Gastric dilatation volvulus (GDV) syndrome often begins with an accumulation of gas, fluid, or ingesta resulting in significant gastric dilatation. Gas content from dogs with GDV is similar to atmospheric air indicating that aerophagia is a likely cause. Simple dilatation may precede volvulus although volvulus without dilatation is reported. Clockwise rotation of the stomach is the most common type of volvulus and occurs in almost all dogs with GDV. Gastric outflow obstruction is usually not a component of GDV although delayed gastric emptying caused by gastric myoelectric dysrhythmias may be a factor.

The systemic pathophysiology associated with GDV is a direct result of the pathoanatomic features that accompany the syndrome. Elevating intragastric pressures of 30–80 mm of mercury initially cause venous stasis which may progress to infarction of the gastric wall. Translocation and even torsion of the spleen results in marked splenomegaly and sequestration of RBCs and clotting factors. Tearing of the short gastric vessels and gastrosplenic ligaments during volvulus may create intra-abdominal hemorrhage. Compression on the portal vein results in portal hypertension and pooling of splanchnic blood. Direct compression of the caudal vena cava further reduces venous return to the heart resulting in decreased cardiac output and corresponding hypotension.

Mucosal ischemia allows increased bacterial and endotoxin translocation and portal hypertension decreases hepatic clearance of these substances. On the cellular level this ischemia and endotoxin mediated endothelial injury activates the arachidonic acid cascade producing specific prostaglandins and pro-inflammatory cytokines. Increased capillary permeability, endothelial damage, neutrophil aggregation, platelet aggregation, and intrinsic clotting system activation occurs and may lead to disseminated intravascular coagulation (DIC). After reperfusion, superoxide, hydroxyl, and oxyradicals can further damage cellular membranes. Reperfusion injury is thought to contribute to increased cell death in the heart, liver, and GI tract.

**DIAGNOSTIC CRITERIA**

**Historical Information**

- **Gender predisposition.** GDV is more prevalent in male (60%) than female (40%) dogs. Intact male and female dogs are approximately 20% more likely to develop GDV than are neutered animals.
- **Age predisposition.** Gastric dilatation has been reported in dogs < 1 year and in dogs > 15 years old, with a mean age of approximately 7.5 years.
- **Breed predisposition.** Large and giant breed dogs are predisposed, with the Great Dane, Saint Bernard, weimaraner, Irish setter, standard poodle, and basset hound being the breeds at highest risk.

**Owner observations.** Owners commonly report vomiting or inappetence followed by hypersalivation, retching, unproductive vomiting with abdominal enlargement, tachypnea, and/or dyspnea. The receptionist staff should be trained to encourage immediate evaluation of large and giant breed dogs whose owners call in and report these signs.

**Other historical considerations/predispositions.** Body weight is important since underweight dogs are approximately three times as likely to develop GDV as are optimum weight dogs. Animals fed once daily are approximately twice as prone to GDV as those fed twice daily. Those animals considered to be fast eaters are approximately five times as prone as those classified as slow eaters. Changes in mealtime, sudden increases in food intake, or activity level increase risk. Previous removal of the spleen for splenic torsion may also predispose dogs. Dogs afflicted with nasal mite *Pneumonyssoides caninum* and those with inflammatory bowel disease (IBD) are also at increased risk.

**Physical Examination Findings**

Clinical findings in dogs with GDV are highly variable. Some patients are bright and alert while others are moribund indicating advanced stages of hypovolemic or septic shock.

- **Cranial abdominal distension is usually present although a small percentage of animals will have gastric and splenic displacement**
without noticeable abdominal enlargement.

- Since the lower gastroesophageal junction and pylorus are occluded, attempts at vomiting are unsuccessful. Increased parasympathetic tone induces hypersalivation.
- Shock status is variable with some animals showing normal membrane coloring and CRT while others are tachycardic with impaired organ perfusion. Pallor or hyperemia of the mucous membranes may be present along with prolonged CRT.
- Heart rate is often rapid, commonly exceeding 150 bpm. Pulse deficits may indicate concurrent ventricular arrhythmias. Femoral pulse is often weak.
- Tension on the diaphragm may impair inspiratory excursions and respiratory rate often exceeds 30 bpm.

**Laboratory Findings**

- **Blood gas abnormalities.** Acid base abnormalities are common with metabolic acidosis seen in >1/2 of patients. Acidemia results from reductions in plasma sodium bicarbonate concentration caused by decreased effective circulating blood volume, hypoxemia, and lactic acid formation. The mean venous pH is approximately 7.1 and the average plasma bicarbonate is about 18 mEq/L, with a mean base deficit of 7–8. Occasionally metabolic alkalosis, respiratory alkalosis, and/or respiratory acidosis are present.
- **Electrolyte abnormalities.** Many dogs have normal serum K+ levels at admission, but within 24 hrs hypokalemia occurs (mean 3.7 mEq/L). Hypokalemia is often compounded by the administration of IV crystalloid solutions deficient in potassium ions. Hypochloremia may be present at the time of admission prior to fluid administration.

- **Hemostatic profiles.**
  - Abnormal hemostatic profiles including prolonged prothrombin time (PT), prolonged activated partial thromboplastin time (APTT), reduced fibrinogen concentration, increased fibrin degradation product (FDP) concentration, reduced platelet counts, and reduced antithrombin III activity may be useful in estimating the incidence of gastric ischemia/necrosis and DIC.
  - Approximately 40% of dogs with GDV develop DIC.
  - In one study, about 70% of 20 dogs with more than one abnormal hemostatic test had gastric necrosis whereas dogs with one or less abnormality rarely had gastric necrosis.
  - In a study of 20 dogs with GDV, thrombocytopenia occurred in 9 of 20 dogs followed by decreased ATIII (8/20), elevated FDP (6/20), prolonged PT (6/20), hypofibrinogenemia (6/20), hyperfibrinogenemia (5/20), prolonged APTT (5/20), and shortened APTT (4/20).

- **CBC.** The PCV and TP values may vary. In the acute stage sequestration of fluids within the gastric lumen causes hemoconcentration, an elevated PCV and TP. Hypoproteinemia subsequently occurs due to transudation of fluid serum proteins into the vascular space. Lowered hematocrits in the range of 25% or less may be caused by hemorrhage from tearing of the short gastric arteries or be due to severe gastric mucosal ulceration and/or necrosis. WBC count is usually normal to slightly elevated. Occasionally the animal with gastric necrosis and impending gastric rupture may have a left shift.

**Other Diagnostic Findings**

- **Radiography.** Plain film radiography is delayed until after decompression and the right lateral abdominal view is most useful. In dogs with gastric volvulus the fundic area is located in a more ventral caudal position whereas the gastric antrum and duodenum is often filled with gas and is located in a more dorsal cranial position. The spleen may be located dorsal to the stomach particularly in the left lateral view. The ventral dorsal view is less useful, but may indicate compartmentalization of the stomach and translocation of the spleen.

- **Gastrocentesis.** If a diagnosis of gastric dilatation is in question, gastrocentesis (trocharization) can be performed with an 18 gauge needle or preferably an over-the-needle catheter. Recovery of intraluminal gastric air or gastric fluid contents is suggestive of GD or GDV. Attachment of a suction device to the needle or catheter often results in significant reduction in abdominal size.

- **Electrocardiography.** At admission most dogs with GDV have a normal ECG, but, within 24 hrs 40–50% of patients will develop cardiac arrhythmias which are usually ventricular in origin. These arrhythmias, in order of frequency, are premature ventricular contractions, paroxysmal ventricular tachycardia, sustained ventricular tachycardia, and second degree atrial ventricular block. The incidence of cardiac arrhythmias appears to be higher in dogs with concurrent gastric ischemia/ necrosis.
### TABLE ONE

**Status Based Medical Management of GDV**

<table>
<thead>
<tr>
<th>Shock status</th>
<th>Mild to moderate hypotension:</th>
<th>Severe hypotension:</th>
<th>Endotoxic Shock:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lactated Ringer’s solution 90 mg/kg over first hour (45 ml/kg is given over first 15 min)</td>
<td>70% hypertonic saline in 6% dextran 70</td>
<td>Lactated Ringer’s solution or hypertonic saline in dextran</td>
</tr>
<tr>
<td></td>
<td>After initial therapy maintenance on 4–6 ml/kg/hr</td>
<td>5 ml/kg over 5 min followed by LRS at 60 ml/kg/first hour</td>
<td>Plus cefazolin or ampicillin 22 mg/kg, IV, and enrofloxacin (Baytril®) 2.5 mg/kg, IV, bid, and metronidazole 15 mg/kg, IV, tid</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Plus Solu-Delta Cortef® 10–20 mg/kg, IV, or dexamethasone sodium phosphate</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Acid base status</th>
<th>pH &gt; 7.2 or base deficit &lt; 12 → no therapy</th>
<th>pH &lt; 7.2 or base deficit &gt; 12, 1–2 mEq/kg NaHCO₃, slow IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrolyte status</td>
<td>Mild hypokalemia (3.5–4.0 mEq)</td>
<td>Moderate hypokalemia (3.0–3.5 mEq)</td>
</tr>
<tr>
<td></td>
<td>Supplement KCl at 20 mEq/L fluids, not to exceed 0.2 mEq/kg/hr</td>
<td>Supplement KCl at 30 mEq/L fluids, not to exceed 0.3 mEq/kg/hr</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Coagulation status</th>
<th>Normal → no Rx</th>
<th>Evidence of DIC–heparin 100 mg/kg, SQ, tid</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Total solid status</th>
<th>&gt; 4.0 g/dl → no Rx</th>
<th>Fresh frozen plasma 20 ml/kg/d (best)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 4.0 dl → Hetastarch 10–20 ml/kg/d Dextran 70 10–20 ml/kg/d</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hematocrit status</th>
<th>&gt; 25% → no Rx if stable</th>
<th>&lt; 25% - whole blood transfusion or reconstituted packed red cells 25 ml/kg over 2 hrs</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Cardiac status</th>
<th>No arrhythmias → no Rx</th>
<th>PVC’s or tach causing hypotension Lidocaine 1–2 mg/kg, slow IV up to 4 times at 3 minute intervals, followed by continuous drip 2–6 mg/kg/hr or procainamide 6–15 mg/kg, IV over 30 minutes then 1–2 mg/kg/hr IV continuous drip. NOTE: No procainamide with hypotension</th>
</tr>
</thead>
</table>

### Summary of Diagnostic Criteria

- Acute/subacute abdominal distension with unproductive vomiting and/or hypersalivation.
- Resolution of abdominal distension after gastric decompression.
- Presence of malpositioned stomach on the right lateral view.
- Malposition of the spleen.
- Presence of metabolic acidosis and hypochloremia and hypokalemia.
- Persistent cardiac ventricular arrhythmias and/or coagulopathy may indicate the presence of gastric ischemia/necrosis.

### Differential Diagnoses

- Simple gastric distension secondary to overingestion of food and/or water.
- Aerophagia.
- Intestinal volvulus.
- Splenomegaly.
- Hepatomegaly.
- Ascites.
- Dysautonomia.
CHECKPOINTS

- **Anesthetic management.** Most anesthesiologists feel that thiobarbiturates and propofol should be avoided due to their hypotensive effects.
- **Timing of surgery.** This is very controversial with many surgeons feeling that early intervention will reduce mortality. Other surgeons feel that a prolonged period of stabilization with surgery after 12—24 hrs is preferable.
- **Gastroscopy method.** Although many techniques have been proposed, and although everyone has their favorite, results seem to be similar with all.

TREATMENT RECOMMENDATIONS

Initial Treatment

Treatment is dependent on clinical condition at admission. Hemodynamically stable patients can sometimes be treated with oro/ogastric intubation prior to fluid resuscitation. Conversely, patients with severe hypotension or endotoxic shock should undergo fluid resuscitation and possibly antibiotic therapy prior to initial gastric decompression. Shock therapy and decompression are often performed simultaneously.

- **Shock therapy.** Mild to moderate hypovolemic shock is treated with lactated Ringer’s solution at 90 ml/kg, administered as quickly as possible to effect cardiovascular stabilization within the first few minutes. In severe shock, IV administration of hypertonic saline (7% NaCl) solution and 6% dextan 70 is initiated with a bolus infusion of 5 ml/kg over 5 mins, followed by administration of lactated Ringer’s solution as rapidly as necessary to restore adequate circulation. CVP should be maintained in the 5—7 cm H2O range, and mean arterial blood pressure should exceed 70 mm Hg during resuscitative therapy.

- **Correction of acidosis.** This is only necessary if pH is $< 7.2$ or base deficit $> 12$ mEq/L. Dosage of sodium bicarbonate is 1–2 mEq/kg, slow IV. After administration, pH is reevaluated and therapy is repeated as necessary.

- **Gastric decompression.** In most cases gastric decompression can be performed without sedation or prior gastrocentesis. **Successful passage of the orogastric tube does not rule out the diagnosis of gastric volvulus.** Placing the dog in a sitting position or an upright position by supporting the elbows may help the distended stomach to fall away from the diaphragm and facilitates passage of a lubricated large diameter tube. If tube passage is unsuccessful gas can be removed from the stomach via gastrocentesis (trocharization) and the process repeated. In intractable dogs, ketamine 3–5 mg/kg and diazepam 0.5 mg/kg, IV, or oxymorphone (Numorphan®) 0.1 mg/kg, IM, IV can be used for sedation. Coffee ground colored ingesta or pieces of blackened gastric mucosa may indicate concurrent gastric ischemia/necrosis.

- **Gastric lavage.** In dogs having minimal gastric ingesta or fluid, gastric lavage is probably not necessary. In dogs with large amounts of gastric ingesta, food can be removed with several liters of warm water and the assistance of gravity flow, abdominal compression, and/or stomach pump syringe evacuation. Performing gastric lavage may increase the possibility of reflux and aspiration pneumonia.

- **Percutaneous gastrostomy.** If gastric decompression can’t be achieved a temporary gastrostomy tube can be inserted. After performing an inverted L block with 2% lidocaine in the left flank, the abdominal musculature is separated and a 20 Fr Foley catheter inserted through a purse string in the gastric wall. The stomach wall is sutured to the internal oblique, the cuff inflated with saline, and routine closure performed.

- **Ventricular arrhythmia therapy.** Treatment of arrhythmias associated with GDV is controversial since the results of several studies suggest that survival rate is the same in treated and untreated patients. However, animals with preexisting heart disease, or those with serious electrical disturbances resulting in reduced perfusion should be treated. A slow bolus of lidocaine at 1–2 mg/kg, IV may be repeated at 3 minute intervals to a maximum of 4 doses. This should be followed by a continuous infusion of 2–6 mg/kg/hr. Dogs refractory to lidocaine may be treated with boluses of procainamide at 6–15 mg/kg, IV over 30 minutes, followed by a continuous infusion of 1–2 mg/kg/hr.

**NOTE:** Procainamide is contraindicated in hypotensive animals.

- **Antibiotic therapy.** Antibiotic therapy is unnecessary in uncomplicated GDV. With prolonged clinical signs and/or suspected gastric ischemia/necrosis combinations of enrofloxacin (Baytril®) 2.5 mg/kg, IV, bid, plus cefazolin or ampicillin 22 mg/kg, IV, tid, will provide broad spectrum bactericidal activity. Those cases with suspected gastric ischemia/necrosis may also be supplemented with metronidazole at 10–15 mg/kg, IV, tid.

- **DIC therapy.** Heparin sodium is administered at 100 mg/kg, SQ.
Corticosteroids. Corticosteroids are unnecessary in uncomplicated GDV. In septic shock corticosteroids may potentially reduce serum levels of circulating endotoxin, promote reticulo-endothelial system clearance, and decrease complement fixation. Corticosteroids should not be instituted until after crystalloid fluid resuscitation. If indicated a single dose of prednisolone sodium succinate (Solu-Delta Corte®) 10–20 mg/kg, IV, or dexamethasone sodium phosphate 4 mg/kg, IV, may be administered.

Anesthetic and Surgical Management

Anesthetic induction and maintenance. Anesthetic induction with drugs which do not initiate hypotension is desirable. The combination of diazepam (Valium®) 0.2 mg/kg and oxymorphone (Numorphan®) 0.2 mg/kg given slowly IV to effect creates minimal hypotension. If the patient is not tachycardic, a combination of ketamine 10 mg/kg and diazepam 0.2 mg/kg can be given IV to effect. A new anesthetic drug, etomadate (Amidate®), has the least hypotensive properties of all agents and can be used at 2.0 mg/kg, slow IV to effect, but is very expensive. After induction, mask down with 2-3% isoflurane (Isovet®), followed by endotracheal intubation and maintenance on 1–1.5% isoflurane. During the course of anesthesia, brachycardia is treated with atropine sulfate 0.22 mg/kg, half dose IV and half IM, and hypotension with dopamine 5–10 mg/kg/min.

Derotation of stomach. After midline laparotomy, the spleen is retrieved and derotated if necessary. Presence of the greater omentum covering the stomach indicates gastric volvulus. The pylorus is located in the left upper quadrant of the abdomen, usually dorsal to the esophagus, and is then derotated in a counterclockwise direction. Examination of the remaining GI tract is performed.

Management of gastric necrosis. If present, gastric necrosis usually occurs at the greater curvature at the junction of the gastrosplicic ligament. Devitalized gastric tissue is denoted by greenish-gray or blue-black tissue which is thin on palpation. Gastric necrosis can be handled by partial gastrectomy using hand suturing, autostapling equipment, or through the use of a simple gastric involution technique. NOTE: Gastric involution involves the placement of an inverting Lembert suture pattern using polypropylene (Prolene®) which inverts the area of gastric necrosis without risking spillage. (Recommended by author.)

Prophylactic gastropexy. Most gastropexy procedures fix the right antral area of the stomach to the right body wall. Accepted methods for gastropexy include the tube gastrosomy, the circumcostal gastrosomy, the incisional gastrosomy, and the belt loop gastrosomy. The ventral midline gastrosomy is the most rapid procedure available and involves the inclusion of the seromuscular area of the gastric wall into the ventral midline closure. This procedure may be best for animals that are unstable under anesthesia.

Splenectomy. After derotation of the spleen, splenomegaly usually resolves and the spleen can be replaced in the abdomen prior to closure. Splenectomy is only performed if splenic arterial thrombosis is present.

Supportive Treatment

Opioids such as oxymorphone (0.05–0.1 mg/kg, IV) or fentanyl (5–10 µg/kg/hr) should be administered during anesthesia to help limit postoperative pain. Postoperatively, patients should receive oxymorphone at that dosage

Plasma lactate concentrations. Measurement of plasma lactate levels may be another more specific means of determining whether gastric necrosis is present and it may also give us an additional parameter for predicting survival.

Free radical scavengers. Several free radical scavengers have been used in an attempt to reduce myocardial and gastric ischemia secondary to reperfusion injury following initial resuscitation of GDV. Lazaroid, an oxysradical scavenger, and deferoxamine, an iron chelator, were shown to be beneficial in reducing free radical induced myocardial ischemia and increasing survival when given prior to gastric decompression. If deferoxamine is used, it is administered at a dosage of 23–30 mg/kg over 10–15 min. while monitoring BP, as it may produce hypotension in unstable patients. In practice, this should be done simultaneously with resuscitation and decompression and only in those patients whose condition permits spending this time for administration. Other free radical scavengers such as IV DMSO or allopurinol were not beneficial. Efficacy of these drugs given after gastric decompression is yet to be established.
Fluid therapy. Following shock therapy and gastric decompression uncomplicated patients need only maintenance crystalloid therapy at twice maintenance rate or 4–6 ml/kg/hr.

Correction of electrolyte abnormalities. Supplementing crystalloid solutions with K⁺ during initial crystalloid resuscitation may result in hyperkalemia and resultant cardiac arrhythmias. For mild cases of hypokalemia (3.5–4.0 mEq/L) add 20 mEq/L of KCl, for moderate cases (3.0–3.5 mEq/L) add 30 mEq/L, and for severe cases (2.5–3.0 mEq/L) add 40 mEq/L to each liter of lactated Ringer’s solution. The rate of K⁺ supplementation should not exceed 0.5 mEq/hr.

Protein/colloid supplementation. Animals recovering from endotoxic episodes or blood loss often require supplementation with fresh frozen plasma, hydroxyethyl starch (Hetastarch), or dextran (Dextran 70) at a rate of 10–20 mg/kg/day.

NOTE: Starch-based colloids may compound coagulopathies and should be given simultaneously with crystalloid solutions.

Blood components. Transfusions should be considered for patients whose hematocrit is <25%, or in unstable patients with a hematocrit <30%. Transfusion of either whole blood or suspended packed red cells at 25 ml/kg is performed over 2 hrs (faster in hypovolemic patients).

Gastric protectants. Animals suffering from GDV undergo severe gastritis, mucosal edema, and in some cases gastric necrosis. Support with H₂ blockers such as cimetidine (Tagamet®) 10 mg/kg, PO, IV, or IM, tid, or ranitidine (Zantac®) 1–2 mg/kg, PO or IV, bid is indicated. Cimetidine prolongs the serum half-life of lidocaine and may require dosage reductions of the lidocaine of 25–50%. Additionally, gastroesophageal reflux and esophagitis if present may be treated via the use of sucralfate (Carafate®) 0.5–1.0 gram PO, tid.

Promotility agents. Gastric atony is common following GDV and treatment with promotility agents such as cisapride (Propulsid®) 0.5 mg/kg, PO, tid, or metoclopramide (Reglan®) 0.5 mg/kg, PO or SQ, tid, may reduce postop vomiting and help encourage food ingestion postop period.

Antiarrhythmic drugs. As needed for continued cardiac arrhythmias. Significant arrhythmias rarely last >2 weeks, and most resolve within a few days of surgery.

Patient Monitoring-Home Management

Water intake. Excessive water intake may result in postoperative bloating episodes and water should not be offered ad libitum. The daily calculated water needs (70 ml/kg/d) should be divided and offered qid for one week postoperatively.

Feeding. The dog should be fed a low fat and protein diet, such as Hill’s Prescription Diet i/d®, which is blenderized. Meals are spaced qid for the first week followed by tid the following week and then bid thereafter.

Continued medications. Some dogs become chronic bloaters and require continued cisapride or metoclopramide therapy indefinitely. Animals with persistent arrhythmias may be maintained on sustained-release procainamide 6–20 mg/kg, PO, qid as needed.

Treatment Contraindications

Alpha, agonists such as xylazine cause severe hypotension and should be avoided.

Phenothiazines such as acepromazine may worsen severe hypotension and should be used with caution.

Pyloric surgery has been shown to increase the morbidity and mortality associated with GDV. Unless a specific pyloric lesion is present at the time of surgery gastric outflow procedures should not be performed.

Dextran are worse than hetastarch with respect to compounding coagulopathies.

PROGNOSIS

Favorable Criteria

The results of medical and surgical management of GDV have improved dramatically over the past 10 years. Recent studies claim mortality rates as low as 15% versus mortality rates in the 30–50% range a decade ago. Other factors that improve prognosis:

- Stable upon admission with good cardiovascular status.
- Absence of gastric ischemia/necrosis.
- One or less abnormal values on coagulation profile.

Unfavorable Criteria

- Presence of gastric ischemia/necrosis. This may be
indicated by a plasma lactate concentration >6mmol/L.

- DIC.
- Two or more abnormal values on coagulation profile.

RECOMMENDED READING


