Influence of dietary composition on gastric emptying and motility in dogs: Potential involvement in acute gastric dilatation

Colin F. Burrowe, B Vet Med, PhD; Ronald M. Bright, DVM, MS; Crispin P. Spencer, DVM

SUMMARY

Gastric dilatation-volvulus is a dramatic and life-threatening disease of large-breed dogs. The cause is unknown, but ingestion of dry cereal-based food is frequently incriminated as a predisposing factor. The purpose of this study was to examine the effect of commercial diets on gastric motility and emptying in the dog. Four large-breed dogs were fed 3 different diets (diet A = canned meat-based; B = cereal-based with 77% added water; and C = dry cereal-based) in a randomized block design. Motility was assessed, using 6 AgAgCl electrodes and 2 strain gauges sutured along the serosa of the stomach and proximal duodenum. Dogs were fed at the same time each day and the time to change from the fed to the fasted pattern of gastrointestinal motility (changeover) was measured. Gastric emptying was assessed by recording gastric radioactivity. After feeding a meal mixed with 14C-labeled resin, the log of activity was plotted against time, and the half-time of gastric emptying (t½ GE) was calculated. Mean (±SEM) times from feeding to changeover for the 3 diets were: diet A, 9.7 ± 0.9; B, 10.5 ± 0.4; and C, 11.0 ± 0.8 hours. Diet had minimal influence on the half time of gastric emptying (diet A, 2.2 ± 0.3; B, 2.6 ± 0.4; and C, 2.9 ± 0.3 hours; P > 0.05). The data indicate that gastric motility and emptying in healthy large-breed dogs were not affected by dietary composition. Because most large dogs are fed cereal-based food for reasons of cost and ease of use, these diets may have been wrongly incriminated as a predisposing factor in gastric dilatation-volvulus.

Gastric dilatation-volvulus (GDV) is a dramatic disease in which affected dogs are frequently presented in severe distress, many of which require surgical intervention. Even with immediate veterinary care, however, the prognosis is often poor and mortality is high. Although the gross pathologic features of GDV,1 surgical aspects of treating the disease,2,3 and the pathophysiologic events that develop after dilatation4,6 have been described, little is known of the etiopathogenesis of the GDV syndrome. Despite intense study and speculation, no single factor has yet emerged as to the cause of GDV, and it is generally agreed that the cause is multifactorial.7 The most frequently incriminated predisposing factor appears to be feeding dry cereal-based foods in 1 large daily meal. Engagement of dry, easily fermentable foodstuffs, with subsequent gas accumulation, failure of the normal evacuation and vomiting mechanisms, and inhibition of gastric emptying (GE) has been associated with many cases of GDV.8

The source of the gas responsible for gastric dilatation remains controversial. Some studies have indicated that gas produced by bacterial fermentation of dietary carbohydrate is the major source,4,8 whereas others have incriminated swallowed air.10 Aerophagia has been reasoned to be the source in most patients.8 Another hypothesis is that the interaction of a greedy eater, fermentative gastric flora, readily fermentable substrate, management practices, or antecedent gastric disease may combine to permit excessive intragastric fermentation,8 particularly by clostridia.9

Bacterial fermentation of carbohydrate also produces volatile fatty acids that inhibit gastric motility and emptying in the ruminant.11 The relevance of ruminant studies to GDV in dogs revolves around the question of whether, because of their physical composition, dry cereal-based dog foods are emptied more slowly from the stomach than are isocaloric quantities of meat-based foods. This delay could permit increased fermentation with production of gas and metabolic byproducts. These byproducts could, in turn, delay GE, cause nausea and aerophagia, and initiate a vicious cycle that may lead to dilatation in susceptible animals.

Purposes of the present study were to evaluate the effect of commercial diets on gastric motility and emptying in large-breed dogs and to determine whether there is a physiologic basis for incrimination of cereal-based products as a predisposing factor in GDV.

Materials and Methods

Dogs—Four healthy female large-breed dogs (1 German Shepherd Dog, 1 Golden

---

Received for publication Apr 15, 1985.
From the College of Veterinary Medicine, Department of Medical Sciences, University of Florida, Gainesville, FL 32610. Dr. Bright's current address is Department of Urban Practice, Box 1071, University of Tennessee, Knoxville, TN 37993.


Published as University of Florida journal series No. 37.

The authors thank Drs. N. Ackerman, N. Guibas, A. M. Merritt, and M. C. Theodorakis for advice and assistance, and D. Moses, J. Kaufman, P. Mallison, and S. MacNamara for technical assistance.


2609
TABLE 1—Composition of diets

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Diet A analysis</th>
<th>Diet C analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Protein</td>
<td>58.5</td>
<td>52.5</td>
</tr>
<tr>
<td>Fat</td>
<td>24.6</td>
<td>26.5</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>7.8</td>
<td>9.5</td>
</tr>
<tr>
<td>Fiber</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Ash</td>
<td>11.2</td>
<td>8.9</td>
</tr>
<tr>
<td>Moisture</td>
<td>76.8</td>
<td>76.6</td>
</tr>
</tbody>
</table>

* Laboratory 1 included fiber in nitrogen-free extract.

Constituents are expressed as percentages of dry matter, except moisture; moisture is expressed as percentage as fed. Carbohydrate (nitrogen-free extract) is the remainder after subtracting protein, ash, and fiber, from 100. Fiber was measured as crude fiber. Diet B was identical to diet C except water was added to 77% in diet B.

Analysis 1 was provided by ABC Research Corp., Gainesville, Fla; analysis 2 was provided by Dr. CA. Ranta, Alpo Petfoods Inc., Allentown, Pa.

1 week after surgical implantation of the electrodes and strain gauges that were used to measure gastrointestinal motility. The logarithm of relative radioactivity in the stomach was plotted against time, and the t4/3 GE was calculated from the slope of the graph.12

Gastrointestinal motility—Gastrointestinal myoelectrical activity was measured, using 5 monopolar Ag-AgCl electrodes embedded in an small plate of dental acrylic.11 Three electrodes were sutured to the serosal surface of the stomach and 2 to the proximal duodenum in a line 2 to 3 cm apart along the greater curvature of the stomach and along the anterior border of the duodenum. The most proximal electrode was 6 to 8 cm from the pylorus. Contractile activity was monitored, using strain gauge force transducers10 sutured in the circular planes adjacent to the 3rd and 4th electrodes. Wires from the electrodes and strain gauges led to a microcomputer (model MDI-15PL) embedded in a stainless steel cannula implanted in the dog's left flank. A reference (ground) electrode was placed subcutaneously in the ventral abdominal wall and connected to the microcomputer. Shielded wires led from a matching microconnector (model MDI-15SSL) to a type 9585A coupler12 to a physiograph.13 A belt pneumographe around the thorax allowed for simultaneous recording of respiration. Measurements began 14 days after surgery. Myoelectrical activity was recorded with the low-frequency cutoff at 5.3 Hz and the high-frequency cutoff at 100 Hz. Data were digitized and stored in a modified Pavlov seng for each recording period.

For measurement of motility, the routine 4 pm feeding was withheld on the day before each experiment. Dogs were fed 40 Kcal/kg at 7 AM on the morning of each experiment; measurements commenced 4 to 5 hours later. Recordings were made until the changeover from a fed to a fasted pattern was observed. To determine this point, recording was continued until observance of the first activity front (phase 3) of the migrating motility complex (MMC), which characterized the fasting state, was observed. Phase 3 of the MMC was always preceded by a period of irregular spiking (phase 2 of the MMC) and a period of spiking or contractile activity (phase 1 of the MMC). The last period of irregular activity before this quiescent period (phase 1) was defined as the actual point of changeover. Myoelectrical and contractile activity were examined concurrently in the stomach and proximal duodenum to facilitate determination of changeover.

Data analyses—The t4/3 GE and the time from feeding to changeover were compared for each dog and diet, using 2-way analysis of variance and the SAS user's guide for 1982. To examine the influence of surgery on GE, data from the 3 evaluations before and 3 evaluations after emptying were compared, using the Student's t test. All data were expressed as the mean ± SEM and were considered significant if P < 0.05.

Results

Gastric emptying—When fed isocalorically, canned meat-based food, dry cereal-based food, and cereal-based food mixed with water emptied from the stomach at the same rate (Table 2). The mean t4/3 GE progressively increased in the order: (1) canned meat-based, (2) dry cereal-based plus water, and (3) dry cereal-based (Table 3), but the increases were not significant. There was, however, a significant variation between each dog fed the same diet (P < 0.01; Table 3). Surgery had no significant effect on the rate of emptying.

Gastrointestinal motility—Dietary composition had no effect on the duration of the fed pattern of motility (Table 4). The time from feeding to changeover progressively increased from (1) canned meat-based, (2) dry cereal-based plus water, and (3) dry cereal-based (Table 4), but the increases were not significant. There was also a wide variation in the time to changeover between individual dogs (P < 0.01; Table 4).

TABLE 2—Effect of diet on half-time of gastric emptying

<table>
<thead>
<tr>
<th>Dog</th>
<th>Diet</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.9 ± 0.9</td>
<td>5.3 ± 1.2</td>
<td>4.0 ± 0.3</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.9 ± 0.3</td>
<td>2.0 ± 0.9</td>
<td>1.0 ± 0.3</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2.6 ± 0.3</td>
<td>2.6 ± 0.7</td>
<td>5.1 ± 0.3</td>
<td></td>
</tr>
</tbody>
</table>

For technical difficulties, only 3 of the 4 dogs were used for this part of the study. Numbers with different lettered superscripts are significantly different (P < 0.05) from one another. There is a significant difference between each dog, but not between the 3 diets. Data are expressed as the mean ± SEM time in hours.

TABLE 3—Influence of surgery on half-time of gastric emptying

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Diet</th>
<th>Time in hours</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre surgery</td>
<td>Post surgery</td>
</tr>
<tr>
<td>1</td>
<td>3.8 ± 0.6</td>
<td>2.9 ± 0.3</td>
</tr>
<tr>
<td>2</td>
<td>3.8 ± 0.6</td>
<td>2.0 ± 0.3</td>
</tr>
<tr>
<td>3</td>
<td>2.4 ± 0.6</td>
<td>2.6 ± 0.3</td>
</tr>
<tr>
<td>4</td>
<td>3.8 ± 0.6</td>
<td>2.0 ± 0.3</td>
</tr>
</tbody>
</table>

Surgery had no effect on the rate of emptying. Data are expressed as the mean ± SEM.

TABLE 4—Influence of diet on duration of the fed pattern of gastrointestinal motility

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Diet A</th>
<th>Diet B</th>
<th>Diet C</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>14.1 ± 0.6</td>
<td>11.9 ± 0.7</td>
<td>12.4 ± 1.4</td>
</tr>
<tr>
<td>2</td>
<td>9.5 ± 1.0</td>
<td>10.4 ± 0.3</td>
<td>11.7 ± 0.5</td>
</tr>
<tr>
<td>3</td>
<td>9.5 ± 0.9</td>
<td>9.5 ± 0.9</td>
<td>8.5 ± 0.9</td>
</tr>
<tr>
<td>4</td>
<td>6.4 ± 0.3</td>
<td>9.6 ± 0.5</td>
<td></td>
</tr>
</tbody>
</table>

Differences were significant (P < 0.05) between each dog, but not between diets. Data expressed as the mean ± SEM time in hours.

Discussion

Results of the present study indicate that the physicochemical composition of the diet does not influence the rate of GE or the duration of the fed pattern of canine gastrointestinal motility. This renders unlikely the hypothesis that dietary composition influences gastric function and predisposes to dilatation.

Mechanisms that control GE are complex; acidity, osmolarity, and fat or protein content of the diet all influence the rate of GE. Liquids are also emptied more rapidly than are solids, which must be broken down to particles < 1 mm in diameter before they can pass through the pylorus. It was for all these reasons that we speculated that 2 meals of entirely different physicochemical composition, such as canned meat-based food and dry cereal-based food, may leave the stomach at different rates. However, Hunt and Stubbs demonstrated that the rate of emptying of a meal in people can be predicted by its nutrient density, and since the meals in our study were isocaloric, even though differing in volume, it is perhaps, in retrospect, not surprising that we were unable to identify any difference in the rate of emptying. The total time taken for meals to empty (2 to 3 hours) would probably provide ample time for gastros contraction and antral contractions to neutralize differences in physicochemical composition.

A variety of techniques have been used to assess GE, including intubation and periodic sampling, contrast radiography, and radioisotope techniques, using external scanning. Intubative techniques have the disadvantage of only allowing the study of liquid meals. Radiologic methods that use liquid contrast materials have the same disadvantage; barium food mixtures are only useful for the detection of gross abnormalities and preclude accurate quantification of emptying. Radioisotopes offer a relatively easy, noninvasive, and accurate method for the study of GE. However, radioisotopes do not take into account the fraction of the isotope that is released into the liquid phase due to elution of the label or grinding. The resin technique used in the present study overcame this problem and included emptying of the total meal because technetium (99mTc) is tightly bound to the resin, which, due to its particle size (< 0.5 mm), is well dispersed throughout the meal. Previous studies of GE in the dog have not involved the use of a commercial diet; however, individual ingredients such as fat, carbohydrate, protein, and liquid have been examined. Gastric emptying values were relatively consistent for each dog (Table 1). The significant difference between GE in each dog has also been reported by others, which highlights the problems associated with developing normal criteria for evaluation of GE in dogs. Fear or excitement probably did not induce delayed GE; the dogs were accustomed to the technique, each having been used in several pilot studies before measurement began, and they usually slept in the slings. However, even if acclimatization was a factor, the experimental design precluded bias in favor of a particular diet.

Feeding disrupts the fasted pattern of intestinal motility in the dog and initiates a different pattern that lasts for 8 to 14 hours. A linear relationship exists between the quantity of food ingested (expressed as kilocalories per kilogram of body weight) and the duration of MMC disruption. Duration of the fed state also depends on dietary composition. When fed isocalorically, isolated nutrients such as arachis oil, sucrose, and milk protein, induce a feeding pattern for periods that are related to the nature of the food, with arachis oil having the greatest effect. Therefore, we assumed that 2 commercial dog foods (1 with a fat content of 26% and the other of 10%, and each contrasting dramatically from the other in physicochemical composition) would have markedly different effects on the duration of the fed state. These assumptions, however, were unfounded. Results of the present study indicated that the effect of a normal mixed nutrient diet on gastrointestinal motility in the dog differed from the effect of the diet’s individual components.

The definition of exactly when changeover occurs depends on the segment of intestine studied. Bueno et al. reported that in the dog, the activity front (or phase 3) of the MMC develops first in the jejunum 8 to 10 hours after feeding, and approximately 1 hour later in the duodenum, at which time there is a transient decrease in antral motility. Bueno et al did not observe a distinct period of antral motility consistent with an activity front preceding that in the duodenum. Therefore, the origin of the activity front (phase 3) wanders, moving to more oral sites after prolonged periods of fasting. Failure to detect an antral MMC was in disagreement with findings of Itoh et al., who, in chronic 24-hour studies of the stomach, defined distinct digestive and interdigestive patterns, with the time from feeding to the appearance of the first activity front ranging from 14 to 16 hours. Itoh et al. stressed the importance of regular daily readings in the maintenance of this pattern. Dogs in the present study missed a meal the day before each experiment, which may explain the variation in the time to changeover.

Commercial dog foods, when fed isocalorically to large-breed dogs, did
not influence the rate of GE or the
time to change from the fed to the
fasted pattern of gastrointestinal
motility. Therefore, cereal-based diets
may have been wrongly incrimi-
nated as a predisposing factor for GDV
in large-breed dogs.

References
1. Blackburn PS, McFarlane D. Acute fa-
tal dilatation of the stomach in the dog (tor-
sion of the stomach). J Comp Pathol 1984;10;4:189-
199.
2. Pass MA, Jarston DE. Treatment of
gastric dilatation and torsion in the dog. J
3. Tweed D, Wingfield WE. Diseases of the
stomach. In: Ettinger SJ, ed. Textbook of veteri-
inary internal medicine. 2nd ed. Philadelphia:
4. Wingfield WE, Cornelius LM, De-
Young DW. Experimental acute gastric dilat-
ation and torsion in the dog. I. Changes in
biochemical and acid base parameters. J Small
5. Wingfield WE, Cornelius LM, De Young
DW. Pathophysiology of the gastric dilata-
tion-torsion complex in the dog. J Small Anim
6. Merkley DF, Heward DR, Eyster GE,
et al. Experimentally induced acute gastric
dilatation in the dog: cardiopulmonary ef-
7. Van Kruijningen HJ, Gregoire K, Meu-
ten DJ. Acute gastric dilatation: a review of
comparative aspects by species, and study in
dogs and monkeys J Am Anim Hosp Assoc
8. Rogolsky B, Van Kruijningen HJ. Short-
chain fatty acids and bacterial fermentation
in the normal canine stomach and in acute
gastric dilatation. J Am Anim Hosp Assoc
1978;14:504–515.
9. Warner NS, Van Kruijningen HJ. The
incidence of clostridia in the canine stomach
and their relationship to acute gastric dilata-
933.
10. Caywood D, Tsague HD, Jackson DA,
et al. Gastric gas analysis in the canine gas-
tric dilatation-volvulus syndrome. J Am Anim
11. Bolton JR, Merrill AM, Carlson GM, et
al. Normal abomasal electromyography and
emptying in sheep and the effects of intra-
abomasal volatile fatty acid infusion. Am J
12. Horwitz W. Official methods of analysis
of the association of analytical chemists. 15th
ed. Washington, DC: Association of Analyti-
cal Chemists, 1975.
13. Theodorakis MC. External scintigraphy
in measuring rate of gastric emptying in
15. Kelly KA. Motility of the stomach and
gastro-duodenal junction. In: Johnson LR, ed.
Physiology of the gastrointestinal tract. New
16. Hinder RA, Kelly KA. Canine gastric
emptying of solids and liquids. Am J Physiol
17. Hunt JN, Stobbs DF. The volume and
energy content of meals as determinants of
18. Malegolada JR. Physiological basis and
clinical significance of gastric emptying dis-
19. Bartelsman JFW. Current tech-
niques in the evaluation of motility disorders.
Scand J Gastroenterol (Suppl) 96 1984:195–
10.
20. Minami H, McCullum RW. The physi-
ology and pathophysiology of gastric empty-
ing in humans. Gastroenterology 1984;
86:1592–1610.
21. Dewever I, Ekhoute C, Vanstraeten G,
et al. Disruptive effect of test meals on inter-
digestive motor complex in dogs. Am J Phys-
22. Lawaetz O, Ollesu HP, Andreasen R.
Evaluation of gastric emptying by a simple
isotope technique. Scand J Gastroenterol
1983;16:737–748.
23. Carlson GM, Radday RW, Hug CC, et
al. Effects of nifedine on gastric antral and
duodenal contractile activity in the dog. J
Diurnal changes in gastric motor activity in
25. Bueno I, Raynor V, Reussbusch Y. Ini-
tiation of the migrating myoelectric complex