GDV: **EMERGENCY AND CRITICAL CARE**
THE TOUGH ONES
Elke Rudloff, DVM, DACVECC

Gastric dilatation-volvulus syndrome (GDV) is a combination of intragastric accumulation of gas and fluid and a malignant rotation of the stomach along its long axis. This results in a variety of life-threatening conditions including circulatory shock and systemic inflammatory response syndrome (SIRS). Untreated it leads to death.

Although most commonly seen in large breed dogs, GDV can occur in any breed or species of animal. Characteristics that are associated with GDV include having a first-degree relative that suffered GDV and owners perception of high-level stress in the dog. Additional factors thought to influence the formation of GDV include lean body condition, increasing age, large drops in environmental temperature over a short period of time, feeding single large meals, overeating, small kibble size, elevated feeding bowls, postprandial exercise, anesthesia, and aerophagia. The addition of table food or canned food to the diet of large and giant breed dogs has been associated with a decreased incidence of GDV. Reduced incidence is seen with dogs getting moderate exercise and receiving egg or fish supplements. Some findings contradict others.

The mechanism that initiates a GDV episode remains unknown. The gas produced is primarily carbon dioxide with some hydrogen, likely from bacterial fermentation. The source of bacteria would be from ingested food or duodenal reflux. Severe gastric distention manifests as visible abdominal distension, restlessness, excessive salivation and swallowing, and nonproductive retching. Intraperitoneal pressure elevation, obstruction of the compliant caudal vena cava, decreased venous return and cardiac output, hypoxemia, as well as microcirculatory injury and maldistribution of blood flow result in signs of circulatory shock. Cytokine production, cellular dysfunction, and reperfusion injury following resuscitation can induce a SIRS and disseminated intravascular coagulation. These consequences can culminate in multiple organ failure and death.

Upper airway obstruction can result from regurgitation and aspiration of swallowed saliva. Hypoxemia, electrolyte disturbances, acid/base imbalances, and release of myocardial depressant factors can exacerbate myocardial dysrhythmias and decrease cardiac output. Cytokine release from traumatized and suffocated tissues can induce the SIRS. Circulatory collapse can induce disseminated intravascular coagulation (DIC). Bacterial and endotoxin translocation through disabled gastrointestinal mucosal barrier into the bloodstream is expected. Because the liver may be compromised from venous congestion and circulatory shock, bacteria may not be removed from the portal system resulting in septicemia.

Cells that have been deprived of oxygen develop an elevation of cytosolic calcium and hypoxanthine. Reperfusion of hypoxic cells with oxygen during fluid resuscitation and gastric decompression fuels oxygen derived toxic radical production, overwhelming the protective antioxidative enzymes. Oxygen-derived free-radical species initiate lipoperoxidation of cell and organelle membranes, destroy enzyme systems, and cleave DNA strands resulting in cellular dysfunction and apoptosis.

These consequences can culminate in multiple organ failure. The key to successful treatment of complications associated with GDV is the anticipation of these complications and appropriate prophylaxis, rather than the one-at-a-time reaction to complications after they have manifested. As resuscitative efforts are initiated, prefluid blood samples are collected for immediate evaluation of the packed cell volume (PCV), total solids (TS)**,** electrolyte, venous blood gas, lactate, azostick and dextrostick, platelet count, activated clotting time, and samples are saved for coagulation profile, serum biochemical profile, complete blood count and urinalysis. Prefluid values provide a baseline from which subsequent values are compared to and monitored. In addition, any significant abnormalities are addressed prior to surgical intervention. It has been reported that initial lactate levels >6 mmol/L in GDV are associated with increased incidence of gastric necrosis and outcome, but these results are not repeatable and a single lactate level should not be used to predict outcome. Serial blood lactate or myoglobin may provide more accurate information when predicting outcome in dogs with GDV. Any clinical evidence of coagulation abnormalities in addition to laboratory abnormalities requires appropriate treatment prior to surgery, such as frozen plasma if DIC or coagulation factor defect is suspected.

Resuscitation
Immediate fluid resuscitation using multiple large-bore peripheral catheters is performed in stages to end-point parameters of improved perfusion, normal heart rate, and normal blood pressure. Isotonic replacement crystalloids

(such as Plasmalyte-A®, Normosol-R®) are always administered in incremental doses of 20–30 ml/kg. Synthetic colloids (hydroxyethylstarch [HES]) are administered at incremental doses of 10–20 ml/kg (up to 20–50 ml/kg). These solutions preserve colloid osmotic pressure during fluid resuscitation. In addition, Oxyglobin™ carries oxygen to tissues with compromised blood flow and has a vasoconstricting effect which might be desired during SIRS states, such as occurs in the GDV patient. If crystalloids are used alone, the bolus doses are increased to 20–50 ml/kg.

The rapidly deteriorating hypovolemic patient without significant hemorrhage may benefit from the infusion of hypertonic saline (4 ml/kg 7% solution) with synthetic colloid and buffered isotonic crystalloids in an effort to augment exogenous fluid infusion with interstitial fluid redistribution. Large volume resuscitation with any fluid product can induce a dilutional coagulopathy and plasma transfusions are often required to prevent hemorrhagic tendencies.

If significant hemorrhage is present, resuscitation to a low-normal blood pressure may be warranted until surgical exploration. This entails careful endpoint resuscitation techniques using crystalloids and colloids to a MAP of approximately 60 mmHg (systolic arterial pressure around 80 mmHg). The goal is to initiate some reperfusion without disturbing any clots that have formed until hemostasis is achieved surgically. A constant rate infusion (CRI) of HES can be administered after resuscitation of the hypotensive animal at a rate of 0.8 ml/kg/hr to help maintain blood pressure until cardiovascular stability is maintained. If the blood pressure is not responsive to fluid resuscitation, dopamine infusion (5–15 mcg/kg/min) may be required and underlying causes of nonresponsive shock investigated. Dobutamine infusion (5–10 mcg/kg/min) may also be necessary if cardiomyopathy is suspected.

Normal to increased blood pressure is evaluated with respect to intravascular volume status. Adequate or increased blood pressure may be a result of a compensatory response to hypovolemia, and aggressive fluid resuscitation is still indicated, as described above.

GDV is a presumably very painful experience. Immediate administration of injectable opioids generally provides some relief and acts as a premedication in anticipation of surgery. Oxymorphone (0.1 mg/kg), hydromorphone (0.1–0.4 mg/kg), methadone (0.1–0.2 mg/kg IV), or fentanyl (5 mcg/kg) can be administered IV, or morphine (0.5–1 mg/kg) can be administered intramuscularly (IM) or by slow IV injection to avoid hypotension. Opioid-induced nausea is rarely encountered in the critically ill animal. A multimodal continuous analgesic approach can be instituted by combining morphine (60 mg) or fentanyl (1.5 mg) with lidocaine (300mg) and ketamine (60 mg) placed in 1L isotonic crystalloid. If analgesic medication is due, a rapid infusion of 2 ml/kg can be administered followed by a continuous rate infusion at 2–4 ml/kg/h. Intraoperatively, the infusion can reduce the dose of vasodilating inhalant anesthetics. Early use of a bolus followed by a CRI infusion of lidocaine has been associated in one study with reduced incidence of acute kidney injury and the incidence of dysrhythmias. The partial agonists/antagonists such as butorphanol and buprenorphine may not provide adequate analgesia for this type of injury and impending surgical intervention. Nonsteroidal anti-inflammatory agents (NSAIAs) should not be used since gastric blood flow is compromised in the GDV patient, and use of NSAIA can result in ulceration.

Any ausculted or ECG dysrhythmia should be treated with supplemental oxygen therapy, fluid resuscitation, and analgesia therapy. Any acid-base and electrolyte (potassium, calcium, and magnesium) abnormalities should be corrected. When improvement of perfusion does not occur, antidysrhythmic medication is administered. The most common dysrhythmia treated in the GDV is a ventricular tachycardia, and lidocaine is administered IV 2–4 mg/kg slow bolus. If this improves the rhythm, then a 50 mcg/kg/min CRI is started. Continuous ECG monitoring is required. Underlying cardiomyopathy is a consideration and an echocardiogram is recommended, time permitted.

Preoperative broad-spectrum antibiotics such as a first generation cephalosporin or ampicillin (20 mg/kg IV) should be administered as the patient is being prepared for surgery. Once the abdominal exploratory has been performed, antibiotics selecting anaerobes or Gram-negative organisms may be added as necessary, but are rarely required.

Complete gastric emptying prior to surgery is not advised. Immediate surgical intervention after resuscitation is preferred so that the gastric wall can be palpated and visualized as the orogastric tube is placed. Percutaneous trocharization of the stomach during initial resuscitation provides immediate release of some gas and fluid when appropriately placed and provides immediate pain relief with less anxiety. The lateral abdominal wall is percussed and the area sounding most tympanic/resonant is trocharized. Risks associated with trocharization include puncture

of intraabdominal organs, including the spleen, which can be malpositioned during GDV. Organs' percussion sounds can be dull/muted. Percutaneous trocharization is not as successful for decompressing food-bloat.

For catastrophic cases that are dying before your eyes, or when immediate surgery is not possible, naso- or oro-gastric intubation can be attempted. There is a risk of gastric rupture with a severely necrotic stomach. Tube passage is not always possible, and passage does not guarantee derotation.

Radiographs are not to be taken until fluid and analgesic therapy and temporary decompression have been initiated, unless euthanasia is an option over surgery. Abdominal radiographs confirm rotation with the classic "shelf" sign, where the pylorus is displaced dorsally and to the left. Thoracic radiographs are helpful in diagnosing underlying pulmonary disease or possible cardiomyopathy. A megaesophagus requires close monitoring. If there is no relief after gastric derotation, then a nasoesophageal tube may be required postoperatively for decompression to minimize regurgitation and aspiration. Radiographs should not delay surgical preparation.

Surgical Preparation

Surgical preparation of the abdomen, ventral thorax, and inguinal area is preferably performed prior to anesthesia to minimize the time under anesthesia. The lateral abdomen is prepared for possible gastrostomy or jejunostomy feeding tube placement. The inguinal area is prepared in case central femoral vascular access is required intraoperatively.

Rapid intravenous anesthetic induction is performed so that immediate control of the airway can be taken. Ketamine (5 mg/kg) and diazepam/midazolam (0.25 mg/kg) or propofol (2–4 mg/kg) can each have negative cardiovascular effects and should be titrated to effect. The amount of pressure placed on the diaphragm by the dilated stomach will hinder effective chest expansion. Assisted ventilation is recommended regardless of spontaneous ventilatory efforts.

Appropriate intraoperative monitoring includes constant ECG for dysrhythmia detection. Blood pressure monitoring is important, especially when the stomach is decompressed completely and the pressure on the central vein is released. A sudden decrease in preload can produce acute hypotension. In addition, many anesthetic agents augment hypotension (iso-, sevo-flurane) or potentiate arrhythmias (halothane). End-tidal CO_2 monitoring will identify inadequate ventilation. Pulse oximetry monitors trends of change in hemoglobin oxygen saturation.

Arterial blood gas analysis confirms that the instruments are working adequately and allow an assessment of the pH status. PCV/TS are essential in assessing the need for red blood cell transfusions, especially in the actively hemorrhaging patient. A PCV <25% (hemoglobin <8 g/dL) indicates the need for red cells/hemoglobin to provide oxygen carrying capabilities. Lactate levels that are not decreasing following resuscitation suggest continued maldistribution of blood flow, and extreme elevations (>10 mmol/L) may suggest hepatic failure or intestinal ischemia.

Surgical Intervention

A routine approach to the abdomen is made from xyphoid to pubis. Because of gastrointestinal distension, the linea is carefully incised along the more caudal aspect of the incision to avoid inadvertent incision into the intestine. If there is significant hemorrhage, sterile laparotomy pads may be required for packing. An attempt is made to pass a lubricated orogastric tube. The surgeon can gently guide the tube into the stomach after palpation of the gastric antrum and cardia. Usually the stomach will be rotated with the pylorus traveling ventrally and to the left, so one hand is used to press the right side of the dilated stomach down and the pyloric region is grasped with the other hand and gently pulled up ventrally and to the right. If passage of the tube is not possible, and the stomach is not able to be derotated, a large bore catheter stylet or needle can be used for centesis or attached directly to a suction apparatus for gas and fluid removal. If there is a large amount of food in the stomach, a gastrotomy is performed and the ingesta shoveled out.

Hemorrhage is controlled with packing and/or ligation. Any questionable stomach wall integrity is noted, and the rest of the abdomen is explored. Gastrectomy may not be required once blood flow is reestablished. The spleen is often congested and may be displaced. Evaluation for thromboses is made by palpation of the splenic arteries and inspection of the color of the parenchyma. Lack of blood supply or dark-purple color means the tissues have lost their blood supply and a partial or complete splenectomy is warranted. The pancreas is inspected for loss of blood

supply and edema. The remaining intestines are inspected for pathology. The genitourinary organs are routinely evaluated.

Once the exploratory is completed the stomach is reinspected. Most commonly the trauma to the gastric wall occurs along the fundus and cardia. If there are any black or dark purple regions, these should be removed with staples or resection. Any area that does not bleed bright red blood is suspect and should be removed. Invagination of ischemic tissues is not recommended because of potential life-threatening hemorrhage that can occur when the tissues slough. In contrast to the serosa, when the mucosa is black resection is not always necessary. The mucosa will regenerate if there is a healthy submucosa. However, it is the author's experience that removal of compromised but viable tissue results in decreased hospital stay. Postoperative monitoring for intragastric hemorrhage is important. The spleen is reinspected and either partially or completely removed if infarcted areas exist. Placement of a closed suction drain in questionable situations permits frequent monitoring of abdominal fluid for the presence of degenerative neutrophils with intracellular bacteria suggestive of septic peritonitis.

The type of pexy performed is completely dependant on the surgeon's preference and the time involved. The incisional gastropexy is rapidly performed and provides reliable adhesion. It is not recommended to suture the gastric wall to the midline abdominal incision, even in the critical GDV patient. Any future abdominal surgeries will be severely compromised by the adhesions formed.

Maintaining gastric decompression postoperatively is recommended in the critical GDV patient. Gastrostomy tube placement allows large volume decompression and removal of large clots that can occur with large resections. Nasogastric tubes are appropriate when gastric resection is not required. Nasogastric tubes are preferably placed intraoperatively with proper placement assured by palpation. Small volume infusion of electrolyte/glucose/glycine containing fluids feeds the gastric mucosal cells, which rely on intraluminal contents for nutrition. It is recommended that a jejunostomy or nasojejunostomy tube be placed if any significant gastric resection or pancreatic trauma occurs. Placed appropriately, intestinal feeding tubes provide immediate intestinal feeding postoperatively. It allows home care if gastric feeding is not possible once the animal is ready to be discharged. It also reduces the cost of parenteral nutrition because caloric requirements can usually be supplied within a few days.

Copious saline lavage and suction of the abdomen is necessary. A routine three-layer closure is performed and placement of dressing over the incision site and ostomy tube sites. Any evidence of peritonitis warrants culture and sensitivity of the peritoneum and possibly abdominal drainage.

Postoperative Care

It is not unusual for the post-operative GDV patient to continue to require large volumes of fluid replacement due to loss into the GI tract. Continuing colloid infusion at a maintenance rate promotes intravascular fluid retention during the healing process. Monitoring nasogastric tube suction volumes assists in more accurately determining volumes lost. When suction volumes decrease, this may indicate when refeeding may be initiated. Infusion of a balanced electrolyte/carbohydrate solution promotes gastric mucosal healing and feeding.

Continuous or intermittent monitoring of the vital signs will detect development of hypotension and/or dysrhythmias that may require immediate therapy. Monitoring PCV/TS, glucose, blood urea nitrogen (BUN), albumin, electrolytes, acid/base status, and lactate levels may uncover organ decompensation. Intravenous analgesia and antibiotic administration is continued until oral feedings and medications are tolerated. The use of promotility agents such as metoclopramide and cisapride may improve gastric emptying more rapidly than without. Reducing gastric acid secretion and administering sucralfate may be indicated in cases with mucosal denuding.

Suggested Reading

1. Brourman J, Schertel E, Allen D, et al. Factors associated with perioperative mortality in dogs with surgically managed gastric dilatation-volvulus: 137 cases (1988-1993). *J Am Vet Med Assoc* 1996;208(11):1855-1858.
2. Beck J, Staatz A, Pelsue D, Kudnig S, et al. Risk factors associated with short-term outcome and development of perioperative complications in dogs undergoing surgery because of gastric dilatation-volvulus:166 cases (1992-2003). *J Am Vet Med Assoc* 2006;229(12):1934-1939.
3. Buber T, Saragusty J, Ranen E, et al. Evaluation of lidocaine treatment and risk factors for death associated with gastric dilatation and volvulus in dogs: 112 cases (1997-2005). *J Am Vet Med Assoc* 2007;230(9):1334-1339.

4. Green T, Tonozzi C, Kirby R, Rudloff E. Evaluation of initial plasma lactate as a predictor of gastric necrosis and initial and subsequent plasma lactate values as a predictor of survival in dogs with gastric dilatation-volvulus: 84 dogs (2003-2007). *J Vet Emerg Crit Care* 2011;21:36-44.
5. Haak C, Rudloff E, Kirby R. Comparison of Oxyglobin and hetastarch during resuscitation of 20 dogs with GDV. In press, *J Vet Emerg Crit Care* 2012;22:201-210.
6. Pipan M, Brown DC, Battaglia CL, Otto CM. An internet-based survey of risk factors for surgical gastric dilatation volvulus in dogs. *J Am Vet Med Assoc* 2012;240(12):1456-1462.
7. Zacher LA, Berg J, Shaw AP, et al. Association between outcome and changes in plasma lactate concentration during presurgical treatment in dogs with gastric dilation-volvulus: 64 cases (2002-2008). *J Am Vet Med Assoc* 2010;236(8):892-897.
8. Goodrich ZJ, Powell LL, Hulting KJ. Assessment of two methods of gastric decompression for the initial management of gastric dilatation-volvulus. *J Small Anim Prac* 2013;54:75-79.
9. Bruchim Y, Ital S, Shira BH, et al. Evaluation of lidocaine treatment on frequency of cardiac arrhythmias, acute kidney injury, and hospitalization time in dogs with gastric dilatation volvulus. *J Vet Emerg Crit Care* 2012;22(4):419-427.
10. Adamik KN, Burgener IW, Kovacevik A, et al. Myoglobin as a prognostic indicator for outcome in dogs treated for gastric dilatation voluvlus. *J Vet Emerg Crit Care* 2009;19:247-253.
11. Van Kruiningen HJ, Gargamelli C, Havier J, et al. Stomach gas analysis in canine acute gastric dilatation with volvulus. *J Vet Intern Med* 2013;27:1260-1261.