and then collecting 10-20ml of fluid by free drainage. DPL has now been largely superceded by ultrasound-guided aspiration.

In house assessment of samples can be usually easily performed to determine fluid PCV, specific gravity, total protein, and most importantly cytological characteristics.

Anaesthesia:

Anaesthetic management will depend on the underlying disease and stability of the patient. Thorough assessment and consideration of the age, health status, concomitant disease and proposed surgery will determine the premedication and induction agents. As there is often a good chance of a full stomach with an acute patient, use of cuffed endotracheal tubes is essential. The possibility of oesophageal reflux should also be anticipated and the patient should be positioned with its head elevated. Tilting the table to allow this also decreases the pressure on the diaphragm if there is a large abdominal mass or ascites present. If reflux occurs, the patient should have its oesophagus thoroughly lavaged before recovery with a stomach tube and water and appropriate drugs such as sucralfate and H2 antagonists/H2 blockers should be provided in the postoperative period.

Adequate perioperative analgesia must be provided. The use of opiate analgesia will provide the best pain management in the perioperative period. Avoid the use of non-steroidal anti-inflammatories, particularly in the sick or dehydrated patient. Avoidance of hypothermia is very important during anaesthesia and particularly exploratory laparotomy as the heat loss from exposed abdominal organs is considerable. Ensuring that the patient does not become hypothermic before surgery is critical as improving body temperature during anaesthesia once it is lost is very difficult. Use of insulation of the head and feet, warming devices such as hot

air blankets (BairHugger) or warm water circulating mats, and warm saline for irrigation is important.

Antibiotics:
Perioperative broad-spectrum antibiotics are indicated at an early stage in the patient in shock or if the gastrointestinal or urogenital tract are to be entered or if a long surgery is anticipated, particularly in a critically–ill patient. Prophylactic use of a broad-spectrum antibiotic administered intravenously at induction of anaesthesia (30 minutes before surgery commences) and repeated at 90 minute intervals during surgery, ensures therapeutic levels of antibiotics at the time of contamination. Only if there is a specific indication to do so (unexpected leakage of intestinal contents or a break in normal asepsis in any way) is a therapeutic course of antibiotics given in the postoperative period.

For upper intestinal surgery/gastric/liver/pancreatic surgery I will administer a first generation cephalosporin, or clavulanate-potentiated amoxicillin intravenously at induction. For lower intestinal surgery I will administer a second-generation cephalosporin or amoxicillin/clavulanate +/- metronidazole at induction. Metronidazole and cephazolin (in combination) could also commenced 24 hours preoperatively.

Surgical considerations
Patient preparation
Prior to surgery, the patient is given an opportunity to urinate and defecate if appropriate.
A ventral midline laparotomy is the most versatile and easiest approach with generally the least postoperative pain and morbidity. The patient has a wide clip to allow at least extension of the approach from the xiphoid to the pubis regardless of the specific area that it is anticipated will be accessed. This means clipping at least 3-5 centimetres lateral to the nipples, 5-10 cranial to the xiphoid and to the mid/caudal pubis. The scrotum is not clipped if possible to avoid postoperative rashes. If it is possible that the approach may need to extend to a sternotomy (possible with diaphragmatic hernias, liver surgery etc) then the clip is extended as necessary. The bladder is emptied at this stage if it is palpably enlarged. The skin is then cleaned and disinfected routinely. If it is possible that the penis or prepuce will need to be accessible during surgery, this is flushed with an appropriate antiseptic (such as 10% povidone-iodine). If this will not be the case then the prepuce may not need to flushed, but is clamped to the side of the surgical field with a towel clamp. Correct stable positioning in dorsal recumbency with sandbags or ties helps to avoid frustration during surgery with a patient that moves off midline.

Impermeable reusable or disposable drapes are applied to the surgical site. Impermeable drapes are required due to the high likelihood of bacterial strikethrough with cloth drapes in the presence of lavage or body fluid. Having additional clean drapes to cover contaminated areas and changing to clean instruments and gloves prior to closure of the abdomen is important with contaminated surgery.

Surgical instrumentation

The minimum instruments that I use in a general surgical pack for abdominal surgery are:
- No.3 and No.7 scalpel handles – no. 10 and 11 or 15 scalpel blades
- Adson thumb forceps
- Debakey tissue forceps
- Curved Metzenbaum scissors
- Curved and straight Mayo scissors
- Eight towel clamps
- Five straight and five curved Halsted mosquito hemostatic forceps
- Two straight and two curved Spencer-Wells or Carmault hemostatic forceps
- Two Mayo-hegar needle holders (small and large)
- One large blunt Gelpi reatractor
- Two Allis tissue forceps
- Large stainless steel bowl
- Small stainless steel bowl
- Ten 10x10cm gauze swabs with radio-opaque strip

One of the most common and fundamental errors is too short an incision due to a failure to clip and prepare the ventral abdomen adequately. Too little access will mean inadequate exploration.

Two 30x30cm laparotomy swabs with radio-opaque strip

Sterile saline (usually use 500ml for moistening swabs with additional 2-3litres of warm saline in case of abdominal lavage)

Suction – Frazier and Poole type suction tips

Electrocautery – bipolar handpiece

Additionally as required:

For retraction:

Malleable handheld retractors

Balfour retractor (18-25cm spread - large dog)

Gosset retractor (10–15 cm spread - medium dog)   Self-retaining

Baby balfour retractor (10 cm spread - small dog)   retractors

Note: it is advisable to cover retractor blades with moistened gauze swabs to protect delicate abdominal soft tissues

For dissection:

Mixter right-angled forceps (small)

Lahey right-angled forceps (large)

Watterson dissecting forceps (non-ratcheted ringed forceps)

For clamping bowel:

Large laparotomy swabs (30x30cm) are essential for abdominal work as they can be used to absorb large volumes of blood/fluid, to protect tissues when moistened, and to elevate and pack off viscera from the rest of the abdominal cavity. Their size means that they are not easily misplaced. For these reasons they should be used instead of the smaller 10x10cm swabs.

Doyen bowel clamp (non-crushing for atraumatic use)

Lang-Stevenson bowel clamp (crushing for occlusion and excision)

Surgical anatomy:

Access for a ventral midline laparotomy is via the linea alba, the midventral aponeurosis of the abdominal muscles extending from the xiphoid to the pubis. The tough fibrous coat of the rectus abdominus muscle is composed of an internal and an external layer. The external layer is composed of this external sheath as well as the aponeurosis of the external abdominal oblique and a portion of the aponeurosis of the internal abdominal oblique muscle. This tough fibrous layer is along the length of the ventral abdomen and is the key suture holding layer. The internal rectus sheath is composed of the aponeuroses of the internal abdominal oblique, the transversalis muscle and the transversalis fascia. This is only present in the cranial two thirds of the ventral abdomen. The cranial third of the external sheath is weaker and in this area both the external sheath and the internal sheath should be engaged to ensure adequate suture holding strength.

In the male dog the prepuce will be encountered in the caudal third of the ventral abdomen. The prepuce is suspended by the preputial muscles and preputial fascia and is supplied by large preputial branches of the superficial epigastric artery and vein on each side.

Just ventral to the cranial ventral abdominal wall is the falciform fat that extends from the xiphoid to the umbilicus. This large fat reservoir is present in all dogs apart from those that are in very poor body condition. Removal of this fat greatly improves visualisation of the cranial abdominal structures.

The ventral leaf of the greater omentum extends from the greater curvature of the stomach to include the spleen on its left side. Opening between the omental leaves reveals the omental bursa and the dorsal leaf of the omentum, within which the left limb of the pancreas is situated. Opening the omental bursa allows evaluation of the rest of the greater curvature of the stomach, the left limb of the pancreas, the medial aspect of the right limb of the pancreas and the splenic vessels.

On the left side the descending colon is suspended by a fold of mesentery called the mesocolon. To examine the structures of the left lumbar gutter (left kidney, ovary, ureter, retroperitoneal space etc.), the descending colon can be drawn to the right side, thereby trapping the loops of jejunum by the 'net' of the mesocolon and moving them to the right. This is called the colonic manoeuvre.

On the right side the descending duodenum is suspended by the mesoduodenum, a fold of mesentery in which the right limb of the pancreas is suspended.

The mesoduodenum can be used similarly to the mesocolon to trap and move the loops of the jejunum to the left (the duodenal manoeuvre), thereby improving exposure of the right kidney, adrenal, ureter, ovary, caudate process of the caudate liver lobe and epiploic foramen. The epiploic foramen is at the cranial extent of the mesoduodenum and is the opening of the omental bursa into the peritoneal cavity. At this level the hepatic artery can be identified caudally within the mesoduodenum, the caudal vena cava is dorsally and the portal vein is ventrally. Care must be taken with the duodenal manoeuvre not to roughly handle the pancreas as this could elicit a pancreatitis.

Surgical Procedure

The key pitfalls to avoid with an exploratory laparotomy are:

- Failure to adequately explore due to too small an incision
  Successful exploration of the entire abdominal cavity involves a methodical systematic approach. This is obvious if the underlying condition is unknown, but it also allows detection and assessment of concurrent abdominal problems. It is important to anticipate the unexpected and be well enough prepared to deal adequately with this. Using the same consistent approach each time, builds up knowledge of 'normal', allowing the rapid detection of abnormal.

- Failure to obtain adequate biopsy material at the time of surgery
  If the animal is ill enough to warrant exploratory laparotomy, it is important to ensure that this intervention is not a waste of time and money. If abnormalities are found at the time of surgery, then these should be biopsied or at least aspirated, being aware of surrounding vital structures. If there are liver biochemical changes then liver biopsies should be taken. If there are gastrointestinal problems, then full thickness gastric and intestinal biopsies should be taken (although the risks of this should be explained to the owner and balanced against the benefits to the patient). Future decisions on treatment for the patient will depend on the accurate information obtained.

If there is a specific area of the abdomen to be explored (for example the lower urinary tract) the incision is centred over this area, although extension of the incision to investigate additional areas can then be performed. However for complete exploratory purposes the linea alba is incised from the xiphoid to the pubis.

The ventral midline skin incision should be made in a confident manner with a large enough scalpel blade (such as no.10 or 22). The incision is deepened into the subcutaneous fat staying on midline, as this is the surest way to encounter the linea alba. This relies on a well positioned straight patient. The blade does not need to be changed after the initial skin incision unless there is a pyoderma. It may be useful, particularly in fat patients to reflect the

subcutaneous fat from the external fascia for 0.5-1cm as this will improve identification of this layer on closure. Avoid extensive dissection though as this will create more dead space. Electrocautery can be particularly useful to manage subcutaneous haemorrhage especially over the cranial ventral midline.

In the male dog curve the skin incision lateral to the prepuce to make a parapreputial incision. The preputial muscle and fascia is incised, taking care to identify and ligate the large preputial branches of the caudal superficial epigastric vessels. Once incised the prepuce is then deflected laterally to identify the external abdominal fascia and linea alba.

The peritoneal cavity is entered by careful pressure from the belly of the scalpel blade or by tenting the linea alba and making a stab incision. Check for adhesions beneath the linea alba, then extend the incision with scissors.

Once entered, remove the falciform fat from the cranial abdominal cavity. Small vessels entering this from laterally and cranially may need to be ligated or cauterised. The use of an abdominal retractor such as a Balfour is very useful to keep the abdomen open and allow exploration. The blades of this instrument are cushioned from the wound edges with large moistened laparotomy swabs.

The exact routine employed to assess the abdominal cavity does not matter, as long as all organs and structures are assessed. The routine I usually employ is to use a 'quadrant' approach as this ensures examination of all areas of the abdomen (see table).

1. Right cranial quadrant	2. Left cranial quadrant
• Diaphragm • Right liver • Gall bladder and portal vessels • Gastric antrum and pylorus • Descending duodenum • Pancreas	• Diaphragm • Left liver • Terminal oesophagus • Gastric cardia, fundus and body • Spleen • Open omental bursa o Assess dorsal stomach, left limb of pancreas
3. Right caudal quadrant	4. Left caudal quadrant

• Jejunum (from duodenal flexure)
 	
• Ileum and caecum
 	
• Mesenteric vessels
 	
• Lymph nodes
 	
• Duodenal manoeuvre (right lumbar gutter)
    o Right kidney/ureter
    o Right adrenal gland
    o Right ovary/uterus
    o Vena cava
	• Ascending, transverse and descending colon
	
	• Colonic manoeuvre (left lumbar gutter)
	    o Left kidney/ureter
	    o Left adrenal gland
	    o Left ovary

5. Pelvic canal	
• Rectum • Bladder • Urethra • Prostate • Vagina and uterus	

Biopsy techniques

Biopsies are taken according to the grossly obvious lesions detected at surgery or if disease of these organs is suggested by the presenting clinical signs or the preoperative diagnostic tests.

- **Liver**

Good size mid-parenchymal samples can be obtained using a sterile cutaneous biopsy punch. The punch is introduced in a rotating manner perpendicular to the liver surface, partially withdrawn and then reinserted at an oblique angle to a deeper level to sever the deep attachments. The punch is then withdrawn and the sample is carefully retrieved from the punch. This technique supplies good samples from difficult areas and avoids damage to the biopsy sample. Post biopsy haemorrhage can be controlled with 1-2 horizontal mattress sutures of an absorbable material passed deeply through the tissue to draw the sides together. Alternatively a haemostatic sponge (such as Gelfoam or Surgicell) or omentum is lightly packed into the defect to control haemorrhage. Two or more biopsy samples from different areas of the liver are usually obtained.

Smaller liver biopsy samples can be obtained from deep mid-parenchymal regions using a Tru-cut needle.

Larger biopsy samples can be obtained from the tips of the narrow hepatic lobes. A loop of synthetic absorbable suture material is placed around the lobe slightly proximal to the tissue to be biopsied. A ligature is then created with this suture which crushes the tissue and occludes the veins and bile caniculi within the crushed area. The sample is then incised from

> The biopsy punch technique is most useful for getting good representative samples from the liver. This can be used with both discrete and diffuse disease.

the stump, at a sufficient distance to avoid slippage of the ligature, but avoiding leaving large amounts of devitalised tissue behind. Larger samples on lobar margins can be obtained using a similar technique with several interlocking mattress sutures. It is important to place all the sutures before tightening them to allow adequate crushing of the tissues and adequate interlocking of the sutures.

- **Pancreas**

Biopsy of the pancreas is a safe and predictably successful procedure, if it is performed with gentle manipulation and care, with almost no incidence of postoperative pancreatitis. In patients with diffuse pancreatic disease a small segment at the periphery of the right or left limb of the pancreas (usually the right as this is more accessible) is identified. An encircling

ligature of 1.5-2 metric (4/0 – 3/0 USP) absorbable monofilament suture is placed around the base of the segment. This is tightened thereby ligating the vessels and pancreatic ducts from this segment and cutting through the pancreatic parenchyma. The distal segment is then excised using a scalpel being careful not to cut the suture. Care must be taken not to ligate the caudal pancreatico-duodenal artery and vein at the distal edge of the right pancreas.

- **Lymph nodes**

Either complete excisional biopsy of enlarged nodes or a wedge incisional biopsy can be performed. With an incisional biopsy a horizontal mattress suture is pre-placed and then tied following biopsy to control haemorrhage. Extreme care is needed when taking lymph node biopsies due to their close proximity to vessels – this is particularly true when biopsying the mesenteric lymph nodes.

- **Gastrointestinal biopsies –**

Endoscopic mucosal biopsies of the gastrointestinal tract should be taken in preference to surgical biopsy, as endoscopic biopsy is a low morbidity procedure whereas surgical biopsy carries the risk of dehiscence and peritonitis

As these involve entering a contaminated environment, care must be taken to avoid spillage of enteric contents. This can be achieved by packing the areas to be biopsied off from the rest of the abdominal cavity with large moistened laparotomy swabs and by exteriorising the loops of intestine from the abdominal cavity. Milking the intestinal contents away from the area to be biopsied and occluding the bowel with assistants' fingers or bowel clamps is then required. I usually take these biopsies last due to their contaminated nature. This is then followed by discarding the dirty swabs, covering dirty drapes, changing gloves and using clean instruments to lavage, suction and close the abdominal cavity.

See next chapter on Principles and Practice of Gastrointestinal Surgery

- **Stomach**

  Carefully examine the stomach for any serosal abnormalities and then fully palpate it for any mural or mucosal abnormalities. These areas can then be biopsied using a full thickness technique. Routinely a full thickness biopsy is taken from mid-way between the lesser and greater curvatures of the ventral surface of the body or fundus of the stomach. Through this small gastrotomy incision multiple mucosal biopsies can be taken from suspicious areas using an alligator punch biopsy forceps. The gastrotomy incision is closed with full thickness simple interrupted or continuous suture pattern of 1.5-2metric (4/0-3/0 USP) absorbable monofilament. This is usually sufficient or this can then be inverted with a seromuscular layer of sutures.

- **Upper intestinal**

  Full thickness biopsies are routinely taken from the duodenum, jejunum and ileum. My preference for performing this is to first place a 1.5metric (4/0) full thickness monofilament absorbable suture either longitudinally or transversely on the anti-mesenteric surface of the intestine (in the area to the biopsied). This allows manipulation of the intestine and biopsy sample. A no.11 or 15 scalpel blade is then used to cut around the suture and take a small full-thickness sample. The suture avoids handling the sample with forceps which often damages it. The suture and biopsy sample ate then both placed in formalin. The resultant small enterotomy is closed with an interrupted or continuous full-thickness layer using 1.5-2metric (4/0-3/0 USP) absorbable monofilament.

- **Lower intestinal**

  It is usually not necessary to perform a routine biopsy of the colon or rectum for most routine investigations of GI disease. Although the reported dehiscence rate for colostomy is no higher than enterotomy, the consequences could be much more serious due to the high number of enteric bacteria present. If there is a suspicious lesion this can be biopsied in a similar manner to the upper intestinal tract.

Laparotomy Closure

To close a ventral midline laparotomy incision, sutures must engage the external sheath of the rectus abdominus muscle. Bites are taken 5-10mm from the edge of the incision with avoidance of the rectus muscle and peritoneum if possible. Suturing the peritoneum is unnecessary as the peritoneum heals by a process of mesothelialisation where the mesothelial cells in the peritoneal fluid attach to denuded areas and proliferate to cover the defect. Suturing the peritoneum has been shown to increase the rate of adhesion formation. Including large bites of muscle is unnecessary and contraindicated, as the muscle has little holding strength so the sutures tear through this tissue probably causing discomfort and loosening the suture. Over the cranial half of the ventral midline the internal sheath is closely bound to the external sheath, and as the external sheath is slightly weaker here, the internal sheath is included in the suture bite to improve the holding strength.

The key points for laparotomy closure are:

➤ Use a strong absorbable suture material of appropriate size for the patient (for example: 2metric (3/0 USP) polydioxanone (PDS) for a cat; 3metric (2/0 USP) PDS for small dog; 3.5metric (0 USP) PDS for medium to large dog; and 4metric (1 USP) PDS for a giant dog). Alternatively use a non-absorbable monofilament for the closure.

➤ A simple continuous pattern does not increase the risk of dehiscence provided it is performed correctly with appropriate suture material – place knots with 6-8 throws at each end (+1 for end of row). Alternatively use simple interrupted sutures but these leave more suture material in the wound and are more time consuming.

➤ Ensure bites engage 5-10 mm (depending on size of patient) of the external fascial sheath of the rectus abdominus muscle.

➤ Pass the needle slightly obliquely through the external fascia, to avoid eversion of the linea.

➤ Close the subcutaneous fascia/fat with a simple continuous layer of absorbable suture material with buried knots. In cats use the rapidly absorbable suture material such as poligecaprone 25 (Monocryl) or polyglytone 6211 (Caprosyn). In the male dog two layers are needed caudally to close the subcutaneous fat and preputial muscle.

➤ Close the skin routinely. Ensure good accurate apposition in the male around the prepuce.

Gastrointestinal biopsy is NOT a benign procedure and should be performed with care and clear consent from the owners. There is a reported 12% mortality rate following routine incisional biopsy in a series of 66 patients at Cambridge University (Shales et al, JSAP 2005).

Prior to laparotomy closure ensure that all surgical swabs and instruments have been removed from the abdominal cavity. A routine of always counting the swabs before surgery and again after closure, using swabs with a radio-opaque line in the abdomen and radiographing the abdomen if a swab is missing should be implemented.

Complications

Wound dehiscence or herniation

This should be a rare complication if a good surgical technique and correct suture material are utilised. Wound dehiscence can be acute or chronic.

☐ Acute incisional hernias are usually seen within the first few days of surgery or at suture removal. The clinical signs are of a wound swelling, skin discolouration, a seroma or a serosanguinous discharge. These swellings are often non-painful. If there has been a significant dehiscence of the linea alba then herniation of abdominal contents into the subcutaneous space will occur, with evisceration only prevented by the skin wound. If skin sutures are removed there is a considerable risk of sudden skin rupture and evisceration.

The identification of a wound swelling, seroma or sersanguinous discharge from a wound

There are many potential causes for incisional hernias, but for acute herniation by far the most common cause is poor surgical technique, such as failure to engage the external fascial sheath or an inadequately tensioned continuous suture line, rather than suture material failure

must therefore be viewed with strong suspicion for an incisional hernia. These wounds are best surgically explored through the original skin wound along their entire length and the linea alba reclosed. Evisceration needs rapid management to stabilise the patient, prevent further damage to the exposed visceral contents, remove devitalised or necrotic intestine, control of abdominal cavity contamination and close the abdominal wound (either immediately or delayed depending on the extent of contamination/infection).

☐ For chronic incisional hernias, the skin has completely healed but the linea alba dehisces leading to herniation. This is often secondary to a systemic disease causing poor wound healing such as Cushings disease.

Wound infection
This should be an uncommon occurrence and can be minimised if care is taken with surgical technique, control of dead space, prevention of self trauma and anticipation/management of concurrent problems (such as Cushings disease or pyoderma).

Adhesion formation

Temporary adhesions result from inflammation and fibrin formation between adjacent serosal surfaces. If these fibrin adhesions develop into fibrous tissue adhesions, then permanent adhesions result. Permanent adhesions will result in the presence of infection, foreign material (such as sutures) and ischaemia. These is general do not cause any problems as they do not constrict or kink vital structures (non-restrictive adhesions). If however this occurs the restrictive adhesions can cause considerable clinical problems as they either constrict a hollow viscus, duct or blood vessel. In the dog and cat there is an active fibrinolytic system so that the development of permanent adhesions is less likely than in other species such as humans or horses. Care must therefore be taken during abdominal position to anticipate or prevent possible adhesions. This is particularly true when using the omentum to reinforce sutured repairs or pack defects/abscesses.

Peritonitis

Non-septic peritonitis can be a consequence of excessive/rough handling causing serosal inflammation, exposure to air and some chemicals such as bile, urine and antiseptics. All abdominal surgery will therefore cause a very mild non-septic peritonitis which should not require intervention. Exceptions to this are with continued inflammation as with a biliary tree or urinary tract rupture.

Septic peritonitis is usually bacterial and is distinguished from non-septic by large numbers of degenerate neutrophils with intracellular bacteria. This is a clear indication for immediate stabilisation and exploratory laparotomy as soon as possible. Following a previous surgery this is generally due to surgeon error, surgical failure, or increased permeability of bowel wall due to devitalisation or distension. The mortality rate for septic peritonitis is high even with early identification and appropriate intervention (see later chapter).

Unretrieved foreign material within the abdomen

As previously mentioned it is essential to account for all swabs, needles and instruments at the end of surgery. These can cause a foreign body reaction which may be very challenging to manage subsequently and may become the subject of legal proceedings against the surgeon.

<u>**General Principles**</u>

Gastrointestinal wound healing

In spite of an apparent hostile environment of digestive secretions, high numbers of bacteria and the effects of the peristaltic contractions; sutured intestinal wounds should routinely rapidly heal when a good surgical technique is employed.

- **Anatomy**

The wall of the gastrointestinal tract is composed of the mucosa, submucosa, muscularis and serosa. Of these layers the key one to recognise and engage on every suture bite is the submucosa, as this is the collagen-rich layer which provides the holding strength of the suture. In comparison the other layers of the bowel wall have negligible strength. The submucosa is recognised as a thin white layer just beneath the bulging mucosa.

- Healing of mucosal injury:

The mucosal surfaces of the gastrointestinal tract are constantly exposed to the constant physical and chemical trauma of normal digestion. These cells must be constantly replaced

> **It is essential to recognise and engage the submucosa in surgical closure of the bowel**

by the process of epithelial cell regeneration, therefore when a severe injury to the epithelial surface occurs (i.e. ulceration), the repair of this defect is just an extension of this process. The process of epithelial cell regeneration is the same as in other tissues, with an initial migration of adjacent cells across the defect followed by increased division of the basal cells behind these.

- **Healing of full-thickness surgical incision**

Successful surgery of the gastrointestinal tract relies on the rapid normal healing mechanisms of these tissues. For a correctly sutured wound the initial short haemorrhage/coagulation/initiation phase is followed by the inflammatory/ debridement, reparative and maturation phases, with the difference to other tissues (such as the skin), that uneventful healing returns the strength of the repair site to the same as normal intestine by 10 – 17 days post surgery.

The initial haemorrhage/coagulation phase of healing forms a fibrin seal over the surgical wound in the first few hours. This supplies a small amount of wound strength but more importantly it prevents migration of bacteria from within the intestinal lumen. The presence of a serosal surface greatly aids this formation of an initial seal as mesothelial cells migrate over the defect. The lack of a serosal surface as in the oesophagus, increases the risk of early wound dehiscence.

Over the next 4-5 days the inflammation/debridement phase occurs. The key cell is the macrophage which debrides the wound and produces growth factors to modulate the reparative phase of wound healing. Epithelial migration across the woundbegins during this time. By the end of this phase the wound is at its weakest due to proteolytic enyzmes that have been produced that weaken the collagen and fibrin at the wound edges and the lack of sufficient collagen deposition. The wound is entirely dependent on the sutures and fibrin. It is at this stage that sutures that are placed too close to the wound edge can tear through the weakened tissue, leading to wound dehiscence. Similarly if the inflammatory/ debridement stage is prolonged or accentuated by inflammatory/infiltrative bowel disease, reduced vascularity, gross contamination, poor tissue handling, inappropriate suture material (such as catgut), inappropriate closure (such as inversion) or concurrent peritonitis, then the risk of dehiscence increases.

The reparative stage of intestinal wound healing occurs between days 3-14 (note how the inflammatory/debridement and reparative phase overlap). Fibrous repair occurs with a rapid gain in tensile strength. The maturation phase, characterised by collagen reorganisation and remodelling occurs from about 10 days.

The excellent blood supply of the gastrointestinal tract allows rapid normal wound healing to occur. All stages of healing may be prolonged, thereby increasing the risk of dehiscence, if this is compromised due to local factors (ischaemic necrosis from foreign body, poor surgical technique etc) or systemic factors (hypovolemia, shock etc). Adequate blood flow and oxygenation are critical for successful colonic healing to occur. Oxygen is essential for the formation of collagen and therefore low oxygen tension predisposes to wound dehiscence.
Increased tension across intestinal anastomosis reduces blood flow and stresses potentially weakened tissues at the wound edge. This may be due to poor surgical mobilisation or accumulation of ingesta, fluid or gas, and increases the risk of wound dehiscence, therefore tension on the closure should be anticipated and minimised.

Basic Tenets of Gastrointestinal Surgery
1. Recognise and incorporate the submucosa into the surgical closure 2. Minimise the inflammatory/debridement phase 3. Maintain a good blood supply 4. Prevent tension across the suture line

Surgical preparation

As surgery involves entering a contaminated environment, care must be taken to avoid spillage of enteric contents. This can be achieved by packing the areas to be biopsied off from the rest of the abdominal cavity with large moistened laparotomy swabs and by exteriorising the loops of intestine from the abdominal cavity. Milking the intestinal contents away from the area to be incised and occluding the bowel with assistants' fingers or bowel clamps (Doyen) is then required.

Intestinal incisions are made with a sharp (no.11) scalpel blade to minimise damage to the wound edges. Incisions should always be made through the healthiest looking tissue.

Closure patterns

Many of the traditional closure patterns for bowel were developed when the suture materials at the disposal of the surgeon (such as silk and catgut) were considerably inferior to the modern synthetic suture materials and there were serious concerns of early wound dehiscence. For this reason the two layer closure techniques, inversion, eversion and invagination techniques were developed but these are now outdated and have been shown to be significantly inferior to appositional techniques.

Appositional closure provides the most rapid healing of all tissue layers in all regions of the intestinal tract. This is because primary wound healing should occur, thereby minimising the inflammatory/debridement phase and ultimately limiting the amount of mural scar formation. Simple interrupted appositional single layer intestinal sutures can be utilised and are ideal in most instances, although simple continuous closure can be utilised in certain situations.

Appositional sutures are technically easy to place, cause little interference to blood supply if placed properly, cause minimal adhesion formation and maintain the size of the lumen. Appositional closure provides the best bursting strength to the repaired intestine.

Single layer closure patterns are ideal for the intestine. It is critical that all sutures engage the submucosal layer for adequate strength. If a two layer closure pattern is utilised (as occasionally in the stomach), the first layer incorporates the mucosa and submucosa, whilst the second layer closes the seromuscular layer, but takes small bites of the submucosa on each bite.

Suture material

Synthetic absorbable monofilament suture material on a swaged taperpoint needle is the best suture (for example polydioxanone (PDS) or glycomer 631 (Biosyn)). Size 1.5metric or 2 metric (4/0 – 3/0 USP) is applicable for nearly all situations, with my preference for 1.5 metric (4/0 USP) for most dog and cat intestine. Absorbable multifilament can be utilised but has the potential disadvantages of causing more tissue drag/damage and potential wicking of bacteria through the suture. Nonabsorbable suture is associated with ulcer formation and is generally not used. If delayed wound healing is anticipated (as with hypoalbuminemia of total protein <15g/l) my preference is to use a longer acting absorbable such as polydioxanone.

The use of catgut is contraindicated due to the inflammatory reaction elicited.

Suturing technique

When suturing intestine the everted mucosal edges are initially trimmed if necessary to aid appositional closure. I take bites 3-5mm from the wound edge and angle the bite so that a smaller amount of mucosa is included compared to the other layers. This helps to prevent eversion of the mucosa. Care is taken with monofilament suture material that secure knots are formed without overtightening the suture and strangulating the included tissue. On the other hand sutures that are too loose increase the risk of leakage of intestinal contents. Knots are placed extraluminally and the sutures are placed about 3mm apart.

Following completion of the closure (interrupted or continuous closure), the tips of a curved mosquito haemostat are used to probe the wounds for any obvious gaps. If there are any then additional interrupted sutures are placed. For intestinal resection and anastomosis, the most common site for dehiscence is on the mesenteric border. This site needs to be carefully probed with special care that secure closure has been achieved. New gloves and a new set of surgical instruments are required to close the abdomen.

Resection and anastomosis

Vascular compromise to the intestines occurs in many conditions and the decision whether to resect portions of the bowel depends on accurate assessment of intestinal viability (see later section in this chapter). If viability is questionable resection is the prudent choice. For intestinal foreign bodies unless perforation has occurred, I remove the foreign body via an enterotomy initially, followed by assessment of viability and then resection and anastomosis if indicated.

Vessels to the section of bowel to be removed are ligated. Milk the intestinal contents towards the centre of the segment of intestine to be resected and place non-crushing (Doyen) forceps

> The most important surgeon-controlled reason for intestinal wound dehiscence is poor suturing technique and not suture material failure.

just before and past the levels of the proposed resection incisions. Alternatively use an assistants gloved fingers to occlude the bowel (my preference if I can get an assistant!). Anticipate luminal diameters of the intended anastomosis. If there will be slight disparity then plan an oblique incision (with less intestine on the antimesenteric compared to the mesenteric side). For more significant disparity an antimesenteric incision on the smaller side increases the surface area of the anastomosis site. Place crushing (Carmault) forceps inside each of the previously placed non-crushing forceps with potential luminal disparity anticipated. This is to prevent leakage from the resected intestine once the incisions are made. Transect the intestine between the forceps (use the crushing forceps as a cutting guide) and remove. The non-crushing forceps (or assistant's fingers) prevent leakage of the retained section, without damaging the blood supply to the tissue.

Perform an anastomosis of the segments of intestine. An appositional single layer full thickness suture pattern is indicated (see earlier). Place a simple interrupted suture at the mesenteric side and another on the opposite anti-mesenteric side. This splits the anastomosis into two sides and helps proper spacing of the sutures. A simple interrupted pattern is traditionally reported, but a simple continuous closure (one each side – two in total) is also reported with no increase risk of dehiscence. The anastomosis should be checked by keeping the intestine on each side occluded and inflating the segment with sterile saline by injection. Under moderate pressure, this helps to identify leaks. Probe the anastomosis site for gaps, paying particular attention to the mesenteric side of the anastomosis. New gloves and a new set of surgical instruments are required to close the abdomen.

Suture line reinforcement

- **Omentum**

The omentum is a specialised development of the peritoneal surface. The omentum helps isolate and seal the source of internal or external contamination by formation of omental adhesions at the site. The omentum also absorps bacteria and particulate matter. The omentum brings a rich blood supply, high absorptive capacity and pronounced angiogenic activity. The omentum enhances healing of ischaemic small intestinal anatomoses and rectal anastomses and increases the bursting strength in experimental colonic anastomsoes.

It is these 'policeman' roles within the abdomen that are utilised when the omentumn is deliberately used to protect an incision into a hollow viscus. If there is minimal contamination or inflammation at the site of injury, it is likely that the fibrinous adhesions of the omentum are broken down by fibrinolysis.

The omentum is also used in other areas such as protection of gastropexy and enterostomy sites; hemostasis for liver or splenic injury (and liver biopsy sites); and packing abscess cavities such as in the prostate or spleen.

- **Serosal patch**

Another option to reinforce intestinal repairs is the use of a serosal patch. This utilises the rapid adhesions that form from the serosal surface of a loop of jejunum to areas of contamination. A loop of jejunum is mobilised to the intestinal site without too much tension or kinking it. The anti-mesenteric surface is sutured along the incision on each side using sutures of monofilament absorbable material. These sutures include the submucosa of both segments of the intestine. For larger defects or anastomosis sites, multiple segments of jejunum can be utilised.

This technique appears to be more effective than the use of omentum alone. Although rarely required, in certain situations such as enterotomy of ischaemic descending duodenum (where resection carries a high morbidity due to the pancreas and common bile duct) it is a 'lifesaver' as it will prevent very serious complications.

Antibiotics

Perioperative broad-spectrum antibiotics are indicated for the patient if the gastrointestinal or urogenital tract are to be entered or if a long surgery is anticipated, particularly in a critically–ill patient. Prophylactic use of a broad-spectrum antibiotic administered intravenously at induction of anaesthesia (30 minutes before surgery commences) and repeated at 90 minute intervals during surgery, ensures therapeutic levels of antibiotics at the time of contamination. Only if there is a specific indication to do so (unexpected leakage of intestinal contents or a

break in normal asepsis in any way) is a therapeutic course of antibiotics given in the postoperative period. The inappropriate use of antibiotics leads to selection of resistant bacteria, wipes out non-pathogenic enteral bacteria and has been shown to increase the risk of wound infection.

For upper intestinal surgery/gastric/liver/pancreatic surgery a first generation cephalosporin, or clavulanate-potentiated amoxicillin is given intravenously at induction. For lower intestinal surgery a second-generation cephalosporin at induction, or, metronidazole and cephazolin (in combination) is commenced 24 hours preoperatively.

Potential Pitfalls

Systemic factors and patient stabilisation

Patients requiring gastrointestinal surgery are generally unwell to some extent. Identification of the underlying cause of the gastrointestinal disease or the presence of concurrent problems is required to determine problems that might be encountered. It is important to identify any fluid, electrolyte or acid-base imbalances prior to general anaesthesia and surgery. Severe derangements should be partially corrected prior to surgery, although the overall plan is to complete correction of these problems is the postoperative period, when the underlying cause has (hopefully) been corrected. For the majority of problem provision of 1-3 hours of preoperative intravenous fluids will better prepare the patient for the anaesthetic and surgery.

Exceptions to this where surgery should be performed as soon as possible are penetrating abdominal wounds, intestinal perforation or volvulus.

Severe hypoproteinemia or hypoalbuminemia (albmumin <15g/l) will have a significant impact on intestinal wound healing. In these cases albumin levels need to be improved by provision of a plasma transfusion. This may need to be continued in the postoperative period. Nutrient requirements need to be anticipated in the postoperative period and these cases benefit from the prophylactic placement of a feeding tube. My preference is placement of an oesophagostomy tube due to its ease of placement and straightforward management.

Assessment of intestinal viability

Accurate assessment of the viability of a segment of intestine can be very difficult. Objective diagnostic tests have been developed and examples include fluorescent dye, surface oximetry and Doppler ultrasonic flow probes. The sensitivity of the tests can be variable however and they are technically cumbersome to perform, with no particular place in clinical practice.

Clinical subjective criteria for viability include colour, the presence of (mucosal) haemorrhage from the cut surface, the presence of peristaltic contractions and the presence of arterial pulsations.

In practice for areas of questionable viability it is better to resect than leave behind and risk

> Criteria for viability include colour, the presence of (mucosal) haemorrhage from the cut surface, the presence of peristaltic contractions and the presence of arterial pulsations BUT if in significant doubt, cut it out (except for the proximal duodenum!)

major complications. This is especially true as resection and anastomosis is a relatively straightforward procedure and there is a major reserve capacity to the intestine (see next section on short bowel syndrome). Situations where resection is likely to lead to significant morbidity (such as the descending duodenum) or is not possible (where there are multiple areas of rupture as with linear foreign bodies), require very careful assessment and the use of suture line reinforcement (see earlier) - generally serosal patching is indicated in these cases.

Short bowel syndrome
The question of how much intestine can be safely removed only really arises when extensive areas of the intestine are affected (such as with neoplasia, torsion or linear foreign body). Short bowel syndrome refers to the clinical signs that occur when extensive resection means that there is insufficient intestine for digestion and absorption. The clinical signs of this are diarrhoea, weight loss and malnutrition. Adaptation of the intestine occurs with some improvement of clinical signs but this may take 1-2 months and the initial nursing required can be intensive (and expensive). Highly digestible diets need to be supplied with careful monitoring of body condition and additional vitamin/mineral supplements.

This surgically created disease will depend on the site and extent of resection. One study found that in dogs with jejunal resections of 50-90%, the rate of short bowel syndrome was

> At least 75% of the jejunum needs to be resected before signs of short bowel syndrome will develop. Resection of the ileum and ileocaecal valve increases postoperative morbidity. The proximal duodenum must be preserved.

low and the length resected was not predictive for clinical signs. At least 75% of the jejunum needs to be resected before signs will develop. Loss of the ileum is less well tolerated and in dogs preservation of the ileo-caecal valve is important, as it prevents bacterial overgrowth of the small intestine. The proximal duodenum must be preserved.

Postoperative feeding

One long standing recommendation in many surgical textbooks is a slow return to oral feeding over a number of days. But there is no evidence to support this approach and it actually runs counter the known importance of enteral nutrition to the maintenance of a healthy gut (the amino acid glutamine is a key fuel source for intestinal epithelium) and wound healing. The idea that by not feeding the risk of intestinal leakage is decreased runs counter to common sense, as there will still be intestinal contents regardless of feeding. The avoidance of malnutrition is essential in optimising wound healing (see later chapter on enteral nutrition) and the resumption of normal intestinal motility is encouraged by feeding.

There is accumulating evidence in the human surgical literature that early enteral nutrition after all abdominal surgeries prevents weight loss, improves wound healing, decreases infection rates and speeds time to discharge.

At Davies Veterinary Specialists, our usual practice is to offer a highly digestible food to the intestinal surgery patient as soon as it has recovered from the general anaesthetic. A little and often approach is taken with the amounts consumed closely monitored. Obviously this is altered on a case by case basis and will depend somewhat on the severity and extent of the underlying or concurrent disease process.

Postoperative ileus

Ileus or bowel stasis can be common but is preventable in the majority of cases. Ileus results from an overactive sympathaetic nervous system that reduces intestinal motility. This is activated by the laparotomy, rough handling of the intestines, long operative time, pain, stress and starvation postop. Clinical signs include pain, vomiting, regurgitation and abdominal distension. The key problem with these signs is that they mirror those of intestinal dehiscence and peritonitis, making differentiation and diagnosis difficult. Ileus can also occur as a consequence of peritonitis so investigation to rule this out is important, particularly in a case that develops ileus 3-5 days postoperatively when it had been progressing well previously.

Treatment is aimed at reducing stress, providing adequate analgesia, initiating appropriate enteral nutrition, and dealing with underlying disorders such as electrolyte abnormalities and sepsis. Drugs to improve motility such as metoclopramide or ranitidine are also implemented until motility improves.

Adhesions

Compared to humans, the development of excessive adhesions in small animals causing postoperative complications (such as intestinal obstruction) is very uncommon. Factors involved include extensive foreign body contamination, ischaemia, haemorrhage or infection.

This is due to a highly effective fibrinolytic system within the abdominal cavity of dogs and cats that breaks down fibrinous adhesions.

Should extensive adhesions causing clinical problems such as obstruction occurs then treatment surgical reduction of the adhesions and released of the entrapped segment of intestine. If viability is questionable then resection and anastomosis is indicated.

Postoperative wound dehiscence and development of peritonitis

Careful monitoring in the postoperative period is necessary to quickly detect the signs of intestinal wound dehiscence and peritonitis (see later chapter). Remember that dehiscence is most likely 3-5 days after surgery but has been reported up to 10 days postop. I routinely keep my patients hospitalised for 3-5 days during which time the rectal temperature is monitored three to four times daily, and sudden changes in appetite, demeanour, defecation frequency, abdominal discomfort/distension or the development of vomiting are assessed for. Any changes warrant further assessment such as haematology, abdominal radiographs or ultrasonography, but the most valuable tool is (ultrasound-guided) abdominocentesis to check for evidence of degenerate neutrophils with intracellular bacteria or gross foreign material. This warrants immediate exploratory laparotomy and peritonitis management (see later chapter).

Once the patient is sent home I warn the owners of the signs of potential peritonitis (emphasis on sudden deterioration in clinical status) and stress the importance of immediate reassessment.

Surgical Disease of the Stomach
- FB and GDV!

Surgical anatomy

The stomach lies in a transverse position, more to the left than the right. In the cat, only the pylorus is located on the right hand side.

- The stomach is divided into five anatomic regions, the cardia, fundus, body, pyloric antrum and pylorus.
  - ➤ Cardia – blends with the oesophagus. The size of the cardiac region varies with species and glands in the cardiac region secrete mucus.
  - ➤ Fundus – a blind pouch located on the left of the cardia. The fundus acts as a storage area for food and is the major force behind the expulsion of fluids. Most of the enlargement that occurs after a meal occurs in this region.
  - ➤ Body – involved in mixing the food with gastric secretions and saliva. Glands in this area contain chief cells, which secrete pepsin, and parietal cells, which secrete hydrochloric acid. The transfer of each chloride ion into the parietal cell is exchanged for a bicarbonate ion, which is secreted into the blood - the 'alkaline tide'.
  - ➤ Antrum and pylorus – act to agitate food and filter chyme into the duodenum. The pylorus also contains the gastrin cells which, on appropriate stimulation, release gastrin into the blood.

- Layers of the stomach
  The stomach has four distinct layers the serosa, muscular layer, submucosa and the mucosa.
  - ➤ The serosa is very thin and elastic. It consists of a single layer of mesothelial cells that are firmly attached to the underlying muscle.
  - ➤ The muscle layer is well developed and is composed of three layers. The outer longitudinal layer passes around the curvatures of the stomach and is continuous with the longitudinal layers of the oesophagus and duodenum. The inner, circular layer is present only over the pylorus and body and contributes to the pyloric sphincter. The oblique layer lies immediately adjacent to the submucosa of the body and fundus only.
  - ➤ The submucosa is a tough elastic layer composed of connective tissue, blood vessels and nerves. In the non distended organ, it forms the gastric rugae. This is the layer with the most significant suture holding capabilities.

> ➢ The mucosal layer consists of the epithelial layer, the lamina propria and the muscularis mucosa (smooth muscle layer). The mucosa is glandular and has a surface mucus barrier to protect it from the products of digestion.

- Vascular supply

  The arterial supply originates from the celiac artery. The main arteries to the stomach are the left and right gastric arteries, which run along the lesser curvature, and the left and right gastroepiploic arteries, which run along the greater curvature. In addition to these arteries, several long branches of the splenic artery supply a portion of the fundus (the short gastric arteries).

  There is considerable collateral supply and overlapping between the regions of the stomach. For example both the short gastric and left gastroepiploic arteries can be ligated at splenectomy without developing areas of gastric ischaemia or necrosis.

- Nerve Supply

  The stomach is innervated by parasympathetic fibres from the vagal nerves and by sympathetic nerves from the coeliac plexus. Both nerves contribute to the myenteric polexus located between the longitudinal and circular layers of the muscular layer and to the submuosal plexus. Intrinsic activity (smooth muscular activity and glandular secretions) is maintained via a variety of neuroendocrine pathways.

Gastric foreign bodies

The most common reason for performing a gastrotomy is to remove gastric foreign bodies.

- Gastrotomy

The stomach is approached via a cranial midline laparotomy. The stomach cannot be completely exteriorised and there is a risk of leakage of gastric contents to the rest of the abdominal cavity. To isolate it from rest of peritoneal cavity, moistened laparotomy swabs are packed around it to control contamination. Stay sutures are used to manipulate the stomach and to elevate it from the abdominal cavity prior to gastrotomy

With any stomach surgery, use stay sutures and laparotomy swabs to control its position and to greatly aid manipulation.

Make the gastrotomy incision midway between greater and lesser curvatures on ventral surface of stomach. Make a stab incision with a no.11/15 blade and extend with scissors. Place further stay sutures at the wound edges for greater control.

With foreign bodies, always suction the remaining gastric fluid and check for additional foreign bodies before closure.

Closure is with monofilament absorbable suture material (PDS/Biosyn/Monocryl) on a taperpoint needle. Chromic catgut is contraindicated as it rapidly loses tensile strength in contact with gastric fluid. I usually use size 1.5 (cat) - 2 metric (4/0 – 3/0 USP), with 3 metric (2/0 USP) in very large breed dogs.
The closure patterns I prefer are:

1. Two simple continuous appositional layers
    - Ⓡ Mucosa + submucosa
    - Ⓡ Muscularis and serosa taking deeper bites of the submucosa

2. Alternatively
    ⇒ A full thickness simple continuous appositional suture layer. Minimise the amount of mucosa engaged in each bite but ensure the submucosa is engaged.
    ⇒ This is generally sufficient, but if there is a concern then this suture line can be inverted with a seromuscular continuous layer (cushing pattern)

Gastric dilation volvulus

Multiple predisposing factors for the development of GDV have been identified, including deep chested conformation, laxity of the hepatoduodenal and hepatogastric ligaments, diet (cereal diets?), post-prandial exercise and once daily feeding.

It is thought that GDV occurs when gas or fluid accumulates in the stomach and the normal means of removing this (vomiting, eructation, pyloric function) have become inoperative. Aerophagia is the main source of the air (bacterial fermentation and acid/bicarbonate reactions are other possible sources). The cause of the aerophagia is not known, but may be associated with rapid eating or excitement and affected dogs may have abnormal oesophageal motility and dysphagia. It is unknown if the gastric dilatation precedes volvulus (with twisting occurring due to the enlarged stomach) or if the dilatation occurs as a consequence of the volvulus (due to lack of outflow from the stomach into the oesophagus or duodenum).

The most common rotation is clockwise (viewed with the dog in dorsal recumbency) and the maximum amount of rotation is 270 – 360degrees. Anticlockwise rotation is rare and the maximum amount of rotation possible is 90 degrees. Clockwise rotation occurs when the fundus passes under the pylorus to position along the right ventrolateral abdominal wall. The pylorus is located on the left abdominal wall, adjacent to the oesophagus. The surgical view is a dilated body ventrally, covered by the ventral leaf of the greater omentum. Anticlockwise rotation occurs when the pylorus and antrum displace dorsally along the right abdominal wall. The distal oesophagus is incompletely occluded and the duodenum is kinked. The fundus and body are not displaced.

GDV causes hypovolaemia, endotoxaemia, hypoxia, cardiac dysfunction and gastric ischaemia.

Hypovolaemia occurs due to compression of the caudal vena cava and portal vein by the dilated stomach. Compensatory flow through the vertebral sinuses is inadequate and pooling of the blood in splanchnic, renal and capillary beds of the hindquarters occurs. Reduced venous return to the heart results in a reduction in cardiac output and triggering of protective mechanisms aimed at preserving the blood flow to the heart and brain. Persistent hypovolaemia will generally lead to metabolic acidosis and cellular hypoxia, however in GDV secretion and sequestration of H^+ into the stomach lumen may counteract the metabolic (lactic) acidosis and counteract the acid/base balance disturbances. Persistent hypovolaemia will lead to cell death and activation of inflammatory pathways leading to DIC.

Endotoxaemia may occur due to ischeamic injury to the intestinal mucosa. Clearance of the endotoxin is retarded due to the venous congestion of the liver.
Progressive dyspnoea due to impingement of the thoracic and diaphragmatic movements due to the physical obstruction of the dilated stomach and the effects of circulating endotoxins causing bronchoconstriction, V/Q mismatch and eventually pulmonary oedema will lead to hypoxia.

Reduced venous return will reduce cardiac output and the increased sympathetic stimulation due to the above mechanisms will lead to a shortened cardiac cycle with reduced diastolic pressures which may lead to myocardial hypoxia and ischaemia. This will further reduce cardiac contractility and dysrhythmias may be potentiated by acidosis, catecholamines, hypokalaemia etc.

Increased intraluminal pressures and venous stasis result in ischaemic damage to the gastric mucosa and submucosa. Continued gastric ischaemia through all the layers of the stomach may lead to gastric rupture.

Treatment

Fluid therapy – shock doses of lactated ringers solution (90ml/kg/hour) are indicated. I usually start this rate of fluids and then reassess the dog's parameters and requirement for continuing at this level after 15 minutes. If still necessary I will continue this fluid level and reassess again after a further 15 minutes. Catheters in both cephalic veins will probably be needed to allow this to occur. Hypertonic saline at 3-5mls/kg/hr may be administered over a 20 minute period. Isotonic fluids at surgical rates (10ml/kg/hour) should always be given after administering hypertonic saline. Colloids such as hetastarch may be administered (5-10ml/kg/hr over 10-15 minutes).

Antibiotics - the use of broad spectrum antibiotics effective against enteric organisms are indicated.

Oxygen – provision of oxygen can improve the dyspnoic dog or the dog in hypovolemic shock.

> The initial priorities are fluid therapy and decompression of the stomach. In practice this means place intravenous catheters, start shock doses of fluids and then attempt decompression of the stomach.

Decompression – this should be carried out after fluid therapy has been established. There are three options: orogastric tube, percutaneous trocar and gastrotomy

- Orogastric intubation depends on the animal compliance and the degree of volvulus. Having a selection of tubes is important. The tube is measured and marked (with tape) at the point where stomach penetration would be expected (xiphoid) to avoid inadvertent penetration of the stomach by over-insertion of the tube. A roll of tape is placed in the mouth between the incisors and the tube is passed through the centre of the hole. I start with the smallest sized tube with liberal lubrication of the end as it passes through the oesophagus. Gentle blowing into the tube may help passage of the tube through the cardia. After successful intubation and decompression, the stomach contents are removed and to do this I will now place a much larger bore tube. Emptying can be difficult as the gastric contents often block the tube and initial gastric lavage is performed to help loosen and liquidify the food contents. Evidence of blood in the gastric contents is an

indication for prompt surgery as it is likely that there is gastric necrosis developing. If complete gastric emptying is not possible then it is best to proceed to surgery and empty the stomach after repositioning the stomach and re-intubation of the stomach via the oesophagus. Gastric lavage is again performed to help remove the contents. In certain situations it is necessary to perform a gastrotomy to remove very solid material within the stomach that cannot be broken down, but this obviously significantly increases the risk of peritoneal contamination. In this case the edges of the gastrotomy are controlled with stay sutures and laparotomy sponges are used to reduce contamination. Although decompression of the stomach prior to surgery can be maintained by placement of a pharyngostomy or gastrostomy tube, this would only be indicated if the patient needed to travel for definitive surgery.

- If the tube passage is unsuccessful, percutaneous gastrocentesis using an 18 gauge needle is helpful in relieving some of the gas in the stomach. This is placed through either the right or left flank at a site where a tympanic tone can be elicited on percussion. Use of a larger trocar should be avoided due to the increased risk of abdominal contamination. Following percutaneous decompression, a repeat attempt at orogastric intubation should be performed.

- Flank gastrotomy is performed in collapsed patients in which orogastric intubation or percutaneous gastrocentesis is not possible and where the patient is too unstable for general anaesthesia and standard midline laparotomy/gastrotomy. A skin incision is made in the right paracostal abdominal wall (after infiltration of local anaesthetic) and the incision is extended through the muscular layers until the stomach wall balloons into the incision. The serosal wall is then sutured to the skin using a simple continuous suture line and a stab incision is made into the stomach lumen.

Surgical treatment

There are three aims of surgery:

1. Correct the gastric malposition

2. Assess and treat ischaemic injury

3. Prevent recurrence

1. Correction of the malposition may require the placement of an orogastric tube to enable continued lavage and decompression of the stomach to facilitate repositioning. Avoiding

performing a gastrotomy to empty the stomach prior to repositioning as this will result in increased morbidity (see above for emptying stomach contents)

2. After gastric repositioning an assessment of ischaemic injury should be performed. Stomach: Gastric necrosis on the greater curvature may be treated by partial gastrectomy or by partial gastric invagination. Gastric invagination utilises a single layer inverting suture pattern that invaginates the affected mucosa into the gastric lumen. The opposed serosal surfaces will heal and the invaginated mucosa will slough off if necrotic. This

> If the patient's condition improves after decompression DO NOT discontinue treatment. Simple dilatation without volvulus is uncommon and it is best to assume that every case has volvulus until the opposite has been proven. Next empty the stomach and then use radiography to check the stomach position. If the stomach cannot be emptied or radiography confirms volvulus proceed to surgery.

however is a likely cause of postoperative ileus, nausea and vomiting as this process occurs. Partial gastrectomy is preferred as long as peritoneal contamination is avoided. This can be achieved with the use of surgical staplers or by careful use of stay sutures, laparotomy swabs, suction and large bowel clamps if sutured closed. Ischaemic lesions on the lesser curvature of the stomach are not amenable to either resection or invagination and carry a poor prognosis. If gastric rupture has occurred, post operative management by open peritoneal drainage is indicated and the prognosis is very poor.
The mortality rates in dogs with gastric necrosis is significantly higher than in patients with a viable stomach (46% versus 18% respectively)

Spleen: after repositioning of the stomach, the spleen should be identified and assessed. Torsion may have occurred, however because of the venous stasis, splenic size is likely to be considerable and this alone does not warrant removal. If splenic necrosis is present, don't untwist the spleen, but remove it without untwisting (see splenic section).

3. Gastric dilation is a recurring disease and attempts must be made to avoid recurrence of the GDV. There are a variety of techniques available to create adhesions between the pylorus and the abdominal wall.

Options for pexy of the stomach include tube gastropexy, incisional and belt loop gastropexy.

Tube gastropexy

Tube gastopexy is quick and simple and allows for continued post operative decompression and permits provision of nutrition for critical patients, however adhesion formation is weak and there is increased risk of abdominal contamination and a disruption to gastric motility. This is potentially the procedure of choice for GDV patients who are septic or collapsed prior to surgery, where rapid anaesthesia, surgery and recovery are required. A longer postoperative stay is also required.

A purse string suture is pre-placed into the seromuscular layer of the stomach at the pyloric antrum. An 18 Fr foley catheter is placed through a stab incision in the abdominal wall and into the pyloric antrum. The foley catheter is inflated with sterile saline and the purse string tied to secure the neck of the catheter securely. The serosa and the peritoneal surface are scarified using a dry swab and sutures of permanent suture material are placed between the stomach wall and the abdominal wall to secure the stomach. The tube is removed after 7 to 10days and the stoma left to heal by granulation.

Incisional gastropexy

An incision is made in the seromuscular layer of the ventral pyloric antrum along the long-axis of the antrum. A corresponding incision is made through the peritoneum and tranversus abdominus/rectus abdominus muscles to position the pylorus in a relatively anatomic position caudal to the last rib on the right ventrolateral abdominal wall. The cranial edges of each incision are then sutured with a simple continuous layer of nonabsorbable monofilament suture material such as polypropylene or a long acting synthetic absorbable such as polydioxanone. The caudal incisions are then sutured. This technique is technically simple, has no reported complications and results in a strong adhesion formation.

Belt-loop gastropexy

A belt-loop technique can also be used to pexy the stomach. This results in superior adhesion formation and pexy strength and does not enter the gastric lumen, however is technically more difficult (resulting in increased operative time).

A U shaped incision is made through the seromuscular layer of the ventral aspect of the pyloric antrum. The incisions are undermined to allow the creation of a flap with at least two vessels from either the lesser or greater curvature included within the base of the flap. Parallel incisions are made in the right ventrolateral peritoneal surface of the abdominal wall, extending deep to beneath the transversus abdominus muscle. These incisions are made roughly parallel to the last rib and are roughly 1.5 times the width of the flap. The muscle between the incisions is undermined to create a tunnel. The elevated seromuscular flap is

passed through the tunnel and sutured back to its original gastric margin using nonabsorbable suture material.

Post-Operative complications

The effects of surgery and anaesthesia may worsen the cardiovascular status of the patient and continued fluid therapy and monitoring of haemodynamic parameters is recommended. Cardiac arrhythmias occur in 40% of dogs suffering from GDV within 36 hours of presentation. Arrhythmias are usually of ventricular origin and are associated with myocardial ischaemia, effects of acidosis and electrolyte disturbances and circulating myocardial depressant factors and reperfusion agents. Treatment is indicated if the arrhythmia occurs at a frequency of greater than 15 per minute or signs of altered haemodynamic function are present. Correction of underlying electrolyte and acid base abnormalities are also indicted. Oxygen supplementation and provision of analgesia is essential.

Significant blood loss can occur in GDV. If blood loss or anaemia is severe, transfusion may be indicated.

Potassium loss occurs through gastric hypersecretion and gastroduodenal regurgitation. This can be exacerbated by gastric lavage, prolonged fluid therapy, anorexia and glucose administration. Hypokalaemia will contribute to production of cardiac arrhythmias and post operative ileus. Potassium levels should be monitored and corrected as necessary.

Feeding in the post operative period should consist of a semi-liquid, low fat, low protein diet until normal gastric motility is restored.

The use of H2 antagonists is recommended to reduce the acidity of gastric secretions and limit disruption to the compromised mucosal layer. Gastric motility modifiers may have a significant role in the post operative management of GDV as there is evidence to suggest an underlying dysrhythmia in dogs suffering from GDV. (eg metaclopromide 0.2-0.4mg/kg tid half an hour prior to feeding).

Operating on the Obstructed Intestine

Surgical Anatomy

The small intestines are approximately 3.5 times the body length in both dogs and cats. They comprise a fairly immobile duodenum, mobile jejunum and short ileum. These are attached to the mesenteric root by the mesentery. Located within the root of the mesentery are the cranial mesenteric artery, intestinal lymphatics and the mesenteric nerve plexus.

- Duodenum

Located dorsally on the right. It arises from the pylorus and then briefly courses cranially before the cranial flexure. It is limited in mobility by the mesoduodenum, the hepatogastric ligament (in which lies the common bile duct) and the duodenocolic ligament which defines the caudal duodenal flexure. The hepatogastric ligament can be partially transacted to improve exposure of the proximal duodenum and pylorus but the bile duct must first be identified and avoided.

The common bile duct and the major and accessory pancreatic ducts empty into the duodenum. These usually enter the lumen between 3 and 6 cm caudal to the cranial duodenal flexure. The proximal mesoduodenum also hosts the right lobe of the pancreas.

- Jejunum

Loops of jeunum form most of the intestinal mass and are very mobile.

- Illeum

The last 15cm of the small intestine is usually regarded as ileum, although there is no clear external demarcation between the jejunum and ileum. It can be identified by its antimesenteric ileal artery.

- Blood supply

The proximal duodenum is supplied by the gastroduodenal and cranial pancreaticoduodenal branches of the coeliac artery. The rest of the small intestine is supplied by the cranial mesenteric artery. The cranial mesenteric artery arises from the aorta at L1 or L2 and gives off two branches; the ileocolic and the caudal pancreaticoduodenal before branching into 12 to 15 jejunal arteries which divide several times to form the terminal arcades. The short vasa recti leave the terminal arcades and penetrate directly through the intestinal wall to develop into the serosal, intermuscular and submucosal vascular plexuses.

All visceral blood drains into the portal vein.

- Peyer's patches

These are aggregates of lymphoid tissue that occur normally throughout the small intestine. They appear as 1.5-2cm circumscribed elevations visible at the serosal surface. They can be prominent and are more numerous in the duodenum and proximal jejunum.

See chapter 2 on Gastrointestinal Surgery for the key principles and pitfalls of intestinal surgery.

Surgical intervention

This is indicated for diagnostic and therapeutic and reasons:

Diagnostic biopsy of the small intestine

- See earlier – Exploratory laparotomy and biopsy techniques

Therapeutic intestinal surgery

- Mechanical obstruction is the most common indication for intestinal surgery in small animals. Obstruction can be caused by foreign bodies, intussusseption, neoplasia and, less commonly, strangulation and adhesions.

Obstructive Intestinal Disease

Pathophysiology

- Obstruction of the proximal small intestine (duodenum) will stimulate vomiting with loss of acid secretions from the stomach and alkaline secretions from the duodenum. Dehydration and electrolyte loss occurs rapidly.

- Obstruction of the distal small intestine (jejunum,ileum) results in accumulation of fluid and gas proximal to the obstruction, leading to bowel distension. This leads to fluid and electrolyte loss and proliferation of bacteria within the intestinal lumen. If the mucosal barrier is impaired, diffusion into the peritoneal cavity can occur leading to peritonitis and toxaemia. Symptoms are less acute than those seen in more proximal obstructions.

- If strangulation of the small intestine occurs (as with mesenteric volvulus or entrapped herniated intestine), the mesenteric veins are more susceptible to occlusion than arteries due to their thinner walls. Occlusion of the mesenteric veins leads to oedema and haemorrhage into the intestine. The mucosal barrier will break down and allow bacteria and bacterial toxins to diffuse out into the peritoneal cavity. In addition, the strangulated section quickly becomes distended with fluid and gas and this leads to the development of hypovolaemia, electrolyte and metabolic disturbances. If the mesenteric arteries are also occluded, death occurs rapidly from perforation of the devitalised gut.

- Intestinal stasis (ileus) occurs in cases of peritonitis and following GDV. Stasis is followed by accumulation of gas and fluid in the intestine that cause distension. Bacteria proliferate and the result is fluid and electrolyte loss and toxaemia.

Foreign Body

Clinical signs are variable and can include anorexia, depression, dehydration, abdominal tenderness, vomiting and/or diarrhoea and possibly palpation of the foreign body or gas / fluid filled intestines. Radiography will usually reveal evidence of distended gas and fluid filled bowel loops which are suggestive of ileus proximal to the site of the obstruction. Dilated bowel greater than 1.6 times the dorsal-ventral width of the mid-body of the L5 vertebra is strongly suggestive of obstruction.

Serial radiographs (taken a few hours apart) may improve confidence of diagnosis if only mild ileus is present. The foreign body may be clearly obvious if it is radio-opaque. Alternatively, the use of contrast agents, such as Barium or BIPS, may allow confirmation of a partial obstruction or may outline a radiolucent foreign body.

Treatment requires correction of the dehydration and electrolyte abnormalities and antibiotics to protect against the bacterial proliferation in the intestine, however if there is fever, abdominal tenderness or evidence of peritonitis, immediate exploratory surgery is indicated as intestinal perforation can be strongly suspected.

In some cases, correction of the dehydration may promote intestinal motility and allow passage of the obstruction. Repeat radiographs prior to surgery may be advisable to ensure the foreign body has not migrated. Even short, sharp foreign bodies such as needles will often pass through the gastrointestinal tract without complication, although animals should be closely monitored for signs of peritonitis or perforation. If obstruction remains despite fluid therapy, enterotomy should be performed. A resection and end-to-end anastomosis should be performed if the bowel wall is necrotic. A complete exploratory laparotomy should be performed to check for additional foreign bodies or any other disease processes, for example neoplasia.

Linear Foreign Body

Linear foreign body, such as a string or stocking, are more commonly seen in cats and can extend from the base of the tongue or stomach to the rectum. Vomiting may be seen, but this is usually less than with completely obstructed cases. Generally there is anorexia and dehydration. Abdominal tenderness may be present and fluid and gas may be palpable within the small intestine. Characteristic plication of the bowel may be present as the intestine is gathered into pleats by peristalsis. Removal of the foreign body is indicated and multiple enterotomy incisions are usually required. The intestines should be checked for any perforations due to the linear foreign body as these are easily overlooked (they are most commonly on the mesenteric surface) and may lead to severe peritonitis. If areas of localised perforations are present, resection and anastomosis should be performed.

The prognosis for linear foreign body in the cat is good, as intestinal perforations are uncommon (this is associated with mortality). The prognosis in dogs is poorer with nearly 30% having gross perforations at the time of surgery.

Intussusception

Produced by vigorous contractions that force the proximal intestines into the lumen of an adjacent relaxed section. A focal change in the intestinal wall may cause a kink and then the invagination. Since the mesenteric vessels are included in the invagination, venous obstruction and rarely arterial occlusion and necrosis of the bowel can occur. Commonly fibrin is deposited between the intestinal segments, rendering the intussusception irreducible. Intussusception can occur anywhere along the intestine, but is most commonly found at the ileocaecal valve. Jejuno-jejunal sites are also seen. The intussusception can extend so that the segment actually protrudes from the anus. This is differentiated from rectal prolapse by passing a blunt probe along side the prolapsed tissue. If a more proximal intussusception the probe can be introduced a long way (not the case with rectal prolapse).

Intussusception is frequently associated with enteritis or disturbances to intestinal motility due to a localised disease such as neoplasia or foreign body or systemic illness. Enteral parasites or poor diet are also implicated, especially in younger dogs.

Obstruction is usually partial initially, but progresses to a complete obstruction and tissue ischaemia may lead to localised or generalised peritonitis. Diarrhoea may be seen and the stools may appear blood stained. A sausage shaped mass may be palpable in the abdomen. The diagnosis can be confirmed on radiography (the mass should be accompanied by distended small intestine containing fluid and gas) +/- barium or BIPS. Ultrasonography may also be useful for diagnosis of intussusception (the classic target-like sign). The intussusception is managed surgically. In early cases, the intussusception may be manually reduced (the intussuscepted portion is squeezed back rather than being pulled back), however in the majority of cases resection and anastomosis is required. Biopsies should be submitted of the involved intestine to identify a cause, particularly in the older animal.

Recurrence of intussusception is reported to occur and some surgeons recommend enteroplication to prevent this; however enteroplication is potentially associated with life threatening complications such as adhesions, strangulation and entrapment. A recent study reported a higher rate of complications associated with enteroplication compared with when it was not performed, therefore it should be used with caution and care needs to be taken with the technique if used. Therefore I generally reserve it for recurrent cases only.

Early resumption of oral feeding to encourage return of normal peristalsis is recommended to limit the potential for recurrence.

Neoplasia

The most common types of neoplasia to involve the small intestine are adenocarcinoma and lymphoma. Other neoplasias include leiomyoma/sarcoma (now often classified as GIST – gastrointestinal stromal tumours – special histopathological techniques are required to differentiate these further), mast cell tumour, haemangiosarcoma, fibrosarcoma etc. Most intestinal tumours are malignant and may spread by direct extension into the abdominal cavity or via lymphatic spread.

- Alimentary lymphoma in the cat is characterised by gastric, intestinal or mesenteric lymph node involvement and is perhaps one of the most common forms of feline lymphoma. Gastrointestinal lymphoma may present as a solitary mass or as a diffuse infiltration of the bowel. Most cats with alimentary lymphoma are FeLV negative.

- Adenocarcinoma are locally invasive. In the dog, they tend to occur in the duodenum. In the cat, they are more likely to be in the jejunum. Collies and German Shepherd Dogs and Siamese cats are predisposed. Adenocarcinoma tends to cause progressive annular constriction and stenosis of the intestine. Ulceration of the surface leads to haemorrhage and development of melena and iron deficiency anaemia. Palpation and radiography can be used to confirm the presence of a mass. Thoracic radiographs should be obtained to assess for metastasis. Ultrasonography may permit a fine needle aspirate of the intestinal mass to be obtained for cytologic assessment of the mass.

- Surgical excision is the treatment of choice for most intestinal tumours. Prior to resection, staging is required and perform a complete exploratory laparotomy to determine if the tumour has metastasised, for example to liver of lymph nodes. Ideally 4 cm margins are obtained and the tissue is submitted for histopathology. Unfortunately surgery is only palliative for most malignant tumours but is curative if benign.

- Lymphoma should be regarded as a systemic disease, even if the mass is solitary. Treatment with chemotherapy is therefore appropriate. Surgical resection of the palpable mass should be performed but should be considered a biopsy procedure only.

Peritonitis:
Diagnosis and Management

Pathophysiology of peritonitis

Peritonitis is inflammation of the peritoneum. Vascular dilatation, increased capillary permeability and the migration of inflammatory cells into the peritoneum occurs in response to inflammatory mediators released predominantly from activated macrophages. These reactions are defensive and result in the neutralisation and phagocytosis of bacteria and rapid lymphatic clearance form the abdomen. Fibrin is employed to seal off perforations and localise infection. Intestinal ileus develops, possibly helping to reduce distribution of contaminated peritoneal fluid. The omentum is able to aid in the walling off of infections within the abdominal cavity due to its mobility. The omentum has a rich vascular and lymphatic supply which increases oxygen tension in the wounded area, thereby creating unfavourable conditions for the obligate anaerobe. The omentum also promotes vascularisation and enhances survival of ischaemic tissue.

If generalised peritonitis develops due to an overwhelming event or continuing contamination, the inflamed peritoneum becomes a freely diffusible membrane, allowing massive influx of fluid and plasma proteins, including fibrin, into the peritoneum. Diaphragmatic lymphatics that normally return fluid to the systemic circulation become blocked by fibrin, which is deposited into the abdomen in an effort to contain the contamination. If the patient is anorexic, vomiting or has diarrhoea, additional fluid and protein deficits will occur. The resulting hypovolaemia leads to decreased cardiac output, poor tissue perfusion, cellular hypoxia and cellular death. Ischaemia and inflammation may result in compromise of the intestinal mucosa and allow bacterial translocation. Inflammatory mediators released by activated macrophages and neutrophils and absorption of bacterial endotoxins leads to further peripheral vasodilation, increased vascular permeability, decreased cardiac function and activation of the coagulation cascade and leads to the systemic inflammatory response syndrome and disseminated intravascular coagulation which eventually leads to failure of the lungs, liver, kidneys and heart, resulting in death of the patient.

Peritonitis is classified as diffuse or localised, acute or chronic, primary or secondary and septic or non septic.

Primary peritonitis occurs without an obvious source of contamination and is thought to result from haematogenous spread of micro-organisms or peritoneal disease such as feline infectious peritonitis (FIP).

In most cases, peritonitis is secondary. Causes of secondary peritonitis include:

$\Rightarrow$ Septic peritonitis

Septic peritonitis is one of the most serious complications of abdominal surgery. Up to 50% of all cases of peritonitis follow dehiscence of surgical incisions into a hollow viscus (for example intestine, bladder, gall bladder). Contamination of the peritoneal cavity with intestinal contents can also occur due to migration of foreign bodies, penetrating injuries or blunt abdominal trauma, such as from a road accident. Migration of bacteria across the gut wall can occur following intestinal obstruction or ischaemia due to alteration of the mucosal barrier. Finally a rupture of a septic focus (eg prostatic, hepatic, splenic, renal or pancreatic abscess, necrotising cholecystitis or pyometra may also lead to a generalised septic peritonitis. Other causes include penetrating wounds and migrating foreign bodies. In most cases, culture of the septic peritoneal fluid usually reveals a mixture of organisms, both anaerobes and aerobes.

$\Rightarrow$ Chemical peritonitis

Chemical peritonitis may develop as a consequence of irritation of the peritoneum with a variety of agents including bile, gastric contents, pancreatic secretions, urine, antiseptics, anti-bacterial powders and radiographic contrast agents, for example barium. The severity of the peritonitis that develops depends on the amount and type of irritant involved. Bile salts and urine are only mildly irritating and contact with the peritoneal surface typically needs to occur for over 24 to 48 hours before serious peritonitis develops (infected urine and bile will cause a rapid onset of serious peritonitis). Gastric and pancreatic fluids are extremely irritating and can incite an intense peritoneal reaction.

 Even if the inciting agent is sterile, bacterial contamination of the peritoneal cavity can occur due to transmural migration of bacteria and bacterial toxins across damaged intestinal barriers.

$\Rightarrow$ Mechanical Peritonitis

Other potential causes of peritonitis include a variety of foreign bodies, particularly those left behind after abdominal surgery – for example surgical swabs. Powder from surgical gloves is associated with occasional peritoneal reactions. Bacteria can also migrate into the abdomen along peritoneal drains and catheters.

Clinical signs

Clinical signs associated with peritonitis can be variable, and can include anorexia; depression; pyrexia or hypothermia; vomiting and diarrhoea; dehydration; ascites with tachypnoea and respiratory distress secondary to marked ascites; serosanguinous or purulent fluid dripping from a previous surgical wound; lack of intestinal sounds on auscultation; and

progressive signs of shock. Postural changes in an attempt to relieve abdominal discomfort may be seen but abdominal pain is not a consistent finding.

Diagnostic investigation

A high index of suspicion is often apparent from the recent history – for example after recent intestinal surgery or an abdominal penetration, septic peritonitis is a major consideration. If patients have obvious free fluid on physical examination, abdominocentesis should be performed. In less obvious cases, abdominal radiography or ultrasonography aid diagnosis.

- Haematology and Biochemistry

Patients with septic peritonitis are often hypoproteinaenic and hyperglycaemic. An increase or decrease in the white blood cell count with an increase in immature (band) neutrophils reflecting inflammation is likely to be seen.

Other biochemical abnormalities can reflect the aetiology of the peritonitis, for example hyperkalaemia associated with urine leakage, increased total bilirubin and liver enzyme activities in bile peritonitis cases, increased urea, creatinine and potassium in cases of urinary obstruction or dehydration, increased amylase and lipase in pancreatitis.

- Radiography

The diagnostic value of radiography depends on the type of injury and the time elapsed since the injury occurred. Typical radiographic changes associated with peritonitis include

 The presence of free gas in the abdomen, however gas can remain in the abdomen for up to three weeks post surgery. The presence of free gas in the abdominal cavity without a recent history of surgery is highly suggestive of a visceral rupture and immediate exploratory surgery is indicated.

 Fluid effusion: This causes a lack of radiographic contrast, however small amounts of fluid are unlikely to be detected.

 The presence of ileus with intraluminal accumulation of gas is a strong indication of peritoneal irritation and further investigation is recommended.

 Contrast studies of the gastrointestinal tract are rarely indicated, due to the considerable time taken to obtain a barium study and the risk of exacerbation of peritonitis should barium leakage into the abdomen occur. Contrast investigation of the urinary system with iodine-based contrast materials may be invaluable if trauma is suspected.

- Abdominocentesis

Obtaining a sample of free abdominal fluid may give a rapid indication for the requirement of exploratory laparotomy. A diagnosis of septic peritonitis is made based on cytological evidence of degenerate neutrophils, intracellular bacteria and foreign material. With most peritoneal diseases, copious free abdominal fluid usually collects and collection of a sample using a sterile four quadrant tap technique is usually easy and straightforward. Avoiding the cranial abdominal midline is important to avoid the falciform fat. It has been shown that at least 25ml/kg of free fluid is required for this technique to be successful, so a negative tap does not rule out a peritoneal effusion.

 If the glucose concentration in the blood is 1.1 mmol/l (20mg/dl) greater than the glucose concentration of the peritoneal fluid, this is highly indicative of septic peritonitis.

→ Diagnostic peritoneal lavage (DPL) can improve the diagnostic yield. This is performed by inserting a DPL catheter sterilely, infusing 22ml/kg of warmed Hartmann's solution under gravity, rolling the patient and then collecting 10-20ml of fluid by free drainage. However samples that have been collected by diagnostic peritoneal lavage will be diluted which may affect results of fluid analysis. DPL has now been largely superceded by ultrasound-guided aspiration.

 In-house cytology of abdominal fluid is essential as in general the delay in results from an external lab might make the results largely academic! Assessment for evidence white blood cell cells and their nature is key. Evidence of degenerate neutrophils with intracellular bacteria or gross foreign material makes a rapid diagnosis of septic peritonitis and gives the indication for surgical management.

→ Questionable results in an otherwise stable patient should be assessed by a qualified clinical pathologist. While awaiting results very close monitoring, hospitalisation and repeat sampling in case of deterioration is prudent.

Management

Preoperative management

Peritonitis is a surgical emergency and mortality associated with septic peritonitis can be high. This is particularly true if management is compromised by diagnostic failure, inadequate treatment, lack of resources or clinical inexperience. An honest appraisal of the situation is essential and for many cases prompt referral for intensive treatment is the optimal management option.

Patient stabilisation is the first priority. Hypovolaemia associated with loss of fluid into the abdomen +/- fluid loss from inappetance, vomiting and diarrhoea is likely to be present. Shock doses of lactated ringers solution (90ml/kg/hour) are indicated. I usually start this rate of fluids and then reassess the dog's parameters and requirement for continuing at this level after 15 minutes. If still necessary I will continue this fluid level and reassess again after a further 15 minutes. Catheters in both cephalic veins or a central jugular catheter (if not placed preoperatively, definitely place a central line once anaesthetised) will probably be needed to allow this to occur. Hypertonic saline at 3-5mls/kg/hr may be administered over a 20 minute period. Isotonic fluids at surgical rates (10ml/kg/hour) should always be given after administering hypertonic saline. Colloids such as hetastarch may be administered (5-10ml/kg boluses over 10-15 minutes). Care must be taken in cats to avoid fluid overload (shock dose of crystalloids 55ml/kg/hr).

If required, blood or plasma transfusions can be considered to correct anaemia or severe hypoproteinaemia. Another alternative is the use of the haemoglobin based oxygen carrying solution Oxyglobin (5-10ml/kg/hr) and this has been shown to be beneficial in the resuscitation of the patient in hypovolemic shock. Oxygen is also provided as this can greatly aid tissue oxygenation in the dog in hypovolemic shock. Electrolyte abnormalities should be addressed if present, for example hyperkalaemia in a patient with uroabdomen.

Antibiotic therapy should begin immediately, with broad spectrum antibiotic used until results of culture and sensitivity are available. The likely bacteria will be enteric and therefore a mixture of gram-positive and gram-negative anaerobes and aerobes is likely.

A recommended regimen includes cefuroxime (22mg/kg intravenously every eight hours) or amoxicillin/clavulanate (Augmentin 20mg/kg intravenously q6-8 hrs) in combination with metronidazole (10 to 20mg/kg daily). This can be administered intravenously for rapid attainment of systemic levels. Intraperitoneal antibiosis is not recommended because all systemic antibiotics will provide adequate levels within the peritoneal fluid. Some antibiotics may be irritant if applied directly to the peritoneum and also in advertent administration of toxic doses may occur with therapy be combined routes.

Surgical Treatment

Surgical actions may potentiate or exacerbate intra-peritoneal contamination, for example breaking down firm adhesions potentially results in dissemination of bacteria and bacterial toxins into the abdomen. Failure to remove necrotic debris or placement of excessive amounts of suture material provide a nidus for bacterial proloiferation and blood clots left in the abdomen provide nutritive media for the growth of bacteria. Peritoneal fibrin should be

removed. Haemoglobin has also been shown to interfere with the chemotactic response of white cells, hence retarding bacterial clearance.

In the relatively common case of an intestinal wound dehiscence, this will generally involve resection and anastomosis of the area (except in the descending duodenum) followed by suture line reinforcement (I generally perform a serosal patch in the face of septic peritonitis as delayed wound healing is likely – see earlier)

In addition copious lavage with large volumes of warm, sterile isotonic fluid should be performed to remove debris and decrease bacterial load and endotoxin concentration

The primary goal of surgery is to <u>find and correct the source of the contamination</u>

 A minimal volume of initial lavage of 200ml/kg of sterile isotonic saline has been recommended or is continued until the returning lavage fluid appears clear.

Lavage fluid should be completely removed from the abdomen before closure, as excessive fluid will lower the oxygen tension of the abdomen, therefore favouring the proliferation of anaerobic bacteria.

 Do not lavage with antiseptic solution as these are often irritant and can affect the phagocytic activity of inflammatory cells. Iodine toxicosis can occur due to excessive absorbance across the peritoneum if iodine solutions are used.

Ongoing Drainage

After successful surgical management of patients with mild chemical peritonitis (for example after a ruptured bladder) the abdomen can be closed without ongoing drainage, but for nearly all other cases ongoing drainage of the abdominal cavity following surgery is essential.

Passive drains placed in the abdomen in a routine, dependent manner can become easily blocked with fibrin, omentum and debris, and also provide a potential route of contamination. The negative pressure within the abdomen also inhibits the egress of fluids. The key options are therefore either CLOSED ABDOMINAL DRAINAGE (with a Jackson-Pratt catheter) or use of OPEN PERITONEAL DRAINAGE.

Closed abdominal drainage depends on the correct type and placement of drain so that it is less likely to block with fibrin or omentum. Generally a multi-fenestrated closed suction drains (the Jackson-Pratt drain is highly recommended) is placed in the peritoneal cavity between the liver and diaphragm (this is the direction of peritoneal fluid flow). In large dogs or generalised peritonitis a second J-P drain is placed caudally ventral to the bladder. These are

then attached to large grenade type suction bulbs to supply negative pressure. The volumes of fluid collected can be measured and the bulbs emptied in a sterile manner. The drains are generally very effective and very well-tolerated. This technique also allows evaluation of fresh peritoneal fluid samples collected from the drain to determine the progression of the peritonitis. As the direction of fluid flow is out of the abdomen the risk of ascending infection is reduced but hygiene and infection control measures apply. For drains in place for more than 3 days, I will generally change the grenade bulb to reduce the growth of contaminants.

Open peritoneal drainage can provide drainage of the entire abdomen. It is accomplished by delaying closure of the linea alba and skin for several days after the initial surgery. The linea alba is instead loosely apposed with a permanent monofilament suture material (eg nylon, prolene) leaving a gap of 2 to 4 cm between the wound edges at the cranial part of the abdomen. The caudal part of the abdomen is closed normally. In dogs, a urinary catheter attached to a closed collecting system is placed. The wound surface is then covered with a thick layer of absorbent towels (eg steralised nappies) followed by an impermeable layer (such as plastic sheeting) then a layer secured in place with gauze bandages. Dressings should be changed as necessary but initially at least twice daily, in a sterile manner. The abdominal wound should be examined at each bandage change and any adhesions broken down and necrotic tissue in the wound debrided. This usually requires sedation or general anaesthesia of the patient. Repeat lavage may also be performed depending on the nature of the discharge. When the drainage from the wound has reduced to a level where only a low level of discharge is noted through the bandage over a 24 hour period, closure of the abdomen in a routine manner can be performed (this is usually after 3-5 days).

Potential complications of open peritoneal drainage include hypoproteinaemia, anaemia, increased adhesion formation within the abdomen, herniation of abdominal contents and self mutilation, hypokalamia and hyponatraemia. Careful monitoring of fluid losses and appropriate intravenous fluid therapy is essential. The morbidity, time, expense and nursing considerations for OPD can be considerable. Our preference is to attempt closed abdominal drainage when possible, but this is decided on a case by case basis. Generally OPD is reserved for cases with severe highly fibrinous peritonitis with marked contamination.

Postoperative care
As well as peritoneal cavity drainage, these patients need very intensive care and monitoring following surgery. Continuous monitoring of fluid balance, electrolyte, acid base parameters and analgesia as well as monitoring of vital parameters is necessary. Nutritional support is absolutely essential at an early stage due to the high risk of significant malnutrition from protein loss and anorexia. Placement of an appropriate feeding tube depending on the underlying clinical problem, should be performed under the initial general anaesthetic.

On admission to ICU these patients will generally have a central jugular line, an indwelling urinary catheter (essential to monitor the effectiveness of intravenous fluid support), a Jackson-Pratt abdominal drain and an appropriate feeding tube (generally an oesophagostomy tube). Therefore pre-emptive assessment of the considerable requirements of these patients is one of the keys to success.

Reported survival rates for peritonitis range from 32-80%, with a trend towards improved survival more recently which is probably associated with improvements in critical care management. A recent study on the management of septic peritonitis in cats and dogs of gastrointestinal origin had a survival rate of just under 90% using closed suction drainage and intensive patient pre-, intra- and postoperative care.