Gastric Dilatation—Volvulus in the Dog

by Doug Lyman, DVM*

Gastric dilatation-volvulus (GDV) is a disease of many animal species and can be associated with a high mortality rate. The syndrome begins with a dilatation of the stomach for some unknown reason and progresses through a complex series of events involving many organ systems. It is the purpose of this paper to review the relevant events occurring in GDV and to relate the currently accepted forms of therapy.[10]

The cause of GDV remains a mystery. Predisposing factors include obstructive or nonobstructive processes that are accompanied by inhibition of the normal gastric motor or secretory functions, overeating, aerophagy, foreign bodies, parturition, pica, trauma, anesthesia and abdominal surgery. Additional factors include excessive exercise following a large meal, rapid eating and once per day feeding.[3] The condition is most often seen in certain large and deep-chested breeds of dogs including Great Danes, St. Bernards, Bloodhounds, Standard Poodles, Boxers, Irish Setters, Weimaraners, English Sheepdogs and Gordon Setters. The disease is also common in young kittens and puppies that eat too rapidly. Gastric torsion has been linked to lengthening of the gastric supporting ligaments and a pendulous food filled or atonic stomach. It is considered a disease of domestication since we have changed dogs from carnivores to omnivores by feeding them commercial diets containing cereals such as soybean oil meal.[1] These highly fermentable diets are related to an increased incidence of GDV since they are high in tryptophan, which has been shown to be a potent inhibitor of gastric emptying.[3] Uncomplicated gastric dilatation is seen most often in puppies that overeat although it can be seen in any dog at any age. The complication of volvulus is most apt to occur in large, deep-chested dogs following ingestion of a large meal of dry, poorly digestible food, excess water or exercise.[3] Bacterial fermentation and gas production are greatly increased in GDV dogs' yet the dynamics of this and the organisms involved remain a mystery.[10] Clostridia have been proposed as playing a role, however *Cl. perfringens* has been found to be a normal inhabitant of the stomach of most dogs so this role has yet to be proven.[9] In summary, except in the case of the over-eating puppy or kitten, the etiology is unknown. It is most likely a multi- factorial disease involving aerophagia, feeding, management practices, hereditary factors and other unknown causes.

Acute gastric dilatation develops because the stomach cannot be emptied thus causing increased intragastric pressure. Normally, such pressure is relieved by vomiting or eructation. With this condition the gas, liquid and solid contents are retained. Analysis of gas from clinical cases has suggested that swallowed air is the major source. Dilatation of the stomach causes it to rotate on its long axis producing a torsion of the gastroesophgeal junction. Distension causes the greater curvature to move ventrally and the pylorus to move dorsally and to the left.[1] The stomach usually moves in a clockwise direction as viewed from the caudal end of the patient. The spleen follows the stomach in its clockwise movement and becomes engorged due to interference with its vessels. When the degree of twisting of the stomach is less than 180 degrees there is incomplete obstruction of the esophagus and pylorus, whereas twists of greater than 180 degrees provide complete obstruction resulting in volvulus.[4] Dilatation

*Dr. Lyman is a 1981 graduate of the College of Veterinary Medicine, ISU.

and volvulus initiate a series of events that ultimately leads to shock. Most notably, the dilated stomach occludes circulation through the portal system and caudal vena cava resulting in hypovolemic shock. Shock is probably also produced by peptides with vasoactive and myocardial depressant effects. The peptides result from pancreatic ischemia which stimulates the release of lysosomal enzymes. These act on plasma proteins to produce the peptides. As blood pressure falls and abdominal perfusion decreases, gut motility slows or ceases, resulting in the production and absorption of endotoxins which further contribute to shock. The reduced cardiac output, hypoxemia and effects of the toxic peptides contribute to myocardial ischemia and hypoxia-producing cardiac arrhythmias that can be fatal. This state of hypoxia, venous stasis and endotoxemia also can lead to DIC which causes platelet aggregation and a depletion of clotting factors thus predisposing the dog to hemorrhage. As gastric dilatation progresses and intragastric pressure increases, venous drainage from the stomach is decreased. This stasis quickly leads to mucosal anoxia resulting in hemorrhage and edema, which, if left unrelieved, could ultimately lead to gastric rupture.[1]

Diagnosis of GDV is generally made on the basis of clinical exam but clinical pathology and radiology are also important to assess the overall condition of the patient. Each of these aspects will be examined separately.

Clinical exam

Cranial abdominal distension to the left, right or both sides is the most prominent sign.[2] The animal becomes restless and retches. Frequent attempts to vomit are usually unproductive. If vomition occurs, the distension will be corrected. Excessive salivation commences and the animal often shows signs of pain upon abdominal palpation. As the syndrome progresses, the dog may become reluctant to move and may assume a sawhorse stance with its mouth open, grunting in pain. The breathing is shallow and rapid due to decreased tidal volume caused by the encroachment of the enlarged stomach on the lungs. The mucus membranes are pale and capillary perfusion time is slow. The temperature becomes subnormal as shock increases. Eventual collapse and death are imminent with a total course of less than eight hours being possible. A reasonably good clinical diagnosis can be made based on the following triad of signs: 1) retching with unproductive vomiting, 2) tympany and a rapidly enlarging abdomen, 3) inability to pass a stomach tube (if volvulus is present).[3]

Clinical pathology

Some findings might include the following:

1. Increased PCV and plasma protein due to loss of fluid into the stomach.
2. Metabolic acidosis due to buildup of organic acids, especially lactate.
3. Increased SGPT (moderate) due to passive congestion causing hepatic necrosis.
4. Slightly increased BUN and creatinine due to decreased renal perfusion.
5. No significant electrolyte changes except that plasma potassium can increase up to 2x within 2 hours following decompression.[1]

Radiology

Radiology is rarely necessary to diagnose acute GDV, but it is important in differentiating simple dilatation from volvulus. It should be noted that this is not always an easy task because the gastric distension tends to confuse the shapes and positions of abdominal structures.[4] It should also be noted that volvulus or splenic torsion can remain after decompression. Therefore, it is necessary that all dogs not requiring a celiotomy be radiographed following decompression. Some dogs will eat and bloat only intermittently despite having persistent volvulus.[1]

Rule Outs and Treatment

A differential diagnosis should include the following:

1. Acute pancreatitis—this usually shows productive vomition without bloat.
2. Intestinal problems—(intussusception, volvulus, obstruction) these are generally less painful than GDV.
3. Acute gastritis and foreign bodies—these are also less severe with excessive vomition.
4. Pyloric malfunction, especially with dilatation in small breeds—this may indicate pylorospasm, stenosis or neoplasia.[4]

Treatment of GDV is divided into three

categories: shock therapy, decompression and definitive surgical correction.

1. Shock therapy: This should include intravenous fluids, corticosteroids and antibiotics, each of which will be discussed separately.
   a IV fluids—an immediate goal of treatment is to improve cardiovascular function by rapid administration of fluids. Lactated Ringers should be given at a rate of up to one blood volume (40 cc/lb) in the first hour.[5] This should increase venous return and lessen vascular stasis in capillaries. Plasma bicarbonate deficits up to 5-15 mEq/l can be expected.[1] Bicarbonate should be given at a rate of 1-2 mEq/lb to correct this deficit.[5] If mild hyperkalemia is a problem, as it is in experimental models, it should be resolved by the bicarbonate which will increase blood pH and drive potassium intracellularly.[1]
   b. Corticosteroids—bolus injections of glucocorticoids should be given to help increase cardiac output, stabilize lysosomal membranes and decrease the body's reaction to while increasing the clearance of endotoxins.[5] It is recommended that Azium[a] be given at 1mg/lb IV and Solu-Delta-Cortef[b] at 5-10 mg/lb. The Azium should take effect about the time the Solu-Delta-Cortef is wearing off.[4]
   c. Antibiotics—an aqueous antibiotic should be used to help control both aerobic and anaerobic bacteria that are absorbed from the gastrointestinal tract and may be producing endotoxin. The drug of choice is chloramphenicol at 25 mg/lb IV.[1]
   d. Lidocaine hydrochloride is given at 4 mg/kg initially and 25-50 mg/kg/min to control any cardiac arrhythmias that may develop.[1]
   e. If necessary, DIC is treated with heparin at 50-150 units/kg every 4-6 hours.[1]
2. Decompression: Once shock therapy has been instituted, efforts should be made toward relieving the gastric dilatation. This can be accomplished by several methods. Ideally, the chosen method should not add to the respiratory or cardiovascular insults, should be rapidly performed, should avoid peritoneal contamination and should allow for ongoing relief of the problem.[7] Of the following procedures, only gastrostomy seems to meet these criteria. At this point, some attention should be given to anesthetic considerations, which can be critical in a shocky animal. Since the heart may be very sensitive to arrhythmias one should be wary of gas and barbiturate anesthetics. Nitrous oxide is definitely contraindicated as it increases the distension of gas pockets in the gastrointestinal tract.[6] If general anesthetics are required, tracheal intubation and assisted ventilation are essential. One should also consider monitoring the EKG. If sedatives are required, it is critical to choose those that cause the least respiratory depression.[7] In light of all this, it becomes apparent that the ideal situation would involve the use of a local anesthetic only, which is often possible since the animal is usually depressed.
   a. Gastric intubation—this involves the passage of a stiff, premeasured vinyl tube into the stomach. If the tube is easily passed and the distension relieved, one should lavage the stomach with warm saline. The animal should then be watched closely for 24 hours for recurrence, which is common. If this is not possible, one should consider placement of a pharyngostomy tube to ensure continuous decompression until normal gastric motility returns.[4] It should be noted that an inability to pass a stomach tube is not diagnostic for volvulus, and that a tube can be passed in some dogs that have volvulus.[1] For this reason, it has been recommended that, following decompression, all animals undergo an upper GI barium series to detect any malpositions. The main disadvantages of gastric intubation are the added respiratory distress from restraint and tube passage, and the threat of perforation of an already friable gastric wall.[7]
   b. Gastrocentesis—this procedure is recommended only as a last resort or when other methods are unavailable. It involves trocharization with a 16-18 gauge needle high in either flank. One should again attempt to pass a stomach tube after trocharization. This procedure risks peritonitis, but this is a life-threatening situation and the

peritonitis can most likely be managed later.[7]

c. Gastrotomy—this doesn't seem to be a good method of decompression as the time spent in preparation and the depressant effects of general anesthesia are both contraindicated.

d. Gastrostomy—there are two schools of thought relevant to decompression and surgical treatment of those cases in which a stomach tube cannot be passed. One approach advocates immediate laparotomy and surgical correction of the volvulus. The other approach involves the use of a temporary gastrostomy to achieve decompression and then allowing a period of time for the patient to stabilize before attempting definitive surgical correction.[7] The latter procedure has lowered the mortality rate from 68% to 33% at one institution.[1] A right paracostal gastrostomy comes closest to the ideal solution since it is rapid, is done under local anesthesia, minimizes peritoneal contamination and maintains decompression as the animal stabilizes. It also allows for direct visualization of the gastric mucosa and wall. This is very important since there is an apparent direct clinical relationship between mortaility and gastric wall necrosis. If necrosis is evident, the patient should be taken in for immediate resection.[7]

3. Surgical correction: Many different methods have been advocated to correct the anatomical and pathological abnormalities that occur, none of which will completely prevent recurrence of volvulus.[4] Ideally, a complete blood profile with serum chemistries as well as an EKG should be performed before surgery to alert the surgeon of any anesthetic or surgical risks.

a. Pyloroplasty—this has been advocated to increase gastric emptying.[7]

b. Splenectomy—this is done only in those cases in which significant thrombosis and devitalization are present.[7]

c. Gastropexy—this entails surgical fixation of the stomach to the abdominal wall which encourages adhesions and decreases motility. However, the adhesions tend to break down rapidly due to strong gastric contractions.[7]

d. Tube gastrostomy—this creates an opening in the stomach for continued decompression and feeding while binding the stomach to the abdominal wall allowing adhesion formation. This procedure requires little time and is the only one to significantly reduce mortality at the Animal Medical Center. This technique promotes gastric fixation by dense adhesions exceeding those obtained by gastropexy alone.[7]

Since these animals are predisposed to relapse, they should be fed small amounts of easily digested food several times per day. Post-prandial exercise should not be allowed and clean fresh water should always be available.[4]

The mortality rate associated with GDV is at least 30% and will be higher if surgery is required. There is a less optimistic prognosis when gastric resection is performed.[1]

All cases of GDV must be considered as true emergencies and consequently one should keep several things in mind. 1) It is mostly a problem of large breed dogs. 2) Animals are predisposed to recurrences of GDV. 3) It must be accurately diagnosed and promptly treated. 4) No single or combination of surgical procedures will completely eliminate recurrences.[4]

REFERENCES

1. Strombeck, DR: Acute gastric dilatation-volvulus, In: *Kirk's Current Veterinary Therapy VII,* WB Saunders, Philadelphia, Pa. 1980, pp. 896-901.
2. Wingfield, WE: Acute gastric dilatation and gastric volvulus, In: *Veterinary Gastroenterology,* Lea and Febiger, Philadelphia, Pa. 1980, pp 449-455.
3. Gabbert, NA: Acute gastric dilatation and volvulus, In: *Canine Medicine,* 4th Ed. American Veterinary Publications, Santa Barbara, Cal. pp 329-333.
4. Grauer, GF; DeYoung, DW: Gastric dilatation and volvulus in the Dog, *ISU Veterinarian,* 40(1):16-23, 1978.
5. Rawlings: Shock therapy and anesthetic management in GDV, *JAAHA,* March/April 12:158-161, 1976.
6. Lumb VW; Jones EW: *Veterinary Anesthesia,* Lea and Febiger, Philadelphia, Pa. 1973.
7. Parks, J: Surgical management of gastric torsion, In: *Veterinary Clinics of North America, Small Animal Practice,* 9(2):259-267, 1979.
8. Greene, RW: GDV complex, *AAHA Proceedings,* 1977, pp 227-229.
9. Incidence of clostridia in canine stomach, *JAAHA* 14(5):618-623, 1978.
10. Wingfield, WE: Pathophysiology associated with GDV in the Dog, *JAAHA,* March/ April 12:136-141, 1976.

[a]Azium (dexamethasone), Schering Corp., Kenilworth, N.J.

[b]Solu-Delta-Cortef (prednisolone sodium succinate), Upjohn, Kalamazoo, Michigan.