Canine gastric dilatation/volvulus syndrome in a veterinary critical care unit: 295 cases (1986–1992)

Daniel J. Brockman, BVSc; Robert J. Washabau, VMD, PhD; Kenneth J. Drobatz, DVM

Summary: *Two hundred ninety-five case records were included in an analysis of dogs treated by a standardized protocol for gastric dilatation/volvulus syndrome between 1986 and 1992. A breed predisposition was demonstrated for Great Danes, German Shepherd Dogs, large mixed-breed dogs, and Standard Poodles. One hundred and ninety-three dogs had gastric dilatation and volvulus (GDV) confirmed at surgery, 66 had simple gastric dilatation (GD), and 36 others had gastric dilatation but volvulus could not be proved or disproved (GD ± V). Among dogs with GDV, the fatality rate was 15% (29/193). Twenty-six (13.5%) dogs with GDV underwent partial gastrectomy, and 8 (31%) died or were subsequently euthanatized. In comparing the group of dogs with GDV that survived to those that died, there were no statistical differences in the age of dog, time between onset of clinical signs and admission, time from admission to surgery, or duration of anesthesia. Cardiac arrhythmias were detected in 40% (78/193) of the dogs with GDV. There also was no statistical correlation between development of a cardiac arrhythmia and outcome in dogs with GDV. The causes of death in dogs with GDV were multiple and varied; presumed gastric necrosis was a common reason for intraoperative euthanasia (11 dogs). Among dogs with GD or GD ± V, the fatality rate was 0.9% (1/102).*

Gastric dilatation/volvulus (GDV) syndrome is a life-threatening disorder primarily affecting large and giant-breed dogs.[1-3] Despite early intervention, fatality rates remain high.[2,4,5,a] Fatality rates between 10 and 60% have been reported in various studies[2-7,a] but these rates are difficult to interpret, because various treatment protocols were used, even within a single institution. Further, dogs in some studies benefited from continuous critical care support, whereas others did not.[7-9,a]

In 1986, guidelines were established at the veterinary hospital at the University of Pennsylvania to standardize the treatment of dogs with GDV syndrome. These guidelines were based on the premise that a short period of medical stabilization, prompt definitive surgical treatment, and postoperative intensive care would reduce the mortality and morbidity associated with the disease. The purpose of the study reported here was to determine whether dogs treated by this standardized protocol for GDV would have reduced mortality and morbidity.

From the Department of Clinical Studies (Brockman, Washabau, Drobatz) and the Center for Veterinary Critical Care (Drobatz), School of Veterinary Medicine, University of Pennsylvania, 3850 Spruce St, Philadelphia, PA 19104-6010.

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Criteria for Selection of Cases

The medical records of dogs with GDV syndrome that were examined at the veterinary teaching hospital between January 1986 and December 1992 were reviewed. A medical record was included in the study only if the record contained results of physical examination performed at the time of emergency admission, a radiology report, a surgery report, and standard protocol treatment documents.

Tests and Procedures

Three groups of dogs were identified: GDV dogs = dogs with GDV confirmed at surgery; GD dogs = dogs with radiographically normal gastric position and in which orogastric intubation was easily performed; and GD ± V dogs = dogs with radiographic evidence of volvulus, but with normal gastric position at surgery. Data obtained from the medical records included signalment, time of onset of clinical signs, time of admission to the emergency room, time anesthesia was commenced, nature and duration of surgery, occurrence of cardiac arrhythmias, length of hospitalization, prevalence and type of postoperative complications, and overall outcome (life or death). Any dog in which the case record contained mention of an arrhythmia (other than transient ones associated with surgical manipulation of the stomach) was included in the data analysis as having an arrhythmia. Further classification of the type of arrhythmia was obtained directly from electrocardiographic recordings in the record or from the cardiology report. Survivors were considered to be any dog that was discharged from the hospital.

Standard emergency room procedures—After physical examination, 2 large-bore catheters (16 gauge) were placed in the cephalic or jugular veins of dogs affected with the GDV syndrome. Blood samples were obtained for analysis of PCV and concentrations of serum total solids, blood glucose, BUN, plasma sodium, and plasma potassium, as well as for the performance of a CBC and serum biochemical analysis. Balanced electrolyte solution was infused at a rate of 90 ml/kg of body weight/h for the first hour. Fluid treatment was adjusted thereafter. Intermittent or continuous electrocardiographic monitoring was performed in all dogs. Gastric decompression was then achieved by a combination of orogastric intubation or needle gastrocentesis. A sedative combination of oxymorphone (0.1 mg/kg, IV) and diazepam (0.5 mg/kg, IV) was administered to noncompliant dogs. A right lateral abdominal radiographic view was obtained after gastric decompression. Additional radiographic views (ventrodorsal and left lateral views) were obtained if the initial radiographic findings were equivocal. A radiographic diagnosis of GDV was made if the gastric pylorus was displaced dorsally relative to the gastric fundus.[9] A

diagnosis of GD alone was made if abdominal radiography revealed normal gastric position with the pylorus to the right of midline in the midventral region of the abdomen and the fundus to the left of midline in the middorsal region of the abdomen. All dogs were continually assessed by intermittent monitoring of peripheral pulse pressure and quality, heart rate, mucous membrane color, capillary refill time PCV, and total solids concentration. Conservative treatment was only undertaken in those dogs (66 dogs) in which orogastric intubation was easy, radiographic views were indicative of normal gastric position, and hemodynamic values were stable or improving. Exploratory laparotomy was performed in all others (229 dogs).

Standard anesthesia and operating room procedures—After gastric decompression, additional catheters were placed IV (eg, saphenous vein), and an arterial catheter was placed in the dorsal pedal or metatarsal artery. Blood pressure was monitored either by direct measurement or indirectly by Doppler ultrasound or oscillometry. Endotracheal intubation was then performed, following further IV infusion of oxymorphone (0.1 mg/kg), diazepam (0.2 to 0.5 mg/kg), and lidocaine (2 mg/kg). Anesthesia was maintained with isoflurane in 100% oxygen, using a low-flow closed system. Treatment with fluids administered IV was continued at a rate of at least 20 ml/kg/h. Fluid rate and composition were adjusted according to alterations in heart rate, arterial blood pressure, peripheral pulse, mucous membrane color, PCV, and concentrations of serum total solids, BUN, blood glucose, plasma sodium, and plasma potassium. Synthetic colloids[b] and blood products were administered to some dogs to maintain a PCV greater than 22 and to preserve intravascular volume (as determined by mucous membrane color, capillary refill time, heart rate, and peripheral pulse pressure and quality). Hypotension was further treated with phenylephrine (10 mg in 250 ml 0.9% NaCl solution) or epinephrine (1 mg in 250 ml 0.9% NaCl solution), given to effect so as to maintain mean arterial pressure above 60 mm of Hg.

After routine aseptic preparation, a cranial ventral midline celiotomy was performed. Further gastric decompression was achieved by needle gastrocentesis or orogastric intubation, and the stomach and spleen were returned to their normal position. A systematic evaluation of the abdominal cavity was performed. The stomach was lavaged with warm water via the orogastric tube if all abdominal organs appeared grossly normal. When present, large pieces of indigestible and digestible solids were removed via gastrotomy. Gastric and splenic blood flow were evaluated subjectively and devitalized tissues were excised either by hand or by use of surgical stapling and ligation instruments.[c] Gastropexy was then performed to anatomically stabilize the pyloric antrum to the right side of the body wall. The abdomen was lavaged with sterile 0.9% NaCl solution and closed in a routine manner. A bandage was placed around the abdomen in dogs undergoing tube gastropexy.

Standard intensive care unit procedures—Dogs were admitted to the intensive care unit immediately after surgery where they were continuously monitored for urine output, heart rate and rhythm, and blood pressure. Concentrations of plasma electrolytes, PCV, serum total solids, blood glucose, and arterial P_{O_2} were determined intermittently. Blood products, synthetic colloids, potassium chloride, dextrose, and O_2 were supplemented as necessary. Dogs were treated with antiarrhythmic agents only if an arrhythmia was associated with poor tissue perfusion (as determined by arterial blood pressure, peripheral pulse quality, mucous membrane color, and capillary refill time), if preexisting cardiac disease was present (as determined from medical history), or when the persistent, closely associated multiform ventricular excitation or superimposition of the QRS wave on the T wave of the ECG trace (R on T phenomenon) was detected. In treating ventricular arrhythmias, if an initial bolus of lidocaine (1 to 8 mg/kg, IV) was successful, it was followed by an IV infusion of lidocaine (0.04 to 0.08 mg/kg/min). Boluses of procainamide (0.5 to 4.0 mg/kg, IV) were used in dogs that did not respond to lidocaine; if successful, it was followed either by an IV infusion (0.04 mg/kg/min) or an IM injection (6 to 8 mg/kg/6 h). Combination treatment (lidocaine 0.04 to 0.08 mg/kg/min, IV; and procainamide 6 to 8 mg/kg/6 h, IM) was used in dogs that were refractory. Treatment was only continued if it had succeeded in abolishing the ECG abnormality. Vasopressor agents were used in persistently hypotensive dogs. Dogs were transferred from the intensive care unit to the ward when objective parameters were stable, their mental status was subjectively good, they were eating small amounts of food, and they were drinking water. At this time crystalloid fluids alone, at a rate of 2 to 4 ml/kg/h, were administered.

Statistical analysis—To assess the association between clinical findings and fatality, categorical data (eg, breed and sex) were analyzed by Pearson's χ^2 test. Continuous data were analyzed by Student's *t*-test. Logistic regression was used to identify clinical factors (eg, duration of signs and duration of surgery) that were independently associated with an increased risk of death. Probability values of less than 0.05 were considered statistically significant. All statistical analyses were performed by use of computer programs.[10]

Results

Two hundred ninety-five cases of GDV, GD, and GD ± V in dogs were identified. These dogs represented 0.8% of the total number of emergency room accessions and 0.28% of total hospital accessions during the period of the study. Of these 295 dogs, 58% (171/295) were male and 42% (124/295) were female. The mean age was 7.5 years. With the exception of 1 Pekingese and 1 Dachshund, all had a large-breed or giant-breed type of conformation. Thirty-five breeds of dogs were represented, including German Shepherd Dogs, 21% (62/295); Great Danes, 14% (41/295); large (> 20 kg) mixed-breed dogs, 10% (29/295); Doberman Pinschers, 6.1% (18/295); and Standard Poodles, 5.1% (15/295). A significant breed predisposition was demonstrated for Great Danes, German Shepherd Dogs, large mixed-breed dogs, and Standard Poodles (Table 1).

Dogs with gastric dilatation and volvulus that survived—Eighty-five percent (164/193) of the GDV dogs survived. Their mean age was 7.25 ± 3.6 years, the

Table 1—Breed predisposition for gastric dilatation/volvulus syndrome in 295 dogs

Breed	Frequency	Odds Ratio	χ^2	P
Great Dane	28	14.5	283.8	<0.0001
German Shepherd Dog	40	4.1	73.0	<0.0001
Large mixed-breed	20	5.2	60.1	<0.0001
Standard Poodle	11	5.5	37.2	<0.0001
Others	196	ND	ND	ND

ND = Not determined.

mean duration of their hospital stay was 4 days (range, 2 to 14 days). The mean time between onset of clinical signs and admission (n = 126) was 5.5 ± 9.6 hours. The mean time between admission to the emergency room and induction of anesthesia was 2.7 ± 3.2 hours. Three percent (5/164) of the dogs required simple gastrotomy, and 11% (18/164) underwent partial gastrectomy. Seventeen percent (28/164) also had partial or complete splenectomy. Tube gastropexy was performed on 86.5% (142/164), a muscular flap gastropexy on 4.9% (8/164), a circumcostal gastropexy on 4.3% (7/164), and a belt-loop gastropexy on 4.3% (7/164). Mean anesthetic time was 2.3 ± 0.7 hours. Forty percent (65/164) of the surviving GDV dogs developed a cardiac arrhythmia. These were characterized as intermittent premature ventricular conductions (n = 32), sustained ventricular tachycardia (n = 14), paroxysmal ventricular tachycardia (n = 12), second-degree atrioventricular block (n = 4), and atrial fibrillation (n = 3). Antiarrhythmic treatment was administered to 34% (22/65) of the surviving GDV dogs with arrhythmias.

Dogs with gastric dilatation and volvulus that did not survive—Fifteen percent (29/193) of GDV dogs did not survive. The mean age of these dogs was 10.2 ± 3.0 years. The mean time between onset of clinical signs and admission (n = 24) was 9.3 ± 15 hours. The mean time between admission and induction of anesthesia was 2.7 ± 3.1 hours. None of these dogs required simple gastrotomy. Twenty-eight percent (8/29) of the nonsurvivors underwent partial gastrectomy. Splenic abnormalities, requiring total or partial splenectomy, were detected in 38% (11/29) of the dogs. The mean anesthetic time was 2.6 ± 1.0 hours. Forty-five percent (13/29) of the nonsurviving dogs developed cardiac arrhythmia. These were characterized as premature ventricular conductions (n = 6), paroxysmal tachycardia (n = 3), sustained ventricular tachycardia (n = 3), and atrial fibrillation (n = 1). Thirty-eight percent (5/13) of these dogs were treated with antiarrhythmic agents, whereas 62% (8/13) were left untreated. All dogs that were recovered from anesthesia (18/29) had a tube gastropexy.

Eleven of the dogs were euthanatized during surgery because of presumed massive gastric necrosis involving the cardia and fundus. One of these dogs had concurrent splenic torsion. Three of the deaths were in dogs with concurrent conditions: gastric carcinoma and multifocal myocardial necrosis, pregnancy and gastric perforation with splenic venous thrombosis, and chronic bacterial endocarditis.

The remaining 15 dogs, which were historically healthy before the acute GDV episode, had multiple postoperative complications, including aspiration pneumonia, disseminated intravascular coagulation, persistent circulatory collapse, septic peritonitis, and cardiopulmonary arrest. Coagulopathy was recognized in 5 dogs on the basis of a tendency to hemorrhage at surgery or detection of petechiae or ecchymoses, prolongation in prothrombin and partial thromboplastin times, or thrombocytopenia. There were no unexplained deaths during anesthesia. Of the 29 GDV dogs that died, 13 developed cardiac arrhythmias prior to death. Six had premature ventricular contractions, and 2 of these progressed to paroxysmal ventricular tachycardia.

There were no statistically significant differences in age of dogs, time lapse between onset of signs and admission, time between admission and induction of anesthesia, or duration of anesthesia between GDV dogs that survived and GDV dogs that died. There also was no statistical correlation between development of cardiac arrhythmias and outcome in GDV dogs.

Dogs with gastric dilatation and dogs with gastric dilatation with or without volvulus—Sixty-six cases were identified as GD and 36 cases were identified as GD ± V. Approximately 99% (101/102) of the GD and GD ± V dogs survived the acute episode. All of the dogs in the GD group underwent nonsurgical emergency treatment. All of the GD ± V dogs underwent emergency exploratory surgery and gastropexy.

Discussion

Findings in the study reported here indicated that dogs with GDV syndrome represented 0.28% of the total hospital population. This is greater than the reported prevalence of 0.13% in a study in California.[11] The reason for this disparity may lie in the overall population considered and the geographic location of the respective hospitals. The decision to use the emergency room accessions for the population in our study, despite its slightly biased nature, was made to provide perspective for the increasing number of veterinarians involved in the primary practice of emergency medicine and surgery.

Our study confirmed a breed predisposition among Great Danes, German Shepherd Dogs, and large mixed-breed dogs. Surprisingly, the Standard Poodle also was at increased risk in our study, a breed not previously reported to be at risk.[2,3,12]

The grouping of animals into 3 distinct groups (GD, GD ± V, and GDV) in this study was somewhat arbitrary and based largely on clinical management. Some investigators have used abdominal radiographic signs to make the distinction between GD and GDV,[13,14] but there is an inherent danger in making such a distinction.[2,3] The grouping used in our study does not necessarily imply differing pathogeneses. Some dogs clearly suffer from simple gastric dilatation without volvulus,[11,15] whereas others develop gastric dilatation as a consequence of volvulus.[11,16] On the basis of results of our study, it would seem that the distinction has some prognostic value because only 1 animal in the GD and GD ± V groups died.

The 15% fatality rate for dogs with GDV reported in this study compared favorably with that of other reports.[3-8,13,14,17-19,a] If GD and GD ± V dogs are included, the overall fatality rate is further reduced to 10.2% (30/

295). Some investigators have included dogs that were dead at the time they were brought in for admission and dogs that did not leave the emergency room in the determination of fatality rates.[3] During our defined study period, 29 dogs could have been included in that category. Most of these dogs were euthanatized at the owners request; a small number arrived dead or died shortly thereafter. None of these dogs received treatment at the hospital in this study. If all of these dogs had GDV, the calculated fatality would be 26% (58/222), rather than 15%. When GD and GD ± V dogs are included, the overall fatality rate would be 18.2% (59/324), rather than 10.2%. With either determination, the fatality rates in our study compared favorably with those of other studies.[3-8,13,14,17-19,a] The only records discarded from our study after the original search were those that were coded either GD or GDV, but in which there was no further mention of gastric disease. These were presumed to be errors in coding rather than errors in diagnosis. All of the remaining records contained data sufficient for inclusion in the study.

The timing of surgical intervention in canine GDV syndrome has been the subject of some controversy. Most investigators recognize restoration and maintenance of adequate tissue perfusion as the single most important goal in the management of this syndrome.[11-23,a] Frendin and Funkquist recommended a prolonged period of stabilization after complete gastric decompression.[24] Funkquist[13] has suggested that gastric decompression would result in repositioning of the stomach in half the cases. However, allowing a stomach to remain in a severely rotated position could have serious consequences.[25] Temporary flank gastrostomy also has been reported to allow for a prolonged period of stabilization prior to major surgery.[4] With this technique, the stomach could be inadvertently fixed in a rotated position. Others have recommended that emergency surgery be performed only in dogs from which blood or coffee-ground-like material was recovered during gastric lavage.[26] This recommendation is based on the assumption that the gastric mucosa will become necrotic in advance of, or at the same time as, the rest of the gastric wall. Evidence suggests that the blood flow to the gastric mucosa and submucosa is normally greater than that to the muscularis and serosa, and more importantly, may be preserved during gastric dilation for longer than the flow to the muscularis and serosa.[27,28] Concurrent vascular, splenic, or hepatic abnormalities also could influence the timing of surgical intervention in some dogs. These complications could render patient stabilization more difficult or be a primary cause of death.[a] The results of our study suggest that an initial period of circulatory resuscitation followed by rapid surgical intervention results in low fatality rates in GDV dogs.

Cardiac arrhythmias have been reported to develop in 40 to 50% of dogs with GD or GDV. The arrhythmias typically are ventricular in origin.[3,5,29-34] It has been suggested that arrhythmias are an important cause of increased morbidity and fatality in dogs with GDV.[29] Matthiesen[5] observed that arrhythmias developed predominantly among dogs undergoing gastric necrosis but were not specific for this complication. In a retrospective study of GDV,[3] Muir recognized arrhythmias in 47 of 120 dogs undergoing surgery. In that study, 11 deaths (23%) were reported in dogs with abnormalities evident on the ECG. Interestingly, the remaining 28 deaths were from a group of 115 dogs in which arrhythmias were not detected (24%). In our hospital practice, antiarrhythmic drugs are given to dogs with preexisting heart disease (eg, dilated cardiomyopathy), if the ECG abnormality is subjectively associated with poor myocardial function, or if serious electrical disturbances are evident (persistent ventricular tachycardia or R on T phenomena). Each clinician, however, makes the final decision about treatment, hence some inconsistencies exist regarding which dogs were treated and when. Results of our study concur with those of other studies in that 40.4% (78/193) of GDV dogs developed an ECG abnormality. However, there was no clear correlation between development of arrhythmias and outcome. Ventricular arrhythmias can influence cardiac output, but there is little evidence to support the contention that they can cause dogs to be at increased risk of sudden cardiac death. Treatment for ventricular ectopy has not been shown to influence the risk of sudden cardiac death in human patients.[35,36] For patients without preexisting cardiac disease, the risk of sudden death, even with sustained ventricular tachycardia, would seem to be minimal.[37] Findings in our study suggested that the fatality rate among the group of dogs with arrhythmias was not statistically different from the fatality rate among the group without arrhythmias. It should be recognized, however, that these data could be subjected to different interpretation. Without a carefully designed prospective clinical trial, the true influence of cardiac arrhythmias (and their suppression) on case outcome cannot be determined.

Gastric resection has been associated with high fatality rates, sometimes exceeding 60%.[14,19,a] Of 37 dogs diagnosed with gastric necrosis in our study, 11 animals were euthanatized during surgery, and 26 animals underwent partial gastric resection. Among dogs treated with partial gastric resection, the fatality rate was 30%. A similar rate has been reported for GDV dogs undergoing partial gastric resection by use of a surgical stapling instrument.[19] In that study,[19] the investigators attributed the improved fatality rates to reduced overall surgery time. The reasons for the improved survival after partial gastrectomy in our study are not so clear. Surgical stapling devices were not used consistently during the period studied and, therefore, cannot be the sole explanation of these results. In all cases, tissue viability was evaluated subjectively by palpation of local vessels and visual inspection of incised stomach wall. Most commonly, gastric resection was carried out until the incised surfaces of the stomach wall were actively bleeding. If gastric blood flow is greater to the mucosa and submucosa, as has been suggested,[27,28] it should be sufficient to resect stomach to a point where the gastric muscularis is actively bleeding. Despite efforts to improve objective evaluation of tissue viability during surgery, subjective observations are sometimes the most useful and practical.[38] Only 1 dog in our study died directly of complications resulting from breakdown of the gastrectomy. These results suggest that outcome following partial gastric resection is determined more by

postoperative intensive care practices than by the actual technique of partial gastrectomy. In a study by van Sluijs[a] several dogs died in persistent circulatory collapse. He commented that, although this was not good, it did offer some hope for other dogs if more aggressive circulatory support was provided. Millis, et al.[20] reported on an association between abnormal hemostatic profile results and gastric necrosis. Determination of coagulation variables in affected dogs may yield useful prognostic information. In general, dogs requiring partial gastric resection have more severe involvement of other organ systems.[5,14,20,39] It is logical to assume, therefore, that a heightened awareness of the requirements of such dogs and the ability to monitor and treat them more aggressively will result in an improved success rate.

There are many uncontrolled variables in any population of animals with a complex condition like GDV syndrome. Pinpointing a single factor that is directly responsible for the success or failure of treatment often is difficult. In the group of dogs that died after surgery in our study, no single causative factor or group of factors could be identified. The dogs that died were a heterogeneous group with various complicating diseases. There were, however, 11 dogs euthanatized during surgery because of presumed massive gastric necrosis. Gastric wall necrosis at the cardia, in particular, was considered to indicate a poor prognosis. With the improved patient aftercare and the advent of surgical stapling devices, resection of the cardia and abdominal esophagus could be considered an option in some dogs.

[a]van Sluijs FJ. *Gastric dilatation volvulus in the dog.* Thesis, Faculteit der Diergeneeskunde, Rijksuniveriteit te Utrecht, Netherlands 1987.

[b]Hespan Du Pont Pharma, Wilmington Del.

[c]TA or GIA United States Surgical Corp, Norwalk, Conn.

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