DEFINITIONS
Enlargement of the stomach associated with rotation on its mesenteric axis is referred to as gastric dilatation-volvulus (GDV). The term simple dilatation refers to a stomach that is engorged with air or froth but not malpositioned. Dilatation refers to a condition in which an organ or structure is stretched beyond its normal dimensions; dilation is the act of stretching a cavity or orifice. GDV is also called gastric torsion or bloat.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY
Classically, GDV syndrome is an acute condition with a mortality rate of 20% to 45% in treated animals. The gastric enlargement is thought to be associated with a functional or mechanical gastric outflow obstruction. The initiating cause of the outflow obstruction is unknown; however, once the stomach dilates, normal physiologic means of removing air (i.e., eructation, vomiting, and pyloric emptying) are hindered because the esophageal and pyloric portals are obstructed.

The stomach becomes enlarged as gas or fluid or both accumulate in the lumen. The gas probably comes from aerophagia, although bacterial fermentation of carbohydrates, diffusion from the bloodstream, and metabolic reactions may contribute. Normal gastric secretion and transudation of fluids into the gastric lumen secondary to venous congestion contribute to fluid accumulation. The cause of GDV is unknown, but exercise after ingestion of large meals of highly processed food or water has been suggested to contribute to it. Epidemiologic studies have not supported a causal relationship between feeding soy-based or cereal-based dry dog food and GDV. However, Irish setters fed a single feed type appear to have an increased risk of GDV compared to those fed a mixture of feed types. Likewise, adding table food or canned food to the diet of large and giant breed dogs is associated with a decreased incidence of GDV. One study suggested that dogs fed a larger volume of food per meal were at significantly increased risk of GDV, regardless of the number of meals fed daily.

In the aforementioned study, the risk of GDV was highest for dogs fed a larger volume of food once daily. Feeding dry dog foods in which one of the first four ingredients are oils or fats may also increase the risk of GDV. Other contributing causes include an anatomic predisposition, ileus, trauma, primary gastric motility disorders, vomiting, and stress. Male gender, increasing age, being underweight, being fed a large volume of food per meal, eating one meal (especially a large volume meal) per day, eating rapidly, having a raised feeding bowel, and having a fearful temperament are predisposing factors that may significantly increase a dog’s risk of GDV. Having a deeper and narrower thorax may change the anatomic relationship between the stomach and esophagus such that the dog’s ability to eructate is impaired. Feeding dogs from a raised feed bowl may increase the risk of GDV because it may promote aerophagia. Some authors have recommended gastropexy to prevent GDV following splenectomy due to possible loss of gastric stabilization by the gastrosplenic ligament, but this has not been proven necessary. Finally, military working dogs appear to be more likely to develop a GDV in November, December, and January, but the reasons for this are uncertain. Atmospheric data have been related to the occurrence of GDV; minimum and maximum atmospheric pressure and the maximum atmospheric pressure the day prior were positively associated with GDV.

NOTE ● Having a first-degree relative with a history of GDV is significantly associated with an increased risk of GDV. Recommend that dogs with a first-degree relative that has had GDV not be used for breeding.

Generally, with GDV the stomach rotates in a clockwise direction when viewed from the surgeon’s perspective (with the dog on its back and the clinician standing at the dog’s side, facing cranially. The
rotation may be 90 to 360 degrees but usually is 220 to 270 degrees. The duodenum and pylorus move ventrally and to the left of the midline and become displaced between the esophagus and stomach. The spleen usually is displaced to the right ventral side of the abdomen.

Caudal vena cava and portal vein compression by the distended stomach reduces venous return and cardiac output, causing myocardial ischemia. Central venous pressure, stroke volume, mean arterial pressures, and cardiac output are reduced. Obstructive shock and inadequate tissue perfusion affect multiple organs, including the kidneys, heart, pancreas, stomach, and small intestine. Cardiac arrhythmias occur in many dogs with GDV, particularly those with gastric necrosis. Arrhythmias may contribute to mortality and require appropriate monitoring and treatment (see discussion under Postoperative Care and Assessment). Myocardial depressant factor has also been recognized in affected dogs, and cardiac damage is common, as seen by increased serum concentrations of troponin. Reperfusion injury has been implicated as causing much of the tissue damage that ultimately results in death after correction of GDV. Lazaroids (e.g., U74389G) which are radical-quenching antioxidants that inhibit oxygen-derived free radical production and lipid peroxidation, appear to reduce reperfusion injury and may eventually increase survival; however, further studies are needed.

Partial or chronic GDV may occur in dogs and usually is a progressive but non-life-threatening syndrome that may be associated with vomiting, anorexia, and/or weight loss. These dogs may have chronic, intermittent signs and appear normal between episodes. Gastric malpositioning may be intermittent or chronic but without dilatation. Plain or contrast radiographs are diagnostic, but repeat radiographs may be necessary if the stomach is intermittently malpositioned.

**DIAGNOSIS**

**Clinical Presentation**

**Signalment.** GDV primarily occurs in large, deep-chested breeds (i.e., Great Dane, weimaraner, Saint Bernard, German shepherd, Irish and Gordon setters, Doberman pinscher) but has been reported in cats and small-breed dogs. Shar peis may have an increased incidence compared with other medium-sized breeds. Basset hounds may have a higher risk of GDV, despite their relatively small size. GDV may occur in a dog of any age but is most common in middle-aged or older animals. The thoracic depth to width ratio appears to be highly correlated with the risk of bloat.

**History.** A dog with GDV may have a history of a progressively distending and tympanic abdomen, or the owner may simply find the animal recumbent and depressed with a distended abdomen. The dog may appear to be in pain and may have an arched back. Nonproductive retching, hypersalivation, and restlessness are common.

**Physical Examination Findings**

Abdominal palpation often reveals various degrees of abdominal tympany or enlargement; however, it may be difficult to feel gastric distention in heavily muscled large-breed or very obese dogs. Splenomegaly occasionally is palpated. Clinical signs associated with shock may be present, including weak peripheral pulses, tachycardia, prolonged capillary refill time, pale mucous membranes, and/or dyspnea.

**Diagnostic Imaging**

Radiographs are necessary to differentiate simple dilatation from dilatation plus volvulus. Affected animals should be decompressed before radiographs are taken. Right lateral and dorsoventral radiographic views are preferred in order to facilitate filling the abnormally displaced pylorus with air so that it can be easily identified. The pylorus is normally located ventral to the fundus on the lateral view.
and on the right side of the abdomen on the dorsoventral view. On a right lateral view of a dog with GDV, the pylorus lies cranial to the body of the stomach and is separated from the rest of the stomach by soft tissue (“reverse C sign” or “double bubble”), On the dorsoventral view, the pylorus appears as a gas-filled structure to the left of midline. Free abdominal air suggests gastric rupture and air within the wall of the stomach indicates necrosis, both of which warrant immediate surgery.

**NOTE**  ● Caution! Positioning these animals for a ventrodorsal view may lead to aspiration. Remember that the right lateral and dorsoventral views are preferred when attempting to diagnose GDV.

**Laboratory Findings**
The complete blood cell count (CBC) is seldom informative unless disseminated intravascular coagulation causes thrombocytopenia. Potassium concentrations may be normal or elevated, but hypokalemia is more common. Vascular stasis may cause increased lactic acid and metabolic acidosis. However, metabolic alkalosis caused by sequestration of hydrogen ions in the gastric lumen can offset the metabolic acidosis, causing the blood pH to be normal (i.e., a mixed acid-base disorder). Respiratory acidosis may be caused by hypoventilation secondary to gastric impingement on the diaphragm and diminished ventilatory compliance. Therefore routine administration of sodium bicarbonate is inappropriate. Plasma lactate concentrations are prognostic, higher values being associated with gastric necrosis and a poor prognosis.

**DIFFERENTIAL DIAGNOSIS**
Simple gastric dilatation occurs commonly in young puppies from overeating and seldom requires specific treatment. The stomach, although greatly enlarged with ingesta and gas, is not malpositioned. Small intestine volvulus is a differential diagnosis because it results in a tympanic and enlarged abdomen; however, dilatation of the intestinal tract is apparent on radiographs. Primary splenic torsion often causes acute abdominal pain; however, abdominal distention is absent to mild. Diaphragmatic herniation may produce clinical signs similar to GDV, particularly if the stomach is herniated and outflow is obstructed. Ascites can cause abdominal distention, but a fluid wave should be felt during ballottement, which distinguishes it from the tympanic abdomen found in GDV.

**NOTE**  ● You cannot differentiate GDV from gastric dilatation without volvulus simply because you are able to pass a stomach tube. Stomach tubes frequently can be passed in dogs with twisted stomachs.

**MEDICAL MANAGEMENT**
Stabilizing the patient’s condition is the initial objective. One or more large-bore intravenous catheters should be placed in either a jugular or both cephalic veins. Either isotonic fluids (90 ml/kg/hour), hypertonic 7% saline (4 to 5 ml/kg over 5 to 15 minutes), hetastarch (5 to 10 ml/kg over 10 to 15 minutes) or a mixture of 7.5% saline and hetastarch (dilute 23.4% saline with 6% hetastarch until you have a 7.5% solution; administer at 4 ml/kg over 5 minutes) is administered. If hypertonic saline or hetastarch is given, the rate of subsequent crystalloid administration must be adjusted. Blood should be drawn for blood gas analyses, a CBC, and a biochemical panel. Broad-spectrum antibiotics (e.g., cefazolin, ampicillin plus enrofloxacin) should be administered. If the animal is dyspneic, oxygen therapy may be given by nasal insufflation or mask.

Gastric decompression should be performed while shock therapy is initiated. The stomach may be decompressed percutaneously with several large-bore intravenous catheters or a small trocar, or (preferably) a stomach tube may be passed. The stomach tube should be measured from the point of the nose to the xiphoid process and a piece of tape applied to the tube to mark the correct length. A roll of tape can be placed between the incisors and the tube passed through the center hole. Attempts should
be made to pass the tube to the measured point. Placing the animal in different positions (i.e., sitting, reclining on a tilt-table) may help if it is difficult to advance the tube into the stomach. Do not perforate the esophagus with overly vigorous attempts to pass the tube. If these attempts fail, percutaneous decompression of the stomach should be attempted. This may relieve pressure on the cardia and allow the tube to enter the stomach. Once the air has been removed, the stomach should be flushed with warm water. Failure to lavage the stomach usually results in rapid re-dilatation after the tube is withdrawn. If blood is seen in the fluid from the stomach, prompt surgical intervention is warranted because this may indicate gastric necrosis. If the stomach tube still cannot be passed and immediate surgical correction is not possible, temporary decompression may be achieved by performing a temporary gastrostomy. A Foley catheter should not be placed in the stomach percutaneously unless the stomach is simultaneously tacked to the body wall because of the high risk of peritonitis if the stomach pulls away from the tube. The disadvantages of a temporary gastrostomy are that the stomach must be closed when the permanent gastropexy is performed, and there is a high risk of peritoneal contamination. However, a temporary gastrostomy maintains gastric decompression if the animal is being referred or if surgery is delayed. If immediate surgery is not possible in an animal in which a stomach tube was passed but that dilates rapidly after decompression, the stomach tube can be exteriorized through a pharyngostomy approach. This prevents the animal from chewing on the tube until definitive surgery can be performed. Radiographs may be taken after the patient has been decompressed and is stable.

SURGICAL TREATMENT
Surgery should be performed as soon as the animal's condition has been stabilized, even if the stomach has been decompressed. Rotation of an undistended stomach interferes with gastric blood flow and may potentiate gastric necrosis.

Preoperative Management
The animal should be given intravenous fluids and antibiotics before surgery (see discussion of Medical Management). Significant electrolyte and acid-base abnormalities should be corrected. A greatly enlarged stomach may hinder respiration and make it difficult for the animal to ventilate during induction of anesthesia. An ECG should be monitored to detect cardiac arrhythmias, which should be treated with lidocaine before surgery if they are significant (i.e., long runs of ventricular tachycardia which can decrease cardiac output).

Anesthesia
Numerous anesthetic protocols have been described for dogs with GDV. If the animal has been decompressed and its condition is stable without significant cardiac arrhythmias, then hydromorphone and diazepam may be given intravenously and the patient induced with etomidate, thiobarbiturates, or propofol. Refer to the surgery of the small intestine preoperative concerns and anesthesia sections for a more complete discussion of management of the patient with the acute abdomen. If the animal is depressed, premedication is probably not needed. Preoxygenation followed by a rapid induction with either ketamine and a benzodiazepine (e.g., midazolam, diazepam) or etomidate should be done. Etomidate is a good choice for induction if the animal's condition has not been well stabilized because it maintains cardiac output and is not arrhythmogenic. Lidocaine and thiobarbiturate may be used if arrhythmias are present; 9 mg/kg of each is drawn up, and half is given initially intravenously. Additional drug is given to effect to allow the dog to be intubated. Generally, no more than 6 mg/kg of lidocaine is given intravenously to prevent toxicity. If bradycardia occurs, anticholinergics (e.g., atropine or glycopyrrolate) may be given. Nitrous oxide should not be used in dogs with GDV. Isoflurane or sevoflurane are the inhalation agents of choice.
Surgical Anatomy
Normally, when viewed from the surgeon’s perspective (i.e., with the animal in dorsal recumbency), the pylorus is located on the dog’s right side, and the greater omentum arises from the greater curvature of the stomach and covers the intestines. The gastric (lesser curvature) and gastroepiploic (greater curvature) arteries supply the stomach and are derived from the celiac artery. The short gastric arteries arise from the splenic artery and supply the greater curvature. Rupture of the short gastric arteries in dogs with GDV is common and may contribute to blood loss and gastric infarction or necrosis. Eighty percent of the arterial flow is to the mucosa, and the remainder is to the muscularis and serosa; therefore, observation of mucosal color is not a reliable indicator of gastric wall viability. The mucosa often appears darkened because of vascular compromise, even when full thickness necrosis is not present.

Positioning
The dog is placed in dorsal recumbency, and the abdomen is prepared for a midline abdominal incision. The prepped area should extend from midthorax to the pubis. If a tube gastropexy is to be performed, the prepped area should be extended cranially and dorsally to allow the tube to be exteriorized behind the caudal right rib.

SURGICAL TECHNIQUE
The goals of surgical treatment are threefold: (1) to inspect the stomach and spleen so as to identify and remove damaged or necrotic tissues; (2) to decompress the stomach and correct any malpositioning; and (3) to adhere the stomach to the body wall to prevent subsequent malpositioning. Upon entering the abdominal cavity of a dog with GDV, the first structure noted is the greater omentum, which usually covers the dilated stomach.

Decompress the stomach before repositioning by using a large-bore needle (i.e., 14 or 16 gauge) attached to suction. If the needle becomes occluded with ingesta, have an assistant pass an orogastric stomach tube and perform gastric lavage.

Intraoperative manipulation of the cardia usually allows the tube to be passed into the stomach without difficulty. If adequate decompression is still not achieved or an assistant is not available, a small gastrotomy incision can be performed to remove the gastric contents, although this should be avoided if possible.

For a clockwise rotation, once the stomach has been decompressed, rotate it counterclockwise by grasping the pylorus (usually found below the esophagus) with the right hand and the greater curvature with the left. Push the greater curvature, or fundus, of the stomach toward the table while simultaneously elevating the pylorus towards the incision. Check to make sure the spleen is normally positioned in the left abdominal quadrant. If there is splenic necrosis or significant infarction, perform a partial or complete splenectomy. Remove or invaginate necrotic gastric tissues. Avoid entering the gastric lumen if possible. If you are uncertain whether gastric tissue will remain viable, invaginate the abnormal tissue. Verify that the gastrosplenic ligament is not torsed, and before closure palpate the intraabdominal esophagus to ensure that the stomach is derotated.

Perform a permanent gastropexy. Gastropexy usually is curative for dogs with partial or chronic GDV.

NOTE To prevent recurrence of GDV the stomach must be permanently adhered to the body wall. However, gastropexy does not guarantee that dilatation or volvulus will not recur; it simply...
Theresa W. Fossam
Gastric dilatation volvulus

makes it less likely. Gastropexy should always be performed in conjunction with abdominal exploration and derotation of the stomach.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS
Absorbable (polydioxanone or polyglyconate) or nonabsorbable (polypropylene) suture material may be used for the gastropexy (0 or 2-0). A Foley catheter is needed for a tube gastropexy. Balfour retractors, hand-held retractors (i.e., Army-Navy retractors or malleable retractors), and extra towel clamps (for placement on the rib when doing a circumcostal gastropexy) are helpful.

POSTOPERATIVE CARE AND ASSESSMENT
Electrolyte, fluid, and acid-basis status should be monitored closely postoperatively. Many dogs with GDV are hypokalemic postoperatively and require potassium supplementation. Small amounts of water and soft, low-fat food should be offered 12 to 24 hours after surgery and the patient observed for vomiting. Gastritis that occurs secondary to mucosal ischemia is common and may be associated with gastric hemorrhage or vomiting. If vomiting is severe or continuous, a centrally acting antiemetic may be given. Secondary gastric ulcers may occur and require treatment. H₂-receptor antagonists (e.g., cimetidine, ranitidine, or famotidine) reduce gastric acidity and may be beneficial. Intravenous fluid therapy should be continued until the patient’s oral fluid intake is adequate to maintain hydration. Patients should be monitored for hypoalbuminemia and anemia in the early postoperative period.

NOTE ● Lidocaine toxicity may be enhanced in patients given cimetidine concurrently.

Ventricular arrhythmias are common in dogs with GDV and usually begin 12 to 36 hours after surgery. Their cause is unknown, but myocardial depressant factor, reduced cardiac output, and myocardial ischemia may contribute. Treatment of cardiac arrhythmias includes maintenance of normal hydration and correction of electrolyte imbalances. Sometimes the arrhythmia can be corrected simply by correcting hypokalemia. Some antiarrhythmic drugs (i.e., lidocaine) are ineffective when the animal is hypokalemic. If the arrhythmias interfere with cardiac output as noted by poor peripheral pulses; are multiform; have subsequent premature beats inscribed on the wave of the previous complex (R on T); or have a sustained ventricular rate above 160 beats per minute, they should be treated with intravenous drugs. A test bolus of lidocaine given intravenously (2 mg/kg bolus, up to 8 mg/kg total dose) can be used to determine responsiveness to this drug. If the arrhythmias diminish or stop, lidocaine should be given by a continuous intravenous infusion of 50 to 75 µg/kg/minute. Low doses should be used initially and increased only if necessary. Signs of lidocaine toxicity include muscle tremors, vomiting, and seizures; lidocaine therapy should be discontinued if these signs occur. Other possibly effective antiarrhythmic drugs are procainamide and sotalol. Procainamide may be given as an intravenous bolus, by continuous infusion, intramuscularly, or orally. Sotalol may be effective in animals that have not responded to lidocaine or procainamide.

COMPLICATIONS
Sepsis and peritonitis may be caused by gastric necrosis or perforation if devitalized tissue is not adequately removed. Diagnostic peritoneal lavage may help diagnose peritonitis. Peritonitis requires immediate surgical intervention. Disseminated intravascular coagulation occurs in approximately 16% of dogs with GDV, but in a recent study was not found to be a risk factor for death. Assessment of clotting parameters and appropriate treatment with plasma, fluids and heparin is necessary because the development of coagulopathies increases risk of death. Cardiac arrhythmias are common in dogs with GDV (45.5%), but in the aforementioned study did not increase the risk of death associated with the condition.
PROGNOSIS
With timely surgery the prognosis is fair. Mortality rates 45% and higher have been reported; however, mortality rates as low as 10% are becoming more common. Gastric dilation without volvulus has a better prognosis than GDV; however, the degree of rotation is not associated with death in dogs with GDV. The breeds at greatest risk for mortality were the bloodhound, German longhaired pointer, and Neapolitan mastiff in one study. The prognosis is poor if gastric necrosis or perforation occurs or if surgery is delayed. Preoperative measurement of plasma lactate may be a good predictor of gastric necrosis and outcome for dogs with GDV. Plasma lactate concentrations under 6 mmol/L suggest that gastric necrosis is not present, and thus a fair prognosis is warranted. Dogs with gastric necrosis are approximately 10 times more likely to die than those that do not have gastric necrosis. Increased urinary 22-dTXB2-to-creatinine ratio following surgery was associated with an increased incidence of postoperative complications in dogs with GDV in one study. Recurrence rates for GDV differ, depending on techniques used, but most have reported rates of less than 10%. Tube gastropexy has the highest reported recurrence rate, varying from 5% to 29%.

Some dogs with GDV respond to tube decompression and medical stabilization alone. Occasionally the stomach becomes normally positioned after the air is removed, or it was only partly rotated (less than 180 degrees) or merely dilated. However, these dogs still have a high likelihood of recurrence, and gastropexy should be recommended even when conservative management successfully alleviates the gastric malpositioning. The reported recurrence rates in dogs operated on for GDV in which the stomach was repositioned but gastropexy was not performed approaches 80%.

Preoperative and postoperative arrhythmias have been associated with an increased mortality in one study. Concurrent splenectomy was also associated with a higher mortality rate in the same study, and careful consideration should be made prior to splenectomy in dogs with GDV; evaluate the spleen after derotation and allow ample time for any congestion to resolve before removing it.