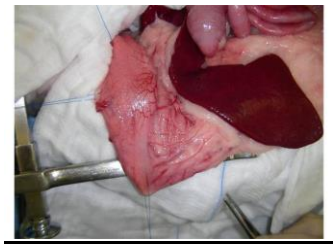
cpdsolutions
transferring knowledge through excellence in training

Gastrointestinal Surgery Mini Series

Session Two: Exploratory Laparotomy and Stomach

Alison Moores BVSc(Hons) CertSAS DipECVS MRCVS
RCVS Specialist in Small Animal Surgery (Soft Tissue), European Specialist in Small Animal Surgery



CPD Solutions Gastrointestinal Surgery Mini Series

Session Two - Gastrointestinal disease: Exploratory Laparotomy and Stomach

©Alison Moores BVSc(Hons) CertSAS DipECVS MRCVS
RCVS Recognised Specialist in Small Animal Surgery (Soft Tissue)
EBVS® European Specialist in Small Animal Surgery

Anderson Moores Veterinary Specialists, Hursley, Hampshire, UK
www.andersonmoores.com

Tips for Improving Exploratory Laparotomy

What are you hoping to achieve by doing an ex lap?

Firstly don't rush into an exploratory laparotomy without taking time to consider what you want to achieve.

There is no indication or justification for performing an exploratory celiotomy without some knowledge of what disease process may be occurring or without an intention to perform biopsies for the purpose of achieving a diagnosis. An exploratory celiotomy is not a substitute for a full diagnostic investigation and should only be performed when the initial investigation indicates a need for collection of biopsy samples of abdominal organs or surgical intervention to control a disease process occurring in the abdomen. If you do get caught with an unproductive exploratory laparotomy then always consider biopsies of the liver and gastrointestinal tract as a minimum.

Diagnosis: Get as much information as possible before surgery - Physical examination; Blood tests; Radiography (with contrast if necessary), ultrasonography, +/- advanced imaging e.g. MRI; Endoscopy; Abdominocentesis.

Only use surgery for diagnosis when all other methods already done: To evaluate gross disease and/or to collect samples that can't be obtained by another route for histopathology, cytology and culture.

Don't stop when you find a lesion – there may be more than one!

Treatment: Avoid the 'peek & shriek'; if you can't deal with all possible scenarios, seek referral instead of operating.

Key points:

- Be thorough & systematic.
- Take appropriate samples.
- Be prepared for the likely diagnoses.

Common mistakes:

- Failure to make a large enough incision.
- Failure to explore the entire abdominal cavity.
- Failure to biopsy.
- Failure to be prepared for the likely diagnoses

Key instruments:

- 4 waterproof (or cotton) drapes – you won't be able to perform a complete surgery through a small fenestrated drape
- Abdominal retractors (e.g Balfour, Gosset) – can't perform thorough ex lap without them
- Suction for peritoneal fluid & lavage solutions
- Laparotomy (large) swabs with radio-opaque markers
- Good lighting
- Stay sutures – monofilament suture & old mosquito forceps

Hints for getting the most out of your ex lap:

Incision:

1. From xiphoid to pubis for thorough exploration – any smaller & you may not be able to open the incision wide enough. It also causes less bruising.
    a. For the caudal abdomen, a caudal half to third incision may be sufficient for superficial or mobile structures, such as the bladder. For deeper structures, a more cranial incision is needed.
    b. For defined and limited surgery, such as an intestinal foreign body or splenectomy, a central half or third incision may be sufficient. However, this relies on a very thorough abdominal ultrasound prior to surgery to ensure no other lesions are present. Unfortunately abdominal ultrasound by a non-specialist often misses even very large and obvious lesions, so a large enough incision is needed to explore the entire gastrointestinal tract (for foreign bodies and masses) and the liver (if neoplastic disease is suspected).
    c. For the cranial abdomen, extending the incision caudally, potentially to the pubis, allows the abdomen to be opened wider, and improves access to the cranial abdomen and the deeper parts of the abdomen.
2. Males –skin incision goes lateral to the prepuce, ligate preputial branch of caudal superficial epigastric artery
3. Enter linea alba at umbilicus as protected by falciform fat – extend with mayo scissors or blade, stay on midline even in males (pull prepuce to one side to see better)
4. Remove falciform fat – small blood vessels at edge, larger vessel cranially needs coagulation or ligature
5. Place moist laparotomy swabs along incision edge & place retractors. Place further swabs at each end. This allows viscera to be exteriorised with minimal damage

Initial exploration:

Exploration of the abdomen must be performed in a systematic manner, utilising the consistent location of each organ between individuals. Thorough exploration of the abdomen is essential for identification of all abdominal pathology. Start the exploration in the cranial abdomen and progress caudally, systematically evaluating all organs. Reflection of the left lateral and medial liver lobes towards the right with caudal retraction of the fundus of the stomach and spleen reveals the gastric cardia and abdominal oesophagus, left aspect of the diaphragm, cranial pole of the left kidney and left adrenal. The quadrate, right medial, right lateral, and caudate and papillary processes of the caudate lobe are inspected visually and gently palpated.

The gall bladder, cystic and common bile ducts should be carefully evaluated. The ability to express the gall bladder does not necessarily demonstrate patency of the common bile duct since reflux of bile into the hepatic ducts can also lead to gall bladder emptying. The stomach is then traced to the pylorus and proximal duodenum. The gastric limb of the pancreas is closely associated with the dorsal leaf of the greater omentum. Visualisation is achieved by reflection of the greater omentum ventrally and cranially or by creating a window in the omental bursa. Elevation of the descending duodenum reveals the right kidney, right adrenal and portal vein. The right ovary and uterine horn are visualized in female dogs. The duodenal limb of the pancreas is inspected. The duodenum is traced to the duodenocolic ligament and beyond to the jejunum. The remainder of the intestinal tract is inspected in an oral to aboral direction. Elevation of the descending colon reveals the caudal pole of the left kidney and the left ovary and uterine horn in female dogs. Elevation of the bladder reveals the ureters entering the caudal and dorsal bladder trigone and both vas deferentia in male dogs.

1. Find the descending duodenum & descending colon that have short mesenteries. Displace medially & use to cradle the intestines for an unimpeded view of the paralumbar areas.
2. Collect samples of peritoneal fluid if not done already & suction all fluid out.

Liver:

1. Palpate with gentle fingertips or flat of hands.
2. Se moist laparotomy swabs cranial to liver to allow caudal liver retraction in deep chested dogs.
3. Biopsy in areas that are easily accessible in case of haemorrhage.

Stomach:

1. Palpate left cranial quadrant to examine oesophagus.
2. Place a stomach tube if stomach is full of fluid or gas, to allow better palpation and prior to any gastrotomy.
3. Palpate & visualise stomach for masses & FBs.
4. Examine dorsal surface by displacing stomach cranially. Only open omentum if stomach or pancreatic disease means that they must be examined closely.
5. Palpate pylorus. Note that it is firm.
6. Place stay sutures to help move stomach around or if performing gastrotomy.

Intestines:

1. Mesoduodenum & mesocolon are short so have limited mobility. They are attached by duodeno-colic ligament caudally
2. Root of mesentery turns anti-clockwise in development at level of LI. Cranial mesenteric artery located in root of mesentery
3. Jejunum - smaller diameter, long, relatively empty, thin wall, arcadial vessels
4. Ileum -thicker wall, antimesenteric vessel (ileal branch of ileocaecal artery), in proximity to caecum, & attached by ileocaecal fold
5. Caecum - lies to the right of the median plane between descending & ascending duodenum. Corkscrew coil in dogs, simple short comma shape in cats
6. Colon - beyond ileocaecocolic junction, close to left abdominal wall. Pale, purple hue, longitudinal muscle striations, segmental vessels, larger diameter, faecal content.

Spleen

1. To resect spleen:
    a. Ligate small hilar vessels.
    b. Ligate medium sized vessels to cranial, mid and caudal spleen, and the short gastric vessels.
    c. Ligate the splenic vessels after the pancreatic branch, plus the short gastric vessels.

Urinary bladder, ureters & urethra:

1. Bladder retroflexion increases exposure of ureters, cervix etc
2. An enlarged bladder will decrease access to the caudal abdomen
3. Place stay suture in bladder apex for easy handling
4. Monitor ureters for peristalsis
5. Ureters can be palpated entering bladder – turn back on themselves

Prior to closure:

- Swab count
- Instrument count
- Change instruments & gloves if there has been contamination
- Place biopsies into appropriate pots
- +/-lavage

Stomach

The stomach is divided into several regions: the cardia, fundus, body, greater curvature, lesser curvature, pyloric antrum and pylorus. The greater omentum is attached to the greater curvature, the lesser omentum to the lesser curvature. The cardia has cells that produce mucus to lubricate food. The fundus and cardia contain cells that produce pepsinogen and parietal cells producing HCl. The antrum has cells with mucus and gastrin-secreting cells.

Vascularization of the stomach is from the right and left gastric arteries to the lesser curvature, by the left and right gastroepiploic arteries to the greater curvature arteries, and from the short gastric arteries to the fundus/body.

The stomach wall consists of four layers - mucosa, submucosa (suture holding layer as it resists tension the most), muscularis and serosa.

Gastric-Dilatation-Volvulus Syndrome

Gastric-dilatation-volvulus (GDV) syndrome encompasses acute gastric dilation, acute gastric dilatation-volvulus and chronic gastric volvulus. The acute conditions are characterised by a dramatic increase in intra-gastric pressure, with significant consequences on the cardiovascular, respiratory and gastrointestinal systems. These two conditions are hard to differentiate clinically, and may indeed be part of the same entity.

The exact cause of GDV hasn't been ascertained. It is known that the majority of gas accumulation within the stomach is atmospheric air, although gas produced from fermentation of food, metabolic reactions, diffusion from blood and gas-producing organisms will play a role. The inciting cause may be gastric volvulus with subsequent disruption of gastro-oesophageal and pyloric function. Evidence for this comes from the fact that some dogs present with chronic volvulus with no history of an episode of acute dilation.

Furthermore, dogs with a previous episode of GDV who are treated with gastropexy (and therefore not at risk of volvulus) are unlikely to have a recurrence, whereas dogs not treated with a gastropexy and at risk of volvulus, have a high chance of recurrence. Other evidence suggests that gastric dilatation is the initiating event. Dilation of the stomach may occur secondary to gastro-oesophageal sphincter and pylorus dysfunction, and eventually volvulus occurs, leading to GDV. Some dogs present with just gastric dilation – it may be that gas accumulates so fast that volvulus can't occur, or these animals may be presented earlier in the course of disease when volvulus has not yet occurred.

Perpetuating factors are also involved, and when the GDV has become established, anatomical and physiological mechanisms will be altered and play a role in the continuation of the disease. Fluid will accumulate in addition to ingested fluid by sequestration (secondary to venous congestion) and accumulation of gastric secretions.

Various environmental, anatomical, physiological and pathological mechanisms have been ascribed to the development of GDV. It is difficult to assess how important individual risk factors are in a disease that has a low prevalence in a hospital population. Theories regarding potential risk factors for the development of GDV include:

- Breed – giant breeds are 1.27 times as likely to suffer a GDV than large breed dogs. Commonly affected breeds include Great Danes, German Shepherd Dogs, St Bernards, Weimeraners, Bloodhounds, Standard Poodles, Gordon setter, Irish setters, Bassett hounds and large mixed breed dogs. The Shar Pei is a medium sized dog that is predisposed to GDV. It has been reported in cats.
- Relations – dogs that have a first degree relative (parent, sibling, offspring) with a history of GDV are at increased risk themselves, and may benefit from prophylactic gastropexy.
- Body conformation – i.e. deep chested breed. It is not known how this may be related, but may be due to the position of the cardia.
- Bodyweight – underweight dogs are at increased risk
- Gastric ligament laxity – gastro-hepatic ligaments are stretched in dogs that have suffered a GDV but this could be a result of GDV rather than the cause.
- Diet – risk factors include dogs fed one meal daily, only one type of food, pieces of food less than 3cm, a raised food bowl and rapid eating (although the latter is not proven in all studies). There has been no proven association between GDV and the dog's appetite and a recent period of exercise has not been conclusively shown to be a risk factor. Feeding table scraps, canned food and feeding between meals may decrease the risk.
- Gastric position - In a cadaver study it was shown that gastric dilation could only result in GDV if the stomach was already malpositioned, but the association hasn't been studied further.
- Gastric motility – gastric emptying is decreased in dogs with GDV. Many dogs have a history of motility disorders, such as vomiting, prior to GDV but it is possible that this is an effect of GDV not a cause. However, it may help to explain why some dogs continue to have episodes of dilation following gastropexy. Be aware that stress and certain anaesthesia drugs may affect gastric motility, which may explain why recent kenneling or a recent car journey will significantly increase the risk of GDV.
- Concurrent disease - dogs are at increased risk if they suffer from splenic disease or inflammatory bowel disease. An association between GDV and previous splenectomy was disproven in one recent study but then demonstrated in another.
- Gastrin – in one study, dogs with GDV had higher serum gastrin levels than control dogs, but these results have not been repeatable

- Aerophagia is a known risk factor for GDV in Irish Setters, and may explain why dogs with nasal mite infestation are at risk of developing GDV.
- Personality – a number of studies have shown that dogs perceived by their owners to be happy are less likely to develop GDV than nervous or fearful dogs.

Pathophysiology of GDV

The development of gastric dilation has a profound effect on the cardiovascular and respiratory systems, as well as local effect on the stomach itself. Numerous secondary events follow so it is important to understand the pathophysiology of this disease in order to recognise and treat them.

Primary cardiovascular effects:

- Venous occlusion
    - Decreased venous return - Decreased preload/ cardiac output/ systemic blood pressure
    - Portal hypertension and splanchnic pooling - Interstitial oedema and hydrostatic pressure - Decreased vascular volume, hypovolaemic shock, poor organ perfusion - Secondary effects

As the stomach dilates the low-pressure veins of the abdomen (the caudal vena cava, portal vein and splanchnic vessels) collapse, significantly decreasing venous return and cardiac preload. This in turn results in a marked hypovolaemia and systemic hypotension. Hydrostatic pressure in the splanchnic vessels increases, with a resultant extravasation of fluid into the gastrointestinal tract, abdominal cavity, and a further decline in intravascular volume. The net result is hypovolaemia and poor tissue perfusion.

Secondary cardiovascular events:

Hypovolaemia and hypoxaemia cause a release of catecholamines. Cardiac muscle work is increased in response, thereby increasing cardiac tissue metabolism. At the same time, blood supply directly to the heart tissue is limited by poor cardiac output, increased heart rate with less time spent in diastole (blood flow to the heart occurs in diastole not systole) and poor cardiac cell contractility in the face of acidosis. Ventricular cells are particularly sensitive to hypoxia, and changes in cell function and cell death result in alterations in electrical activity, with the development of ectopic foci, seen clinically as arrhythmias. Myocardial depressant factor, a substance or substances released from ischaemic gastrointestinal and pancreatic tissue, and toxins and waste products released after reperfusion, will also promote arrhythmia.

Direct respiratory effects:

The dilated stomach presses on the diaphragm and inhibits normal respiratory movements. Initially the animal will compensate by increasing respiratory rate and effort, until decompensation occurs. Failure to effectively achieve oxygen and carbon dioxide exchange causes hypoxaemia and decreased cellular respiration. Hypercapnia will exacerbate the acidaemia associated with poor tissue perfusion and metabolic acidosis.

- Decreased tidal volume and decreased normal excursions -
    - Increased respiratory rate and effort -
        - Increased metabolic demand
        - Decompensation -
            - Increased CO2 -

        - Further increased re respiratory rate and effort
        - Respiratory acidosis Acidaemia
    - Decreased O2 -
        - Decreased cellular respiration

Direct gastrointestinal effects:

- Increased gastric pressure -
    - Decreased myoelectrical activity - Increased gas and fluid
    - Decreased gastric perfusion (also from short gastric avulsion) -
        - Decreased gastric perfusion -
            - Mucosal haemorrhage and necrosis
            - Serosal haemorrhage and necrosis
- Short gastric artery avulsion - Haemoabdomen

Intra-gastric pressure will exceed trans-mural vascular pressure, and decrease perfusion of the gastric wall. The gastric mucosa, with a higher metabolic rate than the muscularis or serosa, is affected first and will eventually become necrotic. Volvulus may cause rupture of the short gastric arteries, which in itself is unlikely to affect gastric perfusion, but may result in significant haemoabdomen and will contribute further to hypovolaemic shock. Distension of the stomach will interfere with normal myoelectrical activity, thus delaying the animal's recovery after treatment. Gastric perforation is unlikely to occur as a consequence of dilation alone, as the bursting strength of the stomach is very high, but may occur secondary to gastric necrosis

Secondary metabolic effects:
Poor tissue perfusion and hypoxaemia lead to a change from aerobic to anaerobic metabolism with the production of lactic acid. Lactic acid is also produced by endotoxins regardless of tissue oxygen tension. The lactic acid cannot be recycled by buffering with bicarbonate, intracellular buffers, or renal excretion, due to poor tissue perfusion, resulting in metabolic acidosis as the most common metabolic abnormality. Initially the respiratory system may be able to compensate, but it is unable to compensate for marked metabolic acidosis, and respiratory acidosis may occur with severe respiratory distress. Occasionally, there may be a degree of metabolic alkalosis if there is significant sequestration of hydrogen ions into the gastrointestinal tract.

Hypokalaemia, secondary to gastrointestinal sequestration and renal loss, is commonly made worse by the administration of large volumes of low-potassium fluid.

Glucose production is initially increased in response to catecholamines, but anaerobic metabolism is inefficient and the poorly perfused liver cannot meet increased glucose demand. The presence of hypoglycaemia is a negative prognostic indicator.

Tissues that are poorly perfused switch to anaerobic metabolism, and lactic acid, waste products and endotoxins accumulate. The arachidonic acid cycle is stimulated, heparin and enzymes are released from damaged cells and increased intracellular calcium promotes the formation of xanthine oxidase, which will form oxygen free radicals with oxygen. These substances are released when the tissues become reperfused and will have detrimental effects on the local vasculature. Furthermore, they will damage distant organs with effects such as myocardial injury and systemic inflammatory response syndrome.

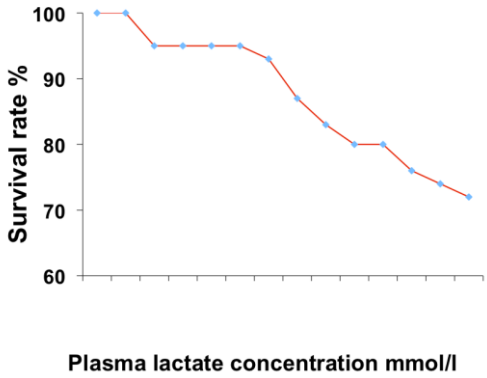
Other secondary effects:

Poor pathogen delivery to haemo-lymphatic tissues
Poor renal perfusion, seen initially as decreased urine output or anuria
CNS effects due to poor perfusion, effects of toxins and limited delivery of oxygen or nutrients.

Diagnosis of GDV

The basic diagnosis of the acute condition is usually easy to make based on a history of restlessness, non-productive retching, and depression or collapse. Physical examination demonstrates moderate to marked tympanic gastric distension. Some animals have a fluid thrill associated with haemoabdomen or fluid sequestration. However, diagnosis of early GDV can be difficult to make in deep chested dogs when the stomach is only moderately distended, but these dogs will already be showing signs of an abdominal crisis and hypovolaemia. Most animals show some evidence of hypovolaemia, and possibly endotoxic shock, characterised by tachycardia, tachypnoea, pale mucous membranes and slow capillary refill times. Mucous membrane colour tends to be injected with a hyperdynamic refill (<1 second) in compensatory shock. As the animal decompensates, it becomes bradycardic, with poor respiratory function, hypotension, hypothermia and very poor pulse quality. Treatment of these animals needs to be aggressive to avoid death.

I like to gain a basic database of blood tests, and run these whilst the animal is being stabilised. If time doesn't allow, the results should be made available in the immediate post-operative period, in order to make decisions regarding medical treatment, and to have a base line available in case of later complications. Haemoconcentration is usually evident and a leukocytosis or leukopaenia may be seen, with the latter more likely in cases of endotoxic shock. Biochemistry findings usually reflect hepatobiliary dysfunction, azotaemia and hypoproteinaemia. Electrolytes may be normal or there may be varying degrees of hypokalaemia. Arterial blood gas, if available, may demonstrate hypoxaemia and hypercapnia, and usually a metabolic acidosis. Measurement of clotting times (APTT and PT are available as a handheld unit) may demonstrate a hypercoagulable state or DIC. It has also been shown that increases in fibrin degradation products and APTT are predictive for gastric necrosis (sensitivity 86%, specificity 100%). Remember that APTT and PT will not be prolonged until 2/3 of clotting factors have been depleted. Plasma lactate concentration is an excellent indicator of outcome and gastric necrosis, and will hopefully become economically viable for many practices. There is a good correlation with outcome, with 99% of dogs with a plasma lactate concentration <6mmol/l surviving, compared with 58% of dogs with plasma lactate >6mmol/l. There is also a significant difference in plasma lactate concentrations in dogs with and without gastric necrosis (6.6 and 3.3 mmol/l respectively). More recently survival of 90% was shown in dogs if lactate was <9.0 mmol/l, whereas 54% survived if lactate was >9.0 mmol/l. This latter group was assessed further and survival was seen to be worse if, after initial fluid therapy, lactate was still >6.4 mmol/l (23% survival), there was an absolute change in lactate of <4.0 mmol/l (10% survival) or less than a 42% change in lactate concentration (15% survival). NB THIS DATA IS MORE RELEVANT TO GROUPS OF DOGS THAN INDIVIDUALS. I find lactate very useful in discussing prognosis with owners, but do not use it as a decision whether to treat or not as I have had good outcomes in individuals with concentrations of 8-12 mmol/l.



Radiography is used to differentiate gastric dilation and GDV. Signs to look for include a soft tissue line causing compartmentalisation of the stomach and the presence of a gas filled pylorus in an abnormal position. On a right lateral radiograph, the pylorus should be fluid filled and located in the cranio-ventral quadrant, and on the right on a VD view. In the typical 180-270º clockwise GDV the pylorus is gas filled, and is usually located in the cranio-dorsal abdomen dorsal to the body of the stomach on a right lateral radiograph. The duodenum and loops of small intestines may also be gas filled and located cranial and dorsal to the stomach. If in doubt, VD and left lateral radiographs can be taken. The presence of gas within the gastric wall is strongly suggestive of gastric necrosis, and free peritoneal gas is diagnostic of gastric rupture.

Medical stabilisation of GDV - Fluid Therapy

I will initiate medical stabilisation as soon as the animal enters the clinic, and the diagnostic tests described above are performed concurrently. It is important to stabilise the cardiovascular system to counteract the effects of hypovolaemia and hypoxaemia. I place two large bore catheters in the cephalic veins for intravenous fluid therapy. Catheters in the pelvic limbs are avoided as fluids placed in theses veins will not contribute to intravascular fluid volume until gastric decompression has been achieved. Later, after anaesthesia, I place a jugular (central) catheter which allows large volumes of fluid to be administered, blood samples to be collected, and is easy to manage for 5 days or more.

Initial fluid therapy begins with a balanced electrolyte/crystalloid solution. Shock fluid rates are given as boluses and fluid rates are continuously assessed using additional boluses as needed. The use of colloids will improve outcome, as they are better at improving cardiovascular volumes and effects last for longer than crystalloid therapy alone. Fluid rates are adjusted according to the dog's response. Blood products are occasionally required, including fresh whole blood if the PCV is <20-25% or plasma if the dog is hypoalbuminaemia or hypercoagulable.

Fluid therapy will usually correct acid-base abnormailites, and I have yet to have to supplement bicarbonate for the treatment of metabolic acidosis associated with GDV. Potassium chloride can be used as supplementation for dogs with hypokalaemia, but do not add maintenance volumes of KCl to shock rate fluids, as exceeding the maximum dose of 0.5mEq/kg/h may be fatal. It is safer to administer KCl through a separate fluid line, or delay administration if the serum potassium is only marginally low.

Most dogs are hypoxaemic, and administration of oxygen, either by mask or nasal prongs, will improve tissue oxygenation.

Medical Stabilisation of GDV - Gastric Decompression

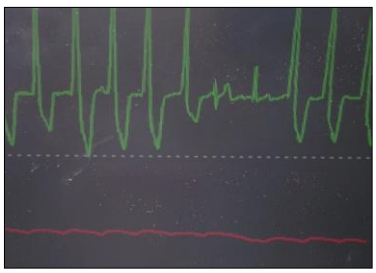
Gastric decompression is required as most of the local effects on the stomach occur secondary to dilation rather than volvulus. I find that gastric decompression via an orogastric tube less successful than described in the literature. Many dogs will need to be very collapsed or require some sedation or anaesthesia. A roll of elastoplast placed in the mouth allows the tube to be placed without the dog clamping its mouth shut. The tube should be gently advanced towards the stomach, allowing for swallowing when the tube passes the pharynx, and encountering some resistance at the gastro-oesophageal sphincter. Gastric contents in the tube confirms placement of the tube but are not an indicator that volvulus is not present. Gastric lavage can be performed as described later.

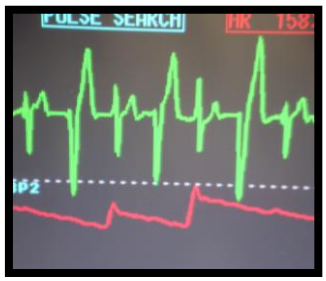
In my hands, percutaneous gastric decompression is adequate to minimise gastric distension prior to surgery. A 14g catheter can be placed into a tympanic area of the abdomen that is then compressed to force gas out. Be aware that gas is likely to accumulate again and further decompression may be required if there is a delay before surgical correction.

Temporary gastrostomy has been reported where definitive treatment is unavailable but is not recommended.

Arrhythmias

Cardiac arrhythmias occur in 40-50% individuals. One quarter of these are seen on initial presentation, the remainder developing over 12-36 hours due to reperfusion injury. Arrhythmias are unlikely to cause sudden death. Treatment was traditionally only given therefore if the arrhythmia was associated with hypotension/poor pulses, if there is a known pre-existing cardiac disease or if there are multiform VPCs or R-on-T phenomenon as the effect of arrhythmia on the cardiovascular system is minimal compared with hypovolaemia and hypotension. However, lidocaine has been shown to decrease other complications, such as development of acute kidney injury, and should be started before stomach decompression.





Initial treatment may consist of bolus of lidocaine followed by a constant rate infusion if the bolus was effective. If the animal is hypomagnesaemic, supplementation can be given, as low magnesium concentration is potentially arrhythmic in any patient. Alternatives if there is no response to lidocaine include beta blockers (e.g. esmolol) or sotolol.

Anaesthesia for GDV Surgery

Premedication with an opioid will provide analgesia without significantly affecting cardiovascular and respiratory functions. Following a period of oxygenation the dog can be anaesthetised with propofol. Dogs with poor cardiovascular status can be more safely induced with a combination of midazolam or diazepam and fentanyl given incrementally to effect. Anaesthesia is maintained with isoflurane or sevoflurane in oxygen. Nitrous oxide is avoided.

An arterial catheter can be placed if possible for arterial blood gas analysis and direct arterial blood pressure measurement when available. Otherwise indirect blood pressure is measured, as well as pulse oximetry, ECG etc. Placement of a urinary catheter allows measurement of urine output.

Fluid therapy is continued at 10-20ml/kg/h. The type of fluid and fluid rate are adjusted depending upon blood pressure, pulse quality and urine output.

If arterial blood pressure cannot be maintained above 60mmHg, drugs such as dopamine or dobutamine can be administered.

Broad-spectrum intravenous antibiotics are given at induction, to allow tissue levels to be well above the mean inhibitory concentrations of bacteria at the time of the first incision, and are given every 2 hours to maintain tissue level. I usually give a second-generation cephalosporin (eg cefuroxime, *Zinacef*) or potentiated amoxicillin (*Augmentin*) at 20mg/kg, and discontinue antibiotics within 24 hours unless there is obvious infection.

Opioids are administered intra-operatively for analgesia, but I initially avoid the use of NSAIDs when gastric viability has not been determined.

Surgery for GDV

A midline laparotomy from the xiphoid to midpoint between the umbilicus and pubis or to the pubis if needed allows thorough abdominal exploration. Balfour retractors are invaluable and the abdominal wall is protected with saline soaked laparotomy swabs. The falciform fat can be removed to improve exposure of the cranial abdomen, and is best achieved with electrocoagulation to control haemorrhage.

Upon entering the abdomen, the greater omentum is seen overlying the stomach in cases of GDV.

Surgery for GDV - Repositioning the stomach

I usually derotate the stomach before decompressing it, but some surgeons may find it easier to locate the abdominal oesophagus and guide a stomach tube through to decompress the stomach before attempting derotation. The stomach usually twists in a clockwise direction of 180-270º. I place one hand on the pylorus (or close to it) on the left side of the abdomen and lift it ventrally and to the right, whilst at the same time pushing the body of the stomach dorsally and to the left. The stomach should be examined to ensure that it lies in the correct anatomical position. In some situations, the stomach is torsed in an anti-clockwise direction, or the degree of rotation may be greater, but the technique described will allow most cases to be repositioned.

The stomach is decompressed by passage of a stomach tube. If necessary, due to a large volume of gastric contents, lavage with 7-10ml/kg of warm water or saline repeated 5-10 times until gastric contents are removed. The surgeon can assist by gently massaging the stomach and directing the tube. In extreme circumstances, when the stomach contains foreign material or excessive amounts of large food particles, gastrotomy may be required, but this is rare.

Surgery for GDV - Gastric necrosis

Initially the stomach may be hyperaemic and have suffered areas of petechial haemorrhage. The viability of the stomach and spleen should be assessed 10-15 minutes after derotation. Necrosis along the greater curvature and fundus is seen in 10% cases. The stomach may be obviously necrotic with a thin black-purple wall and a clear line of demarcation between viable and non-viable tissue, but signs of ischaemia are subtler. Assessments should be made of colour, thickness of tissue on palpation, vessel pulsation or bleeding of the cut surface. If in doubt, it is safer to assume tissue is necrotic and resect it, than leave necrotic tissue in situ. Partial gastric resection usually involves 20-50% of gastric tissue.

Various methods of treating gastric necrosis have been described including partial gastric resection (stapling or suturing) and gastric invagination. I prefer to resect the tissue to minimise the amount of necrotic tissue left in the stomach lumen, which may act as a nidus for bacterial proliferation or endotoxin production. The necrotic portion is grasped with Allis tissue forceps and elevated by an assistant, and the line for resection is assessed. It may be necessary to resect a large portion of fundus, including an area on the left dorso-lateral surface. The fundus should not be narrowed beyond the diameter of the abdominal oesophagus. Resection can be performed using stapling equipment, which is much quicker than suturing, but the disadvantage is the cost and availability. Following stapling, the staple line can be over sewn with a continuous inverting pattern (Cushing or Lembert) using 2 metric monofilament absorbable suture (eg PDS). For partial resection when stapling equipment is unavailable, stay sutures are placed 5cm away from necrotic tissue (2 or 3 metric Prolene) and used to elevate and manipulate the stomach. Blood vessels on the greater curvature may need to be ligated to allow access. After packing off the stomach from the abdomen with moist laparotomy swabs, the necrotic stomach is resected, and the stay sutures are used to avoid spilling gastric contents. Alternatively, intestinal forceps (eg Doyen) can be placed across the stomach. The stomach is sutured with a continuous appositional or inverting pattern, followed by a second continuous inverting pattern. Hand suturing for resection of stomach has a risk of spillage of gastric contents and is not advised if the stomach is distended with food or fluid, so other methods are used instead.

To invaginate the stomach, a continuous inverting suture is placed approximately 1cm away from the necrotic tissue, and the necrotic stomach is pushed intraluminally as the suture is placed. This suture line is reinforced with a second inverting layer wherever possible. One must take care to engage the sutures in the submucosa, and to avoid placing sutures in necrotic tissue. Up to 75% of the stomach has been invaginated without complications. Invagination is easy to perform by a single surgeon, whereas resection usually requires an assistant.

The spleen is assessed for torsion and the presence of avulsion or thrombosis of arteries and veins. The spleen should be removed if viability is in doubt.

Surgery for GDV - Gastropexy

There are many methods of performing gastropexy, all of which should form a permanent adhesion between the pyloric antrum and the abdominal wound. It is not acceptable to just suture them together without first creating an incision or wound in the tissues, as a firm adhesion will not form. A right-sided pexy is performed, as torsion is more likely to occur with a left pexy.

I usually perform a linear incisional gastropexy as I find it quick and simple to perform and it has been shown to form firm adhesions with minimal complications. The stomach is manipulated to find an area of the abdominal wall against which the stomach will sit comfortably. A 5cm incision is made through the peritoneum and transverse abdominal muscle approximately 6-8cm from the linea alba. A similar incision is made through the serosa and muscularis of the pyloric antrum and the edges are gently undermined. The mucosa should not be penetrated, but can be sutured if this inadvertently occurs. The cranial edge of the abdominal wall incision is sutured to the cranial incision in the stomach, in a simple continuous pattern using 2 or 3 metric monofilament absorbable or non-absorbable suture (eg PDS). The caudal edges of the wound are sutured similarly.

Some surgeons choose to perform a belt loop or circumcostal gastropexy, but they are technically more demanding. The circumcostal gastropexy provides the firmest adhesion initially, but it is not known how strong the adhesion needs to be to prevent volvulus.

I don't recommend the tube gastropexy, as recurrence of GDV is greater, it needs to be left in place for 14 days, and is associated with more complications. I also personally don't consider suturing the stomach into the midline laparotomy incision although it is reported – I worry about the risk of gastric penetration at subsequent laparotomy, and adhesions would make further surgery difficult, unlike more traditional pexies.

Finally, a complete abdominal exploration is performed and the abdomen is flushed with sterile saline and lavaged. The abdomen is closed with a simple continuous suture of 3.5 metric PDS (larger suture in giant dogs) in the linea alba and the remainder of the closure is routine.

Prophylactic gastropexy

Many owners of at risk large and giant breeds will consider prophylactic gastropexy. Dogs are considered high risk if they are an at risk breed, have a first degree relative with a history of GDV, have a previous history of gastric dilation, have a nervous temperament, and/or have a history of chronic torsion. It can be performed during neutering in females or as a separate laparotomy or laparoscopy procedure. In Great Danes, where the lifetime risk of GDV is 37% (more than any other breed), prophylactic gastropexy will decrease mortality rates by 30 times. The benefits are less dramatic in other breeds.

Post-Operative Care after GDV

Animals are closely monitored for at least 48 hours post-operatively. Vital parameters, including mucous membrane colour, CRT and pulse quality give a subjective indication of the effectiveness of fluid resuscitation. It is my experience that the majority of dogs are still hypovolaemic on recovery from anaesthesia, and careful attention to replacing fluid losses and maintaining intravascular volume and perfusion will give the best outcome. Fluids are continued until the animal is eating and drinking normally. Monitoring arterial blood pressure and central venous pressure allow fluid rates to be adjusted as necessary.

Colloids and blood products may continue to be useful in this period. The placement of a urinary catheter until the dog is able to walk outside comfortably allows measurement of urine output, which should be maintained above 1-2ml/kg/h. A continuous ECG should be in place until all arrhythmias have ceased but I only treat based on the criteria described before. Oxygen supplementation is useful in the early post-operative period when animals may still be hypoxaemia.

Water is offered when the animal is comfortably conscious followed by food. Smaller meals may be required if extensive gastric resection has been performed.

Complications after Surgery for GDV

- Megaoesophagus/atony – consider treating all GDV dogs with a pro-kinetic agent eg metoclopramide
- Post-operative dilation – seen in approximately 5% cases, may be related to underlying anatomical or physiological abnormalities. Treat with pro-kinetic drugs and decompression
- Aspiration pneumonia- usually associated with megaoesophagus. Needs aggressive treatment
- Gastric ulceration/perforation – poor prognosis, occurs 3-7 days post-op. minimise risk by careful evaluation of gastric viability
- Persistent hypovolaemia
- Hypokalaemia
- Sepsis/SIRS – may be related to reperfusion injury, pulmonary tissue is very sensitive to toxins
- DIC
- Hypoproteinaemia
- Intestinal intussusception
- Hepatopathies
- Haemorrhagic gastritis

Outcome after Surgery for GDV

Mortality rates in cases of gastric dilation alone are 0.9%. Overall mortality rates for GDV have been reported as 10-60%. A large university study, using standardised treatment protocols, demonstrated a mortality rate of 15% in dogs without gastric necrosis and 30% of dogs with necrosis requiring partial gastric resection. Other studies have shown mortality rates of 32% in dogs requiring splenectomy and 55% in those that also had a partial gastric resection. Outcomes are related to the primary and secondary pathophysiology events, and better outcomes in more recent studies probably reflect better intensive care and nursing practices. Factors that have a significant effect on short-term survival include recumbancy on presentation (mortality 4.4 times that of dogs who are walking), depression or coma (mortality 3 and 36 times that of alert dogs), pulse rate and quality/CRT/mucous membrane colour, pre-operative arrhythmia and gastric necrosis. There is a trend for increasing mortality, but no statistical difference, in older dogs and males. The length of time between the onset of clinical signs and presentation, and time between presentation and surgery is not related to outcome. I therefore aim to operate within 1-2 hours of presentation to allow a period of medical stabilisation.

Recurrence is between 70 and 80% if a gastropexy is not performed, compared with 5% in dogs who have a gastropexy. Recurrence is higher with tube gastropexy than the other methods. Long-term survival is longer in dogs that have had a gastropexy, and this is related to recurrence, as further episodes of GDV may be fatal. I therefore perform a gastropexy in all animals with GDV, and perform an elective gastropexy in animals that have had an episode of dilation alone. If the gastropexy cannot be performed at the time of derotation for technical reasons, an elective gastropexy should be performed when the dog is stable.

Recommendations to Prevent Recurrence of GDV

- Avoid raised feeding bowls
- Feed more than once daily
- Food particles should be >3cm, add canned food and table scraps
- Avoid exercise for 1-2 hours before and after eating
- Gastropexy in animals with just gastric dilation

Unusual Presentations of GDV

- Age – youngest reported case 7.5w
- Species – reports of cases in small breeds and cats
- Acute GDV may derotate and leave a partial gastric necrosis – do not dismiss clinical signs of the 'acute abdomen'
- Inflammatory bowel disease may be associated
- Gastric rupture – very poor prognosis
- Chronic volvulus
- Concurrent disease especially in older animals

Gastric Foreign Bodies

Gastric foreign bodies are rarely obstructive unless they become lodged at the pylorus. Associated clinical signs may be those of gastritis (vomiting) or the foreign body may be an incidental finding. Small objects can pass through the gastrointestinal tract without perforation.

The owner may see the animal eat the foreign body. Simple radiography, contrast radiography or ultrasound is diagnostic and 95% are seen on plain radiography. Some foreign bodies are only visualised on endoscopy.

Treatment of Gastric Foreign Bodies:

An emetic may be given to an animal so that it vomits the foreign body but this should only be performed if there is not risk of oesophageal obstruction or perforation.

Withdrawal by endoscopy may be possible for appropriate shaped foreign bodies using grasping forceps or a basket forceps and is successful in two-thirds of animals. Gastrotomy is indicated if endoscopy fails or for sharp or linear foreign bodies.

A cranial laparotomy is performed, the stomach is mobilised and elevated using clamps or stay sutures to minimize spillage and it is isolated in the abdomen by swabs. The gastrotomy is performed in the centre of the body of the stomach, between the greater and lesser curvatures in an avascular area. The incision is extended using Metzenbaum scissors. The foreign bodies are removed with forceps and care is taken to ensure that all have been removed. Suction may be necessary if the stomach is distended. The stomach is closed using a single or double layer suture. The rest of the gastrointestinal tract is checked for other foreign bodies or concurrent disease.

Gastric Ulceration and Rupture

Pathophysiology:

- Disruption of gastroduodenal mucosa
- ↓ gastric mucosal protective mechanisms

↓ prostaglandins
↓ blood flow
excess gastric acid secretion
↓ bicarbonate production
↓ gastric mucus production

Causes:

- Hepatic disease

↓hepatic degradation of gastrin and histamine - ↑ gastric acid secretion
Portal hypertension & gastric vessel thrombosis - ↓ mucosal blood flow

- Renal disease

↓ renal clearance of gastrin
Gastrin hypersecretion

- Gastric neoplasia
- NSAIDs (including COX-2 selective drugs)

Direct effect of weakly acidic drug on mucosa
Systemic inhibition of COX - ↓prostaglandin (gastroprotective) - ↓mucosal blood flow, ↓epithelial mucus, ↓bicarbonate secretion, ↓epithelial turnover
Especially pylorus
Worse if combine with corticosteroids

- Corticosteroids

Unknown mechanism - ↑ gastric haemorrhage, ↑ enteritis
Worse if combine with NSAIDs

Presentation & Diagnosis:

	Presentation	Diagnosis
Ulceration	• Vomiting, haematemesis (digested blood), melaena, anorexia • Anaemia only	• Endoscopy
Rupture	• Septic peritonitis (lecture 3)	• Peritonitis (imaging, cytology) • Site of rupture - laparotomy

Ulceration – Medical Treatment:

- If blood loss not severe and no risk of rupture
- Correct underlying cause
- Drugs to ↓ gastric acidity and promote mucosal protective mechanisms

Histamine- receptor antagonists (antacids) e.g. ranitidine, famotidine
Proton pump inhibitors e.g. omeprazole
Sucralfate – binds ulcers, stimulates local PG
Synthetic prostaglandin analogue e.g. misoprostol

Ulceration – Surgery:

- If marked haemorrhage or imminent rupture
- Remove ulcer (histopathology - ? tumour)

+/- concurrent endoscopy to find ulcers

+/- partial gastrectomy
+/- serosal patch if can't resect

- Correct underlying cause
- Start medical treatment

Perforation – Surgery:

- Suspected tumour

Resect – 2cm margins (if possible), +/- pylorectomy
Stage – LN & liver biopsy

- NSAID

Debride and close or resect