Introduction

Gastric dilation-volvulus (GDV) is an emergency condition involving abnormal, excessive gas distension and rotation of the stomach. The syndrome of GDV – both the gastric pathology and associated cardiovascular and other organ consequences - is a major medical and surgical emergency, involving significant stress to both the patient, requiring the clinician to possess a sound and thorough knowledge of the pathophysiology, patient problems and therapy involved with the condition.

The Pathology of GDV

The body of the stomach normally rests in the left cranial abdomen, with the pyloris and gastric outflow tract located in the right cranial abdomen.

When gastric dilatation and volvulus occur, the stomach essentially becomes filled with gas and twists on its longitudinal (cranio-caudal) axis. The most common type of torsion is a 180 degree clockwise torsion – although 270, 360, clockwise and anti-clockwise torsions can all occur. In a typical 180 degree clockwise torsion (viewed standing behind the patient), the pylorus moves from the right cranial abdomen, ventrally, around the fundus of the stomach and rests in the left abdomen, beside the cardia and abdominal oesophagus. Additionally, the greater curvature of the stomach is displaced further to the left, and dorsally, displacing the spleen caudally, ventrally and to the right – a movement that sometimes causes a tear in the greater omentum due to pressure, and a tear in the splenic artery. The rotation of the stomach obstructs both the oesophagus and the pylorus. The obstruction of both pyloris and cardia prevents gastric emptying and eructation, resulting in progressive expansion of the gastric volume. Continued gas production (from carbohydrate metabolism) and the production of normal gastric secretions further contribute to the volume of gas and fluid contained in the stomach.

A diagram showing the typical displacement of the pyloris in a 180 degree clockwise torsion, as viewed from the ventral aspect of the dog at surgery. De-rotation of the stomach is achieved by gently grasping the pyloris and bringing it ventrally and to the right side of the dog whilst simultaneously applying dorsal and left rotation to the body of the stomach.
Cardiovascular Pathophysiology

Gastric dilatation-volvulus syndrome can produce significant alterations to cardiovascular performance and tissue perfusion in affected patients. Experimental models of gastric dilatation-volvulus produce a 5-50% decrease in cardiac output, and a 50% decrease in coronary blood flow. This can result in cardiac ischaemia and myocardial necrosis, as well as poor global tissue perfusion.

In addition, GDV reduces surface oxygen tension in the duodenum (80%), jejunum (45%), liver (45%), and pancreas (45%). Following gastric de-rotation and resuscitation, the oxygen surface tension does return to normal in the tissues above, with the exception of the liver, which most likely suggests a high metabolic, and oxygen demand in hepatic tissue in the recovery phase of the illness.

The dominant mechanism for reduced tissue perfusion in the patient with GDV is compression of venous structures within the abdominal cavity, which reduces venous return to the heart, and reduces venous drainage from abdominal viscera.

- Direct compression of large veins and vascular beds is caused by distension of stomach, and increased intra-abdominal pressure
- Caudal vena cava compression decreases venous return to the heart. To some extent, this is partially compensated for by collateral circulation via vertebral venous sinuses, and the ayzygous vein – meaning some patients with GDV may not show significant symptoms of cardiovascular compromise early in their illness. However, most patients with GDV show clinical signs of hypovolaemic shock on presentation.
- Portal venous blood flow is reduced, and post-hepatic portal flow is also commonly decreased, leading to hepatic engorgement and distension. In addition, the portal hypertension reduces splanchnic blood flow, and causes congestion and hydrostatic pressure movement of fluid from the intravascular space to the interstitial space, and gastrointestinal tract lumen.

Decreased venous blood return ultimately leads to decreased cardiac output and hypotension. Ultimately, global tissue perfusion is compromised and further organ damage is likely without prompt intervention. Interestingly, cardiovascular abnormalities may persist following gastric decompression, due to release of vasoactive peptides and cytokines from previously ischaemic re-perfused tissues. Inflammatory and vasoactive cytokines reduce cardiac contractility, cardiac output, and alter vascular responsiveness and systemic vascular resistance. Increased sympathetic tone and adrenal axis stimulation that occurs in response to shock and decreased cardiac output in patients with GDV will restore some cardiac contractility, and increases heart rate, and systemic vascular resistance. However, vasoconstriction produced by sympa-ho-adrenal axis stimulation is uneven, and generally results in vasoconstriction in splanchnic tissues, such as the gut, pancreas, and kidneys, reducing oxygen delivery in these tissues. Additionally, increased sympathetic tone and catecholamine release caused by decreased blood pressure, acute gastric distension, increases cardiac contractility and raises myocardial oxygen consumption, in a heart suffering from decreased coronary blood flow resulting from decreased venous return, tachycardia and decreased systemic blood pressure. This produces myocardial ischaemia and necrosis.
Shock in Gastric Dilatation-Volvulus Syndrome

Shock may be broadly defined as inadequate tissue perfusion and oxygen delivery. In GDV, the cause of shock is multifactorial, and includes the following

- Hypovolaemia secondary to
  - Venous obstruction in the abdominal viscera, with subsequent translocation of intravascular fluid into interstitial spaces, the gastrointestinal tract, and abdominal cavity
  - Reduced venous return to the heart secondary to compression of the portal vein and caudal vena

- Septic/endotoxic shock results from portal venous occlusion, and loss of the integrity of the gastrointestinal mucosal barrier. Bacteria translocate from the gastrointestinal lumen into the circulation via the lymphatic channels, portal veins, and via the peritoneal surface (and then lymphatic vessels). Damage to the Kupfer cells in the liver favours dissemination of bacteria and their toxins into the systemic circulation. Endotoxin release, and tissue hypoxia lead to the following
  - Activation of the arachidonic cascade
  - Activation of complement
  - Activation of fibrinolytic pathways.
  - Activation of kallikrein-kinin cascade

  Activation of complement, fibrinolysis, and kinins produces sequelae, including hypercoagulation, release of inflammatory cytokines, and activation of coagulation. These substances perpetuate micro-vascular injury and loss of capillary wall integrity, and ongoing tissue necrosis.

- Cardiac arrhythmias develop as a result of cardiac ischaemia, elevated sympathetic tone, raised serum catecholamine levels, and electrolyte and acid base abnormalities.

Vascular congestion reduces gastrointestinal perfusion leading to hypoxia and tissue ischaemia. Tissue ischaemia results in anaerobic metabolism, and the production of lactic acid – frequently at a rate that exceeds clearance by the liver and kidneys. This can make blood lactate a useful measure of global tissue perfusion and may provide some indication of gastric wall health.

Respiratory Pathophysiology

In gastric dilatation-volvulus syndrome, the dilated stomach exerts force on the diaphragm, which increases inspiratory resistance, and reduces tidal volume – with most patients compensating for this with increased respiratory rate. Initially, this increase in respiratory rate may produce a respiratory alkalosis. However, as dilatation continues, increase respiratory rate and efforts may not be sufficient to meet minute volume required for aerobic metabolism in the body, leading to the development of a respiratory acidosis, and accelerated ischaemic tissue injury.

Perfusion to the stomach is compromised during torsion. The abnormal position of the stomach interrupts the delivery of oxygenated blood to gastric tissue. As a result, sections of the gastric wall become hypoxic and start to die. At surgery, these sections of the stomach often appear darkened and bruised, taking on a greyish-green to purple or black coloration. Devitalised sections must be carefully evaluated and possibly resected during corrective surgery; an event that potentiates further complications.
The Pathophysiology of Gastric Pathology

Gastric Ischemia and Oedema

Studies of experimentally induced GDV cause an almost immediate 92% decline in gastric surface oxygen tension suggestive of profound gastric ischaemia. The normal gastric mucosa, however, has a high metabolic demand, and requires 80% of gastric blood flow to sustain its metabolic requirements. These data suggest the stomach is likely to readily suffer ischaemia and resultant dysfunction in GDV. An understanding of the pathophysiology of gastric ischaemia can therefore assist in treatment decisions in GDV – prior to surgery, during surgery, and following surgery to correct the volvulus.

The origin of gastric ischemia in GDV includes the following:

- Physical compression of gastric capillaries and veins vessels due to high gastric intra-luminal pressure, results in gastric venous outflow obstruction, and gastric oedema – secondary to increased tissue hydrostatic pressure and reduced capillary blood flow
- Increased sympathetic tone results from hypovolaemia and acute stress, which produces gastric and intestinal vasoconstriction, and further compromises gastric blood flow

The origin of gastric mucosal oedema includes the following:

- Increased hydrostatic pressure in capillaries from venous outflow obstruction
- Gastric hypoxia and anaerobic tissue metabolism leads to gastric vascular endothelial cell damage, loss of capillary wall integrity and extravasation of intravascular fluid into tissue spaces.
- Coagulation and infarction of vascular beds may result due to vascular stasis and pooling of blood in microvasculature
- Tissue hypoxia and damage in the gastric tissues results in activation of an inflammatory response, margination of neutrophils, and extravasation of fluid can leukocytes into the interstitial tissue spaces
- Post-resuscitation - visceral hyper-perfusion in previously hypoxic tissues produces profound vasodilatation, and liberation of oxygen-free radicals into the tissues and systemic circulation.

Gastric acid and Gastric Ulceration

Despite the severity of damage that potentially occurs to the gastric wall, large gastric ulcers are not reported significantly in scientific literature following GDV. However, all patients suffering from GDV are at potential risk for gastric hyper-acidity and gastric ulceration due to loss of mucosal integrity, as well as gastric hypo-motility (gastroparesis), increasing the likelihood of gastric acid penetration to the submucosa.

Pathology of Disordered Gastric Motility

Most patients have disordered gastric motility following GDV, evidenced by symptoms of nausea, gastro-oesophageal reflux or vomiting. Disordered gastric motility following GDV usually results from damage to neuro-plexus in the gastric wall, and is associated with

- Intermittent gastric tachy-arrhythmias
- Decreased electromechanical coupling
- Decreased contractile amplitude

Rarely, gastric motility disorders may become permanent, due to gastric ischaemia, gastric over-distension, and myo-necrosis of the longitudinal muscle layer and/or the myenteric plexus.
Pathology of Abdominal Organs in GDV

Hepatic Pathology

Hepatic tissues suffer from reduced perfusion, venous outflow obstruction, and reduced tissue oxygen delivery in GDV. Hepatocyte damage is therefore likely to occur. Histopathological sections of liver tissue in GDV show congestion, neutrophil margination, moderate to severe hepatocyte necrosis, and haemorrhage in up to 70% of dogs following GDV. Hepatic necrosis is likely caused by hypoxia, endotoxin absorption, venous occlusion, and post-resuscitation reperfusion injury to hepatic vasculature and parenchyma. Clinically, this may be seen as reduced appetite, nausea, vomiting and mild to moderate increases in hepatic enzymes such as ALT and AST. Severe acute hepatic necrosis is an extremely uncommon event in GDV.

Pancreatic Pathology

Pancreatic tissue suffers from venous occlusion, reduced arterial blood supply and resultant capillary flow stasis during GDV. This results in pancreatic cellular damage. Most patients with GDV will have mild increases in amylase and lipase enzymes associated with pancreatic oedema, and up to 40% of dogs will have mild to moderate pancreatitis following surgery to correct GDV – the treatment of which consists of providing supportive care with intravenous fluid therapy, analgesics, antiemetic therapy and nutritional support.

Renal Pathology

The kidneys receive reduced arterial blood flow in GDV, often producing mild to moderate elevations in blood urea nitrogen (BUN) and creatinine. These elevations are pre-renal in most patients, normalizing following intravascular volume resuscitation.

Splenic Pathology

Splenic displacement in GDV is common, owing to the proximity of the spleen to the stomach through the gastro-splenic ligament. As the stomach rotates, it pulls the spleen dorsally and to the right. The splenic veins may become partially obstructed, leading to venous congestion, and micro-thrombosis within the splenic vasculature. Splenomegaly ensues.

Depending on the degree and (potentially) the duration of rotation, the splenic vessels may avulse/rupture and haemorrhage into the abdominal cavity which results in hypovolaemia.

If the spleen does not return to its normal size once the torsion has been corrected, is grossly engorged, if there are obvious infarcts, or if avulsed vessels are noted, or the spleen is torsed, the spleen is typically resected.

Intestinal Pathology

Decreased cardiac output, portal blood flow occlusion, gram negative bacterial toxins, and neurological mediated responses in the gut due to gastric distension all contribute to extensive oedema, neutrophil margination, and haemorrhage in the intestines, with extensive epithelial sloughing. Necrosis of the longitudinal muscle layer of the duodenum and jejunum (50% of dogs) may occur, which may contribute to the development of functional ileus in patients following GDV.
Pathology of DIC in GDV

Disseminated intravascular coagulopathy (DIC) is a common possible sequel to GDV, and results from the development of widespread vascular injury during the development of gastric distension and volvulus. Widespread vascular injury causes widespread activation of coagulation, which may lead to depletion of intravascular clotting factors, and the development of DIC. In addition, the presence of metabolic acidosis, and pancreatic enzyme activation of Hageman factor also contribute to a state of hypercoagulation, microthrombosis and DIC.

Aetiology of GDV

There is no conclusive evidence pertaining to a predisposing factor for GDV. However, certain characteristics appear to lend themselves to an increase in the likelihood of experiencing GDV.

Age

There is a greater tendency to experience GDV as age increases. The likelihood of GDV also increases after 4½ years of age.

Size

Larger breeds of dogs are more often affected than smaller. Deep-chested conformation seems to predispose large breed dogs even further. Among medium-sized dogs for example, the Shar Pei and Basset Hound are more frequently seen for GDV than other breeds of similar size.

Breed

There is a greater tendency for purebred dogs to experience GDV than mixed breed. It has been suggested that the likelihood in some breeds may approach a fourfold increase in incidence. Great Danes have the highest likelihood for developing GDV (42.4% chance); while German Shepherd Dogs, Standard Poodles and Weimaraners also exhibit a tendency towards the development of GDV. There have also been reported cases in Dachshunds and cats, so no breed or species can be assumed entirely without risk. Within breeds, it is possible to see an increased occurrence of GDV in individuals that exhibit exaggerated physical characteristics such as deeper chests or narrower abdomens.

Feeding Patterns

Feeding commercial pet foods once daily increases the likelihood of a dog to experiencing GDV. This tendency has been observed in various studies and theories offered in explanation are plentiful. It is interesting to note that domestic feeding patterns differ from feeding patterns observed in feral dogs. Recommendations based on this idea highlight the importance of feeding two or more smaller meals per day.

Although many large-breed dog owners feel that it is helpful to elevate their pet's food bowl, in fact, the opposite is true. Studies have shown that raising food bowls leads to an increased incidence of GDV, particularly for giant breeds.

Pre-/post-meal exercise has often been implicated as a cause of GDV. Again, no conclusive evidence is widely available, but pre- and post-meal exercise is often cited in history presented at time of admission.
Personality

A relationship between stress, anxiety and stress and incidence of GDV has been noted. For example, dogs in stressful situations such as boarding, unfamiliar surroundings, or ill health have exhibited a higher incidence of GDV.

Clinical Signs

Typically, dogs with GDV present to the clinic with a short history of having non-productive retching, or having frequent attempts to vomit. Occasionally, small amounts of food may be present in the vomitus, and the presence of food in vomitus does not preclude the presence of GDV. In addition, some dogs may have an episode of abdominal discomfort, abdominal bloating and ineffectual vomiting while at home, but may present with no obvious symptoms.

Common clinical signs observed on presentation include abdominal tympani, excessive salivation, cranial abdominal discomfort, abnormal splenic position on abdominal palpation, and clinical signs of sympathetic and adrenal gland stimulation such as elevated heart rate, poor femoral pulses, panting etc.

It is advised to take a right lateral abdominal radiograph in patients suspected of having GDV that have a suspicious history, but having equivocal clinical signs.

A typical appearance of a 180 degree clockwise gastric torsion in a right lateral radiograph.
Notice the compartmentalisation of the stomach, and the dorsal displacement of the duodenum in this view.
Radiographic Findings

The right lateral abdominal view provides the radiographic view of choice in patients with suspected GDV. The presence of fold that appears to compartmentalize the stomach, and a gas filled pylorus dorsal to the fundus are diagnostic for the disease. The presence of free abdominal gas suggests rupture of viscous and/or oesophageal rupture.

![Radiographic Image]

*A typical appearance of a 180 degree clockwise gastric torsion in a right lateral radiograph. Notice the compartmentalisation of the stomach, and the dorsal displacement of the duodenum in this view*

Clinical Pathology

Patients with GDV should have a minimum clinical pathology database performed in order to detect potentially life-threatening disorders such as coagulopathy prior to surgical intervention. In general, the database should include the following:

- Packed cell volume and total plasma protein evaluation
- Platelet estimation
- Activated clotting time (or PT/APTT)
- Blood glucose
- Serum electrolyte status

If the patient has a history of prior or concurrent illness that may influence the potential for survival in surgery or the post-operative period, the database should be extended to include a complete blood count/haematology and routine serum biochemistry and urinalysis.

Frequently, minor deviations from normal laboratory values are encountered in patients with GDV, which can occur for a variety of reasons, the most common of which are described below:
**Packed Cell Volume**

An initial increase in packed cell volume (PCV) may be noted early in GDV secondary to splenic contraction, and a relative loss of plasma into the interstitial spaces. Following intravenous fluid therapy with crystalloids +/- synthetic colloids, the PCV, total plasma protein and serum albumin concentration will fall. Post-operatively, the PCV and total plasma protein may fall secondary to continued fluid loss from plasma into third spaces, the gastrointestinal tract and tissue interstitial spaces.

**Potassium**

Potassium concentrations are often-times normal at the time of case presentation, but may vary as follows:

- Potassium levels may increase early during the course of GDV, and may even dramatically elevate during decompression due to release of potassium from damaged cells into circulation.
- Hypokalemia is a frequent finding post-operatively, and is associated with loss of potassium in vomitus, sequestration within the stomach and intestinal tract, due to diuresis following fluid therapy to manage shock and hypovolaemia, and hypovolaemia induced hyper-aldosteronism.

**Phosphorus**

Phosphorus concentrations may rise progressively due to release from the intracellular environment during tissue cell damage and the breakdown of ATP during hypoxia. Following emergency stabilisation, serum phosphorus concentrations are usually normal.

**Sodium, Chloride, Calcium**

Remaining electrolytes usually show little change in GDV patients. Alterations in serum concentrations are occasionally seen in patients with persistent regurgitation, vomiting or diarrhoea.

**Glucose**

Glucose concentrations are not uncommonly elevated early in GDV, and are most often associated with stress. Following acute resuscitation, glucose levels usually remain within the normal range, although in patients with severe sepsis, hypoglycaemia or hyperglycaemia may be observed.

**Creatinine Phosphokinase**

Creatinine phosphokinase is occasionally mild-to-moderately elevated secondary to ischaemic myopathy in the caudal skeletal muscles. Patients of high bodyweight who remain recumbent may also show moderate elevations in creatinine phosphokinase.

**Blood Gas and Acid-Base Status**

Patients with GDV frequently have one or more derangements of blood gas or acid-base status, secondary to respiratory compromise, reduced cardiac output and altered microvascular blood flow in abdominal organs. Common types of acid-base abnormality in GDV patients include:

- Respiratory alkalosis: secondary to tachypnea
- Respiratory acidosis: secondary to inadequate minute volume
- Metabolic acidosis: secondary to lactic acidosis
- Metabolic alkalosis: secondary to sustained vomiting of gastric acid

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Treatment of Gastric Dilatation-Volvulus

Successful treatment of gastric dilatation-volvulus syndrome requires the clinician be cognizant of patient problems, and potential complications, so that appropriate therapy may be implemented in a timely manner. Adopting a predictive approach to problem identification and management has been shown to reduce mortality and morbidity associated with GDV syndrome.

The potential problem list that exists for patients with GDV is as follows:

- Hypovolaemic shock
- Septic shock
- Cardiogenic shock
- DIC
- Cardiac arrhythmias
- Respiratory compromise
- Gastric distension and torsion
- Gastric wall ischaemia and necrosis
- Disordered gastric motility
- Pancreatitis
- Ileus
- Sepsis and SIRS
- Splenic avulsion, torsion, engorgement
- Abdominal haemorrhage
- Post-operative malnutrition
- Hypoproteinaemia

Because of the range of symptoms, presenting patient status, and potential complications, it is impossible to offer a “one size fits all” approach to the management of the patient with GDV. However, algorithmic approaches to complex diseases such as GDV can direct the clinician through management of the disease, drawing attention to key aspects of patient management so that important diagnostic and therapeutic steps are not overlooked. What follows is an outline of a clinical approach to the patient with GDV...
A Suggested Algorithm for the Management of Gastric Dilatation-Volvulus

1. Assess
   a. Heart rate
   b. Pulse rate
   c. Cardiac rhythm
   d. Temperature
   e. Capillary refill time
   f. Abdominal cavity

2. Needle decompress the stomach with one or two 18G needles placed in the stomach percutaneously at the area of maximum tympani to improve patient comfort while the condition is discussed with the owner (disease, treatment, prognosis, and costs)

3. Place a 20 or 18G IV catheter in a peripheral vein in the forelimb (not hindlimb). Obtain blood for the following
   a. PCV/TP
   b. ACT – a prolonged ACT on presentation is associated with a greater complication rate. However, patients with a coagulopathy on presentation still have good survivability if managed appropriately
   c. Lactate – In one study, blood lactate <6 mmol/L was associated with improved outcome (up to 99% survival rate). Patients with gastric necrosis have been found to have a mean blood lactate level of 6.6 mmol/L. Recent studies suggest that absolute values of blood lactate are less important that the trend in blood lactate – meaning that a blood lactate measurement that is falling is correlated with improving tissue perfusion and an improved prognosis over a blood lactate concentration that rises or remains static despite aggressive resuscitation efforts.

4. Fluid therapy – begin an infusion of lactated Ringers’ solution (LRS) at 10 ml/kg/hr. If the patient is showing clinical signs of shock, such as elevated heart rate, poor pulses, depressed mentation or altered mucous membrane characteristics, begin intravenous LRS at a rate of 40-60 ml/kg/hr, given in 10 minute boluses of 10 ml/kg. The use of a synthetic colloid such as hydroxy-ethyl starch (HES) at 5 ml/kg given IV over 10 minutes early in fluid resuscitation efforts is associated with a prolonged duration of action of any crystalloid fluid therapy used and may improve cardiovascular stability. Hypertonic saline (HTS) may be used in place of the initial bolus of LRS. HTS may be given as a 7% solution at a dose rate of 3-5 ml/kg IV over 10 minutes, either alone or in combination with a 5 ml/kg bolus of HES, and followed with LRS at 10 ml/kg/hr.

5. Analgesia – Opiate analgesia administered by constant rate infusion is preferred for management of patients with GDV.
   a. Fentanyl is initially administered as a bolus of 4-8 µg/kg IV and followed immediately by a constant rate infusion at 4-8 µg/kg/hr is the initial analgesic of choice, as it provides rapid analgesia, and reduce doses of induction and maintenance anesthetic agents.
   b. Morphine used at 0.1-0.2 mg/kg may be given by slow IV injection if fentanyl. Morphine can be administered following initial IV dose at 0.1-0.3 mg/kg/hr constant rate infusion
   c. Ketamine may be administered at 0.5-1.2 mg/kg/hr following a 0.30-0.5 mg/kg IV bolus and administered in addition to fentanyl or morphine in patients showing continued discomfort despite opiate analgesia
   d. Lidocaine may be administered as a bolus of 1 mg/kg slow IV followed by a constant rate infusion of 1.8-4.8 mg/kg/hr in addition to fentanyl or morphine +/- ketamine. Lidocaine administration raised the threshold for ventricular arrhythmias common in GDV.

6. Anaesthesia – anaesthesia should be induced with an induction drug familiar to the clinician. Avoid hypotensive agents such as alpha-2 agonists, propofol and mask inhalant inductions. Acceptable choices for anaesthetic induction include diazepam-ketamine combination or alfaxalone. Anaesthesia
should be maintained with isoflurane at the lowest achievable concentration, in combination with fentanyl (or morphine) +/- ketamine +/- lidocaine constant rate infusion. Should an increase in anaesthetic depth be desired, increasing the CRI of analgesia, or administering intravenous induction agent is preferable to increasing isoflurane inhalant concentration, as this produces less vasodilatation and hypotension.

7. Gastric Decompression – there is open debate amongst specialist emergency clinicians over the timing of gastric decompression, with some preferring oro-gastric decompression prior to surgery and some preferring to delay this until gastric de-rotation has occurred. The author's preference is to attempt oro-gastric intubation and gastric lavage prior to surgery whilst the abdomen is clipped and prepped for surgery. Following anesthetic induction, pass a large bore stomach tube (2-inch diameter) to allow decompression of the stomach. Following decompression, perform a thorough gastric lavage with warm water until the stomach is empty. This procedure may take up to 30 minutes to complete, and has the advantage that when the stomach is investigated at surgery, any necrotic areas of the stomach are easily identified, as gastric decompression and lavage will allow return of blood to viable areas of the stomach prior to surgery.

If there is difficulty passing a large bore stomach tube initially, passing a smaller diameter tube may allow removal of the gastric gas-cap, and will facilitate passing of a larger tube for gastric lavage. In addition, re-positioning of the patient from right to left lateral recumbency, or relieving gastric distension using 1-2 18G hypodermic needles may facilitate subsequent passage of a stomach tube.

8. Surgery – The immediate aim of surgery is to return the stomach to its normal position and evaluate it and the spleen for signs of irreversible vascular compromise. If present, necrotic portions of stomach and spleen should be removed. The stomach should be emptied completely. Finally, a gastropexy should be performed in an attempt to prevent recurrence of the volvulus. A detailed description of the surgical process is presented below.

**Surgical Management of GDV**

**Access and Initial Assessment**

1. Following routine aseptic preparation a cranial ventral midline laparotomy is performed by making an incision from the xiphoid to the caudal umbilicus.
2. Remove the falciform ligament to facilitate visualisation of the anterior abdomen, stomach, liver and pancreas.
3. Following removal of the falciform ligament, the stomach is usually immediately visible and covered by greater omentum when a clockwise volvulus of 180–270 degrees has occurred. Gastric decompression performed prior to or at this stage will help subsequent manipulation and relocation of the stomach.
   a. Gastric decompression can be achieved intra-operatively by needle gastrocentesis, if the stomach is still tightly distended. Alternatively, for a less distended stomach, a non-sterile assistant, with the intraoperative guidance of the surgeon, can gently place an orogastric tube.
4. After gastric decompression, the pylorus is identified in the anterior left quadrant of the abdomen and grasped gently with the surgeons' right hand. If the gastric rotation is in a clockwise direction, downward pressure on the right side of the visible portion of the stomach along with gentle traction on the pylorus will aid counter-clockwise rotation of the stomach to its anatomically normal position. The spleen should follow passively.
5. Following gastric de-rotation, a systematic evaluation of the abdomen should be performed.
   a. Any abdominal fluid should be aspirated with the volume and packed cell volume of removed fluid noted. Haemo-peritoneum often results from avulsion of the short gastric branches of the splenic arteries. Active sites of haemorrhage should be identified and ligated as early in the exploration as is reasonable.
b. Careful inspection of the stomach and spleen should then be carried out, followed by a complete visual assessment of all other abdominal organs.

**Assessment of the Gastric Wall**

The junction between the fundus and body along the greater curvature of the stomach is the most common site of gastric necrosis following gastric dilatation-volvulus (GDV). Evaluation of tissue blood flow remains subjective, and assessment immediately following gastric de-rotation may differ from an assessment made 10 minutes later due to reperfusion of tissues following decompression. Visual evaluation of gastric wall colour, digital palpation of splenic and gastric vessels for arterial pulsation, digital evaluation of gastric wall thickness and the presence or absence of arterial bleeding from the cut gastric wall are all useful assessments. Absence of pulsation in the gastric and splenic vessels, serosal surface tearing, grey/green or black discoloration, very ‘thin’ gastric wall or absence of bleeding from the cut edge of the stomach are all indications of vascular compromise that will ultimately lead to gastric wall necrosis.

Viability of the stomach wall can be theoretically assessed by pulse oximetry, Doppler blood flow measurements and fluorescein dye evaluation. It is important to note that these tests are only assessing vascularity and not mucosal integrity. During surgery the visual inspection and palpation of the stomach wall are still standard and used to decide if resection of parts of the stomach is indicated.

**Serosal** colour can be black, blue, grey or brick red. Usually if the serosal colour stays dark (black, blue or grey) after de-rotation and decompression a resection of this area should be performed. If small cuts into the muscularis of these regions do not actively bleed, then partial gastric wall resection should be performed. Vascular patency and gastric wall thickness are other criteria used frequently.

**Mucosal** colour should not be used as an indication for resection. In GDV patients it is not uncommon to see black gastric mucosa. Mucosal haemorrhage or vascular obstruction is not correlated with survival of gastric tissue - however they may give indication the patient may develop gastric ulceration.

Once viability of a region is questioned and gastric resection becomes an option, the surgeon needs to decide if the affected region can be resected easily. The fundus region is usually the affected area in GDV patients. This region can be easily resected by ligating the vessels from the gastro-epiploic artery and the short gastric vessels as needed. Resection is performed in viable bleeding areas and a two-layer closure is performed. Alternatively a stapling device can be used to resect the non-viable stomach. If the necrotized area includes parts of the cardia, the pylorus or is so large that the lesser curvature is compromised, then resection becomes more than a challenge. Prognosis in these circumstances is poor, and euthanasia may be suggested to the owner.

**Partial Gastric Resection**

In patients that require partial gastric wall resection, a full-thickness gastric wall resection is carried out to achieve active bleeding at cut edges of the resection, to ensure healing without further necrosis. Closure of the stomach following partial resection should be in two (or occasionally) three layers. A simple continuous
appositional suture pattern in the submucosa is followed by a continuous appositional pattern in the muscularis and serosa is generally performed. Oversewing the suture line with a continuous or interrupted inverting pattern such as a Cushing or Lambert can reinforce this closure. Absorbable monofilament sutures such as Polydioxanone (PDS-Ethicon) and polyglyconate (Maxon-Davis and Geck) are suitable suture materials. Alternatively, surgical stapling devices can be used to perform partial gastric resection. The use of gastrointestinal anastomosis instrument (GIA50, US Surgical) has been described for this purpose; however, some authors prefer to use a thoraco-abdominal stapler (TA-90, US Surgical) with a 4.8 mm (green) staple cartridge. Again, this closure should be reinforced using a continuous or interrupted Cushing or Lambert inverting pattern to over-sew the staple line.

Occasionally the cardia or the abdominal oesophagus will become necrotic secondary to longstanding or severe twisting causing damage to the left gastric arterial blood supply to the stomach. This area should be examined carefully. Resection of the abdominal oesophagus and gastric cardia is technically demanding, and the outcome following such a resection, even in healthy animals, is unpredictable. Since necrosis at this site is usually seen in animals that are already severely compromised, the prognosis for recovery is poor.

Invagination of necrotic areas should be avoided as the invaginated are may act as a focus for inflammation, ongoing release of inflammatory mediators, gastric ulceration and bleeding and sepsis.

**The Spleen**

The decision to perform splenectomy in the patient with GDV is controversial. Splenic engorgement is found in association with splenic venous compression, reduced splenic perfusion and splenic parenchymal cell damage. Splenectomy performed early in surgical management of GDV reduces the quantity of bi-products of splenic hypo-perfusion released into systemic circulation. A review of 102 cases of GDV found no increase in hospitalization time or mortality rate when splenectomy was performed in patients with splenic engorgement compared to patients with GDV and normal splenic size that did not have splenectomy performed. Patients with gross engorgement of the spleen that did not have splenectomy had a higher incidence of ventricular arrhythmias, nausea and prolonged hospital stays compared with splenecomised patients. Regardless, the spleen should be evaluated thoroughly. If the spleen is torsed – DO NOT UNTWIST IT – remove the spleen if it is torsed or has poor or compromised vascular supply, and consider removal if gross splenomegaly is present.

**The Gastropexy**

There are a number of gastropexy techniques described in the literature. The author’s preferred technique is the incisional gastropexy. Other techniques such as belt loop gastropexy and circum-costal gastropexy are more time-consuming, and technically more difficult, but offer little or no advantage over incisional gastropexy in terms of stability or longevity of the pexy.
Post-Surgical Care

Following surgical correction of GDV, the patient must be supported appropriately in order to achieve a successful recovery. Failure to identify and act on potential complications can affect the patients’ recovery. The key points in post-operative care are outlined below:

1. Fluid therapy – continue infusion of LRS at 1.5-2 x maintenance rates (3.5-5 ml/kg/hr) for the first 12-24 hours following surgery. Infusion with synthetic colloids such as hydroxy-ethyl starch (HES) at 20 ml/kg/day minimizes fluid loss from the intravascular space; maintains colloid oncotic pressure and improves tissue oxygen delivery during recovery. Fluid type should be changed to a maintenance fluid.
type (+/- synthetic colloid) following 24 hours of fluid therapy to ensure normal electrolyte balance is maintained.

2. Analgesia – fentanyl or morphine constant rate infusions should be continued for 24-35-hours following surgery (+/- ketamine +/- lidocaine) following which a transition to transdermal fentanyl or oral opiate such as buprenorphine or tramadol may be considered. Patients with good analgesia have reduced sympathetic tone, and improved blood supply to the gastro-intestinal tract i.e. they recover faster.

3. Gastric motility – begin a CRI of metoclopramide following a subcutaneous bolus dose of 0.5-1.0 mg/kg to assist in controlling nausea and vomiting. Metoclopramide increases gastric peristalsis, reduces pyloric tone and increases lower oesophageal tone, making it ideal for the management of the post-surgical GDV patient. In addition, a nasogastric tube may be placed to facilitate suction of gastric contents every 1-3 hours, as this will reduce nausea, retching and the risk of regurgitation and potential aspiration of gastric fluid. If required, anti-emetics such as maropitant citrate or dolasetron may be administered to control nausea. However, metoclopramide CRI and naso-gastric suctioning usually prove sufficient in most patients.

4. Electrolyte status – hypokalaemia is common in patients following GDV, and contributes to the presence of cardiac arrhythmias post-surgery, and disordered gastric motility and gastric and intestinal atony. Electrolytes should be monitored every 12-24 hours if available, and corrected as indicated. If electrolyte analysis is not readily available, maintenance intravenous fluids should be supplemented with 20-30 mEq KCL for each litre of maintenance intravenous fluids.

5. PCV/TP - monitor PCV/TP and ACT every 12 hours. Hypoproteinaemia should be managed with an infusion of HES or Pentaspan at 20 ml/kg/day +/- plasma transfusion at 10-20 ml/kg as required. A coagulopathy indicated by prolonged ACT should be treated with whole blood or fresh frozen plasma at an initial dose of 10 ml/kg IV over 4 hours.

6. Gastric mucosal health – many cases of GDV do not require administration of gastric protectants once torsion is corrected. However, ranitidine may be used at a dose of 0.5 mg/kg IV q 8 hrs to reduce gastric acid secretion and reduce the likelihood of gastric ulceration. Begin oral administration of lectade or gastrolyte at 1 ml/kg PO q 4 hrs as soon as the patient is able to tolerate oral medications to aid in maintaining enterocyte health and gastric motility. Begin feeding the patient a low-fat diet such as Hill's i/d or boiled chicken once the patient is able to tolerate oral liquids.

7. Cardiac arrhythmias – ventricular tachyarrhythmias are common in patients following GDV. However, only clinically significant arrhythmias require treatment with anti-arrhythmic medications. Tachyarrhythmia’s requiring treatment include those that cause a reduction in cardiac output significant to the patient (i.e. signs of weakness, panting, collapse, poor pulse quality, and evidence of poor perfusion etc.) Prior to administration of anti-arrhythmic medications, ensure the patient is euvoaemic (has adequate fluid therapy), assess pain and manage appropriately – this may involve an alteration of analgesic drug dose or medication; check electrolyte levels and treat accordingly (especially hypokalaemia and hypo-magnesaemia), and characterize the arrhythmia. Guidelines for ventricular arrhythmias that require treatment include: sustained heart rate above 140/min; systolic blood pressure less than 100 mm Hg, pre-existing or concurrent cardiac disease such as dilative cardiomyopathy, or when R- on T- phenomenon is observed. Treatment is usually commenced with lignocaine at 2 mg/kg IV given over 2 minutes, and repeated every 5-10 minutes up to a cumulative total dose of 8 mg/kg. A constant rate infusion may be required.

8. Antibiotic therapy – is usually required due to the compromise of the gastric and intestinal mucosa during the gastric dilatation. Cephalexin or cephazolin, or amoxicillin given at 22 mg/kg IV q 8 hrs are good initial choices that should continue for 5 days following surgery.
Conclusion

In summary, the patient with GDV can be successfully managed in the majority of cases. Mortality rates vary depending on a number of factors. Patients with gastric necrosis, requiring gastrectomy, splenectomy, and patients with cardiac arrhythmias prior to surgery may have increased risk of mortality – but not in all cases. However, appropriate and timely management of pre-surgical and post-surgical abnormalities can greatly minimize the risk of mortality. In all cases, adherence to the basics of fluid therapy, timely gastric decompression, support of gut health, and cardiovascular and clotting abnormalities is essential to improving patient outcome.

References