

Gastric ulceration subsequent to partial invagination of the stomach in a dog with gastric dilatation-volvulus

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Case Description—An 8-year-old castrated male German Shepherd Dog was evaluated because of abdominal distension, retching, and vomiting.

Clinical Findings—Gastric dilatation-volvulus was suspected on the basis of the dog's signalment, history, clinical signs, and results of clinicopathologic analyses and abdominal radiography. Celiotomy was performed, and gastric dilatation-volvulus was confirmed along with splenomegaly. Gastric invagination was performed over an area of gastric necrosis. The dog was reevaluated 21 days later after an episode of collapse. Findings of physical examination and clinicopathologic analyses were suggestive of internal hemorrhage. Abdominal ultrasonography and subsequent celiotomy revealed severe gastric ulceration at the gastric invagination site, splenic torsion, and a focal splenic infarct.

Treatment and Outcome—Splenectomy and gastrectomy of the necrotic tissue were performed. The dog was discharged from the hospital, and the owner was instructed to administer gastroprotectants and feed the dog a bland diet. The dog was reported to be healthy 3.25 years after surgery.

Clinical Relevance—Findings suggest that complications associated with the gastric invagination procedure include severe gastric ulceration that may require subsequent surgery. Prolonged treatment with gastroprotectants following gastric invagination surgery may be necessary to avoid gastric ulceration in dogs. (*J Am Vet Med Assoc* 2006;228:1895–1900)

An 8-year-old 34-kg (75-lb) castrated male German Shepherd Dog was evaluated at the Matthew J. Ryan VHUP because of a distended abdomen and a 1-day history of regurgitation, retching, vomiting white foam and saliva, and whining. Abnormalities detected on initial physical examination included lethargy, tachycardia (190 beats/min), pale and tacky mucous membranes, abdominal distension, and a palpably large mass in the midabdominal region. Initial IV fluid support included administration of 4 L of crystalloids for volume expansion; 240 mL of fresh, frozen plasma was thawed and also administered because of concerns about the dog's cardiovascular status and the potential for development of a coagulopathy or disseminated intravascular coagulation.

Serum biochemical analyses, a CBC, and a coagulation profile were performed. Serum biochemical abnor-

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ABBREVIATIONS

VHUP	Veterinary Hospital of the University of Pennsylvania
GDV	Gastric dilatation-volvulus
GSO	Gastric spiral organism

malities included hypoglycemia (49 mg/dL; reference range, 65 to 112 mg/dL), hyperphosphatemia (7.4 mg/dL; reference range, 2.8 to 6.1 mg/dL), hyperproteinemia (7.7 g/dL; reference range, 5.4 to 7.1 g/dL), hyperalbuminemia (4.1 g/dL; reference range, 2.5 to 3.7 g/dL), hyperlactatemia (7.3 mmol/L; reference range, 0.3 to 2.5 mmol/L), low alanine aminotransferase activity (3 U/L; reference range, 16 to 91 U/L), high aspartate aminotransferase activity (85 U/L; reference range, 23 to 65 U/L), high γ -glutamyltransferase activity (47 U/L; reference range, 7 to 24 U/L), and high creatinine concentration (2.0 mg/dL; reference range, 0.7 to 1.8 mg/dL). The CBC revealed mild, mature neutrophilia (15.1×10^3 neutrophils/ μ L; reference range, 3.10×10^3 neutrophils/ μ L to 14.4×10^3 neutrophils/ μ L), lymphocytopenia (0.475×10^3 lymphocytes/ μ L; reference range, 0.900×10^3 lymphocytes/ μ L to 5.50×10^3 lymphocytes/ μ L), an increased RBC distribution width (16.7%; reference range, 11.6% to 14.8%), and a moderate number of echinocytes. Results of the coagulation profile were within reference limits.

Right lateral thoracic and abdominal radiography revealed evidence of hypovolemia and GDV with secondary megaesophagus. To immediately relieve gas distension, an 18-gauge needle was used as a trocar and inserted into the stomach. Following mild sedation achieved via administration of diazepam (0.25 mg/kg [0.11 mg/lb], IV) and hydromorphone (0.1 mg/kg [0.05 mg/lb], IV), an unsuccessful attempt to pass a stomach tube was made. A 16-gauge needle was then used as a trocar and inserted into the stomach; on this attempt, gas and 450 mL of malodorous, dark-brown fluid were removed. The dog was then prepared for surgery.

Anesthesia was induced with diazepam (0.5 mg/kg [0.23 mg/lb], IV), hydromorphone (4.0 mg/kg [1.82 mg/lb], IV), and lidocaine (2.0 mg/kg [0.91 mg/lb], IV) and maintained with isoflurane (1.5% to 4.0%) in oxygen. The abdomen was opened via a ventral midline incision. Surgical exploration of the abdomen confirmed GDV (Figure 1). The palpable mass was the spleen, which was congested; on closer inspection, the organ appeared viable with adequate venous drainage. The stomach was repositioned, an orogastric tube was passed, and the stomach contents were evacuated. Viability of approximately 10% of the stomach wall along the greater curvature of the fundus was uncertain

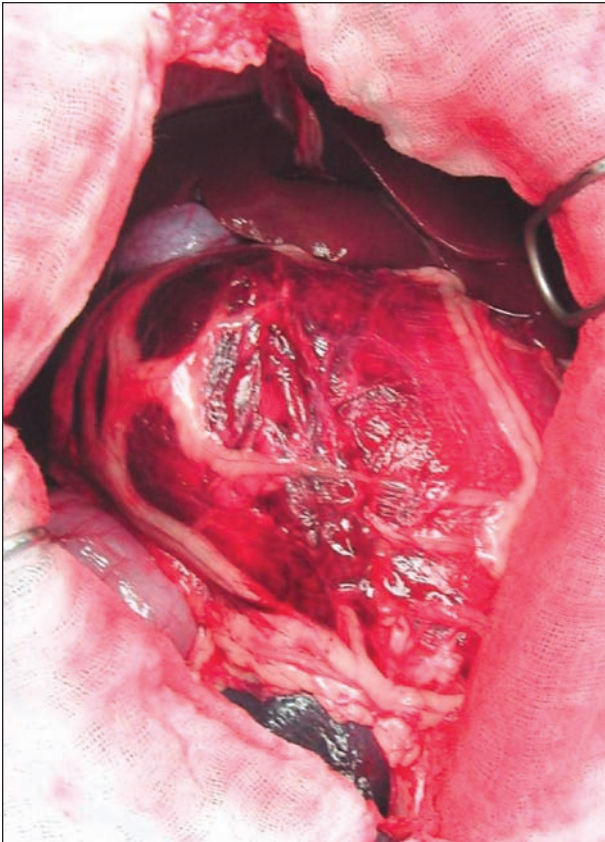


Figure 1—Photograph obtained during exploratory laparotomy performed in a German Shepherd Dog that had clinical signs suggestive of GDV. Notice the distended, omentum-covered stomach with a dark, discolored gastric wall. The top of the image is toward the dog's head.

because of tissue discoloration and palpable thinning (Figure 2). Invagination of the thin region of the gastric wall and a surrounding margin of healthy tissue was performed with 2 layers of 2-0 polydioxanone suture in a continuous, inverting Lembert pattern (Figure 3). An incisional gastropexy was performed to secure the pyloric antrum to the right body wall. Complete, routine abdominal exploration did not reveal further abnormalities, and the incision was closed in a routine fashion.

The dog recovered from anesthesia without complications. Continued postoperative care included administration of hydromorphone (0.05 mg/kg [0.023 mg/lb], IM, as needed) for pain control and IV administration of a crystalloid solution (5 mL/kg/h [2.27 mL/lb/h]). Because gastric ulceration is a known adverse effect of the gastric invagination procedure,¹ ranitidine (2 mg/kg, IV, q 8 h) and sucralfate (1 g slurry, PO, q 6 h) were administered to decrease gastric acid secretion and act as a gastric wall protectant, respectively. The dog was discharged 2 days after surgery; at that time, it had a good appetite and treatment with medications had been discontinued.

Twenty-one days after surgery, the dog was evaluated by a referring veterinarian because of a 2- to 3-day history of listlessness, inappetence, and weakness that culminated in collapse. Physical examination by the referring veterinarian revealed that the dog had lost 3.1

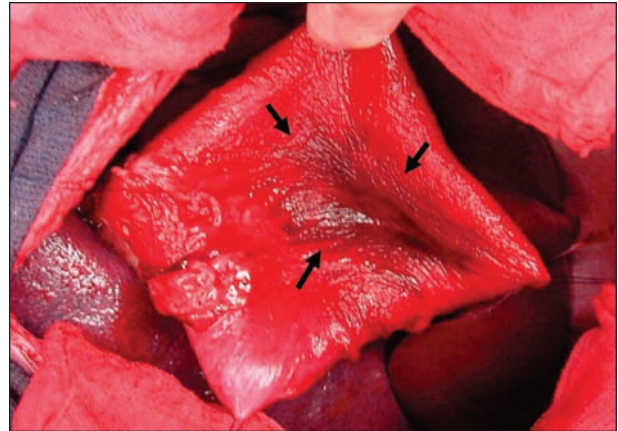


Figure 2—Photograph obtained during surgical treatment of GDV in the dog in Figure 1. Following gastric derotation and decompression, a focal area of thin, discolored gastric wall (black arrows) is evident along the greater curvature of the fundus. The bottom of the image is toward the dog's head.

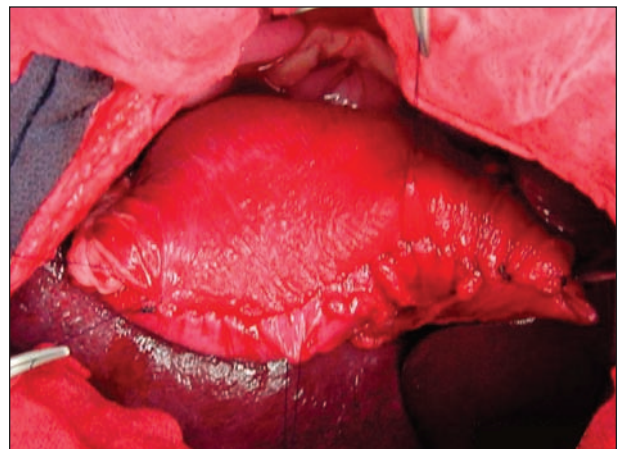


Figure 3—Photograph obtained after invagination of the compromised gastric wall in the dog in Figure 1. The healthy edges of the gastric wall have been apposed. The bottom of the image is toward the dog's head.

kg (6.82 lb); mucous membranes were pale, and a palpable mass was detected in the midabdominal region. Serum biochemical abnormalities at that time included hypoproteinemia (4.5 g/dL; reference range, 5.0 to 7.4 g/dL), hypoalbuminemia (2.1 g/dL; reference range, 2.7 to 4.4 g/dL), low alanine aminotransferase activity (8 U/L; reference range, 12 to 118 U/L), and low creatine kinase activity (52 U/L; reference range, 59 to 895 U/L). A CBC performed by the referring veterinarian revealed regenerative anemia characterized by low hemoglobin concentration (7.4 g/dL; reference range, 12.1 to 20.3 g/dL), low Hct (24.5%; reference range, 36% to 60%), and low RBC count (3.4×10^6 RBCs/ μ L; reference range, 4.8×10^6 RBCs/ μ L to 9.3×10^6 RBCs/ μ L) with reticulocytosis (2.3%; reference range, 0.2% to 1.0%). The dog was admitted for monitoring, and lactated Ringer's solution was administered IV. Right and left lateral thoracic and abdominal radiography revealed hyperinflated or hypoperfused lungs and a mild pneumoperitoneum consistent with recent abdominal surgery. Abdominal ultrasonography revealed a hypoechoic area at the tail of the spleen.

Subsequent blood analyses performed by the referring veterinarian revealed a further decrease in the Hct (13%), although serum total protein concentration (5.3 g/dL) was within reference limits. The dog was immediately referred to the VHUP for further evaluation and treatment.

On arrival at the VHUP, the dog was alert, mildly hyperthermic (rectal temperature, 39.7°C [103.5°F]), and tachycardic (120 beats/min) with intermittent ventricular premature contractions and occasional periods of ventricular tachycardia. Pulse quality was poor, and mucous membranes were pale and moist with a capillary refill time of 1 to 2 seconds (reference range, < 2 seconds). Melena was detected via rectal palpation. Abnormal results of serum biochemical analyses performed at the VHUP included hypoproteinemia (3.8 g/dL), hypoalbuminemia (1.5 g/dL), hypocalcemia (8.8 mg/dL; reference range, 9.8 to 11.7 mg/dL), low alanine aminotransferase activity (12 U/L), low alkaline phosphatase activity (18 U/L; reference range, 24 to 174 U/L), low total bilirubin concentration (0.1 mg/dL; reference range, 0.3 to 0.9 mg/dL), and low cholesterol concentration (97 mg/dL; reference range, 128 to 317 mg/dL). A CBC revealed microcytic (mean corpuscular volume, 61.0%; reference range, 62.7% to 75.6%), hypochromic (mean corpuscular hemoglobin, 20.1%; reference range, 22.5% to 26.9%), poorly regenerative anemia characterized by low Hct (15.8%) and reticulocytosis (2.1%; reference range, 0.2% to 1.0%). Additionally, mature neutrophilia (21.7×10^3 neutrophils/ μL) and lymphocytopenia (0.663×10^3 lymphocytes/ μL) were detected. Results of a coagulation profile were within reference limits. Following a transfusion of 2 units of packed RBCs (16 mL/kg [7.27 mL/lb]), the Hct increased to 28%, mucous membranes were pink, and pulse quality improved.

Thoracic radiography revealed a small volume of pleural effusion. Abdominal radiography identified a loss of abdominal detail consistent with a peritoneal effusion and possible splenomegaly. Abdominal ultrasonography identified a splenic infarct secondary to a thrombus at the tip of the spleen, edema in the fundic portion of the stomach consistent with the recent gastric surgery and involution, and a mild peritoneal effusion. Gastric ulceration and splenic infarction were primary differential diagnoses, on the basis of which the decision to pursue exploratory laparotomy was made. Prior to the laparotomy, endoscopy of the upper portion of the gastrointestinal tract (esophagus and stomach) was performed to identify any ulceration that might not be visible during exploratory laparotomy.

Anesthesia was induced with hydromorphone (0.5 mg/kg), lidocaine (2.0 mg/kg), diazepam (0.5 mg/kg), and etomidate (0.4 mg/kg [0.18 mg/lb]) and maintained with isoflurane^a (3% to 4%) in oxygen. Gastrointestinal endoscopy revealed a large focal area of gastric ulceration. Exploratory laparotomy revealed a splenic torsion with a focal splenic infarct. A total splenectomy was performed. Stay sutures were placed in the stomach wall, and a 5-cm gastrotomy incision was made along the ventral surface of the fundus adjacent to the palpable thickening at the site of the previous gastric invagination. A 5 × 3-cm gastric ulcer was

identified at the previously invaginated area of the stomach (Figure 4). The ulcerated area was resected, and the stomach was closed in 2 layers; by use of 2-0 polydioxanone suture, the mucosa and submucosa layers were closed with a simple continuous suture pattern and the seromuscular layer was closed with a continuous, inverting Lembert suture pattern. Samples of splenic tissue and the resected ulcerated tissue were submitted for histologic evaluation. Abdominal exploration also identified abnormal positioning of the left kidney. The kidney was lacking the typical peritoneal covering and was therefore no longer contained within the retroperitoneal space. Prior to routine abdominal closure, a renopexy was performed to position the left kidney next to the left abdominal wall.

Histologic analysis of the splenic tissue confirmed marked congestion and lymphocytolysis indicative of necrotic changes most likely secondary to anoxia, which suggested that complete occlusion of the arterial supply had occurred. Examination of the gastric biopsy specimen confirmed a gastric ulcer; the transition from the area of intact mucosa to ulcerated tissue was abrupt. Spiral bacteria were found within the mucin. The lamina propria had a minimal amount of inflammatory infiltrate consisting of neutrophils and fibrin, and myofiber degeneration with interstitial fibrosis was present. Some vessels contained fibrin thrombi and evidence of necrotizing vasculitis. No neoplastic tissue was identified.

Postoperative care included administration of oxymorphone (0.1 mg/kg, IM, as needed) for pain control and crystalloid (1.5 mL/kg/h [0.68 mL/lb/h], IV) and colloid support (1 mL/kg/h [0.45 mL/lb/h], IV). Cimetidine (6.5 mg/kg [2.95 mg/lb], PO, q 8 h) and sucralfate (32 mg/kg [14.55 mg/lb], PO, q 6 h) were administered to reduce gastric acid secretion and as a gastric wall protectant, respectively. The melena, ventricular premature contractions, and pulse deficits resolved within approximately 24 hours. The dog was discharged from the hospital 3 days after surgery; at that time, it was mildly anemic (Hct, 23% to 26%). The owner was instructed to administer cimetidine (6.5 mg/kg, PO, q 8 h) and sucralfate (32 mg/kg, PO, q 6 h)



Figure 4—Photograph obtained during exploratory laparotomy and gastrotomy performed 21 days after completion of the gastric invagination surgery in the dog in Figure 1. Notice the severe mucosal ulceration that was exposed via eversion of the previously invaginated stomach wall. The right of the image is toward the dog's head.

for 14 days and feed the dog a bland diet (small, frequent meals) for 3 weeks. The client was contacted approximately 3.25 years after the second surgery for follow-up information; the owner reported that the dog's activity level and behavior had returned to normal within 1 week after surgery and that no subsequent medical problems had developed.

Discussion

Gastric dilatation-volvulus is a surgical emergency in dogs and can result in varying degrees of hypovolemic or cardiogenic shock.^{1,6} In affected dogs, the development of a tense, distended stomach results in reduced cardiac return secondary to caudal vena cava and portal vein compression, respiratory acidosis caused by respiratory tract compression, and metabolic acidosis caused by lactic acid production associated with vascular stasis.^{1,5,6} Gastric vascular compromise results in gastric ischemia, which may lead to disturbed gastric motility; gastric gas and fluid accumulation; infarction, ulceration, necrosis, and, ultimately, perforation of the gastric wall; and peritonitis.^{1,4,8} Splenic vascular congestion resulting from GDV can lead to the development of splenomegaly, splenic thrombosis, and possible splenic torsion.⁵ Avulsed gastric branches of the splenic arteries can also result in hemoperitoneum.^{3,5} The etiology of GDV is most likely multifactorial, but dysfunctions of the gastroesophageal sphincter and pyloric outflow mechanism are prerequisites to the development of gastric dilatation, whether it precedes or follows volvulus of the stomach.^{1,3,5,9} Risk factors for development of GDV in dogs include large or giant breed, a first-degree relative with a history of GDV, increasing age, a large thoracic depth-to-width ratio, stress, and diet or feeding behavior.^{1,2,5,8,10} Gastric dilatation-volvulus can be a fatal condition with mortality rates of 11% to 90%.^{4,7,8,10} Higher mortality rates have been associated with gastric necrosis and range from 46% to 90%.^{4,7,8,11} Other reported risk factors for increased mortality rates associated with GDV have included splenectomies, cardiac arrhythmias, and increased serum lactate concentration.^{7,11}

During emergency stabilization of a dog with GDV, gastric decompression is recommended via orogastric tube placement or trocar insertion. Subsequent treatment of GDV warrants immediate surgical intervention to correct gastric positioning (thereby preventing the development of additional gastric ischemia), identify and address previous compromise of the gastric wall, and perform gastropexy to prevent the volvulus from recurring. It is generally accepted that necrosis of the stomach is identified grossly by color, thickness, and integrity of the gastric wall.^{1,3,5,7,8} The most commonly described procedures to treat nonviable tissue are partial gastrectomy or gastric invagination.

Partial gastrectomy has been associated with a high incidence of postoperative complications and mortality rates (reported to be as high as 63% to 90%^{4,14}); however, it is unclear whether these results are associated with the gastrectomy procedure or the underlying gastric wall necrosis and systemic consequences.^{6,8,12} The most common and devastating complication associated with partial gastrectomy is peri-

toneal contamination, which can result in sepsis, septic shock, and subsequent death.^{1,4,13,14} Contamination may occur during the procedure itself or develop after closure through suture dehiscence. Subsequent or progressive gastric necrosis, gastric perforation, ventricular arrhythmia, hypoproteinemia, hypokalemia, and anemia are other complications reported to develop in dogs with GDV that are treated with this procedure.^{3,14}

Although partial gastrectomy has historically been the preferred surgical technique to eliminate nonviable gastric tissue resulting from GDV, gastric invagination is included in the veterinary surgical literature as an acceptable treatment for gastric wall infarction or necrosis and does have theoretical advantages.^{1,13} Continuous inverting sutures are placed in viable gastric tissue beyond margins of the ischemic tissue, which is concurrently inverted into the gastric lumen. Sutures are tightened to allow apposition of the stomach wall via placement of a second layer of continuous inverting sutures, if possible.¹ The goal of this procedure is sloughing and subsequent digestion of the devitalized tissue into the gastric lumen as the apposed healthy margins of tissue heal together. Gastric invagination does not require entry into the stomach lumen, and the risk of suture dehiscence is theoretically less likely to result in peritoneal contamination. Compared with partial gastrectomy, invagination is associated with decreased duration of anesthesia and is less technically demanding.^{1,13}

In an experimental study,¹³ gastric vascular compromise was induced in 21 healthy dogs via multiple short gastric artery ligations (group 1) or complete vascular isolation (group 2) before gastric invagination was performed on the isolated stomach wall. The dogs were euthanized 14 days after surgery. In the 13 dogs in group 1, tissue compression from the invagination and tissue infarcts from the ligated vessels resulted in deep liquefactive areas of necrosis in 9 dogs and granulation tissue and mucosal ulcers in 7 dogs; 1 dog had a large, deep, fibrinous ulcer of the invaginated segment. Of the 8 dogs in group 2, 4 had melena and 7 had evidence of substantial sloughing of the invaginated tissue. Most of the invaginated gastric segments had large ulcers covered by fibrin, necrotic debris, and neutrophils; destroyed muscularis mucosa; and dense granulation tissue and inflammatory cells extending to the serosal layer. The authors concluded that healing and the long-term effects on function of gastric tissue after invagination require further evaluation.¹³

The gastric ulcer that developed in the dog of this report was much more severe than those discussed in the aforementioned report¹³; the ulcer was diagnosed 24 days after surgery, whereas dogs in the experimental study were not evaluated for longer than 14 days after surgery. Excessive bleeding in the dog of this report likely resulted from erosion and ulceration of gastric mucosa, which exposed patent branches of the left gastroepiploic artery. Because the area of gastric wall in which ischemia most commonly develops is along the greater curvature of the gastric fundus, the left gastroepiploic artery is commonly involved in the ischemic segment. Given that the purpose of invagination is to permit intraluminal sloughing of ischemic

tissue, it may be prudent to consider ligation of the portion of the left gastroepiploic artery that will be invaginated with the ischemic segment of stomach wall. Following the anticipated sloughing, this may reduce the chance of subsequent uncontrolled arterial hemorrhage. Aggressive and prolonged prophylactic treatment with gastroprotectants may also be considered because of the potential for gastric ulceration following gastric invagination surgery. In the dog of this report, ranitidine and cimetidine were used as gastric acid secretion inhibitors after the first and second surgeries, respectively. Other gastric acid inhibitors (eg, famotidine and nizatidine) would also be appropriate unless contraindicated in a particular patient. Ant ulcer drugs, such as sucralfate (which was used in the dog of this report), can aid in mucosal healing.

Splenic torsion in the absence of GDV is uncommon and generally occurs in large deep-chested dogs.^{1,12,15-17} Specifically, it is most commonly reported in Great Danes, Saint Bernards, and German Shepherd Dogs.¹⁵ In 1 retrospective study¹⁸ of 19 cases of isolated splenic torsion, German Shepherd Dogs represented approximately one fourth of cases and were found to be at significantly increased risk, compared with the hospital population; 15 of the 19 affected dogs were male. The most common clinical signs in dogs with splenic torsion are nonspecific, such as anorexia, malaise, lethargy, vomiting, and signs of depression.¹⁵⁻¹⁸ Mild anemia, high WBC count, hemoglobinemia, and hemoglobinuria are commonly identified clinicopathologic abnormalities.

The exact etiology of isolated splenic torsion is unknown. It has been hypothesized that splenic torsion is preceded by an acute occurrence of gastric dilatation. The gastrosplenic ligament or splenocolic ligament (or both) is disrupted by the gastric dilatation, which may cause the spleen to rotate. Even if the dilatation subsides, the spleen may remain in a rotated, abnormal position.^{5,12,15} Splenic torsion typically involves occlusion of the splenic vein and results in splenomegaly rather than infarction; however, congestive splenomegaly may lead to venous thrombosis.^{1,12,15,18} Although splenic infarction is uncommon, areas of splenic infarct may develop if the arterial supply is occluded.^{1,15} In the dog of this report, splenic infarction could have developed secondary to the splenic torsion or possibly resulted from occlusion of the gastroepiploic artery during the invagination procedure, particularly if the remaining splenic arterial supply was compromised. The redundant blood supply to the spleen and the fact that the infarct was located in the tail of the spleen suggest that occlusion of the gastroepiploic artery was an unlikely cause of the splenic infarct.

In humans, the association of GSOs, specifically *Helicobacter pylori*, with increased gastric acid production and secretion leading to gastroduodenal ulcer disease is well documented.¹⁹⁻²³ The severity of disease in humans depends on individual host factors and the pathogenicity of the species of GSOs.²³ Studies have revealed that *Helicobacter*-like GSOs are present in 100% of clinically healthy dogs,²² 100% of healthy research Beagles,^{22,24} 61% of clinically abnormal dogs,¹⁹ 95% of dogs with gastritis,²⁰ and 61% to 95% of vomiting dogs.²² Gastric spiral organisms reside in surface

mucus, glandular lumina, and parietal cells, typically in the cardia and fundus of healthy dogs and those with signs of gastritis.^{20,22} The importance of gastric infection with *Helicobacter*-like GSO in dogs is unclear and controversial.¹⁹⁻²¹ Unlike findings in humans, studies²¹⁻²⁵ in dogs to date have not revealed evidence to suggest a relationship of *Helicobacter* infection to changes in gastric function, gastric acid secretion, or development of gastrointestinal ulcers.

A review of the literature did not identify any information on GDV or splenic torsion associated with abnormal positioning of the kidneys. In the dog of this report, renopexy of the left kidney was performed to prevent subsequent torsion of the vascular pedicle.

Because of life-threatening sequelae that are reported with GDV, the general recommendation is to treat an affected dog as a surgical emergency. Surgery was performed in the dog of this report as soon as the diagnosis was made and the patient was adequately stabilized. Invagination of the thin, presumably ischemic gastric tissue was performed instead of gastric resection to reduce the duration of anesthesia and the risk of peritoneal contamination. To the authors' knowledge, this incident is the first reported complication associated with the gastric invagination technique performed in a clinical situation to treat suspected gastric necrosis associated with GDV in a dog. The fact that the dog recovered fully without specific treatment for *Helicobacter* infection and findings of a review of the literature support the authors' conclusion that the presence of spiral bacteria, most likely a *Helicobacter* sp, in the dog of this report was probably not a complicating factor. Additionally, the dog was not affected by other common causes of gastric ulceration including liver disease, treatment with nonsteroidal anti-inflammatory drugs, pancreatitis, hyperparathyroidism, delayed gastric emptying, or neoplasia.¹ Further investigation of the use of gastric invagination in conjunction with GDV surgery must be performed to better determine whether the benefits outweigh the risks for this technique, compared with treatment via partial gastrectomy.

- a. Iso-Flo, Abbott Laboratories, North Chicago, Ill.
- b. Zantac, GlaxoSmithKline, Research Triangle Park, NC.
- c. Amidate, Abbott Laboratories, North Chicago, Ill.
- d. Numorphan, Schering-Plough, Union, NJ.

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Selected abstract for JAVMA readers from the American Journal of Veterinary Research

Evaluation of isoflurane and sevoflurane vaporizers over a wide range of oxygen flow rates
Tamas D. Ambrisko and Alan M. Klide

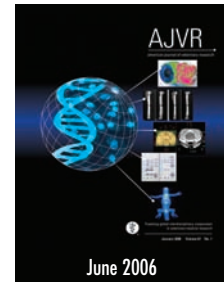
Objective—To examine the accuracy and precision of isoflurane and sevoflurane anesthetic vaporizers.

Sample Population—5 identical isoflurane and 5 identical sevoflurane vaporizers.

Procedures—Oxygen flow rates from 0.02 to 10 L/min were used with different vaporizer dial settings. Agent concentrations were measured at the common gas outlet by use of a refractometer. Accuracy was defined as the difference between measured agent concentrations, and dial settings were expressed as a percentage of the applied dial settings. Precision was defined as SD of the measured agent concentrations for each combination of dial setting and flow.

Results—Isoflurane values were generally greater than the dial settings. Accuracy of the isoflurane vaporizer was > 20% when 0.6% and 1% was dialed. Accuracy of the sevoflurane vaporizer was always within \pm 20% but decreased at 0.02 L/min flow and at combinations of high flow and high dial settings. Overall precision of the isoflurane vaporizer was better than that of the sevoflurane vaporizer.

Conclusions and Clinical Relevance—A possible explanation for the inaccuracy of the isoflurane vaporizer may be that it was manufactured to be accurate with air but not oxygen, which must be accounted for when using the vaporizer with oxygen, especially with nonbreathing systems. The sevoflurane vaporizer may not deliver accurate agent concentrations at high flow and high dial settings. Both vaporizers are suitable for clinical use with a wide range of oxygen flow rates if these precautions are properly addressed. (*Am J Vet Res* 2006;67:936–940)



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