The Influence of Diet and Feeding Frequency on Gastric Function in the Dog

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The effects of four different feeding regimens on canine gastric function were investigated. Irish setters were fed either commercial dry dog food or meat and bone rations once or three times daily. Gastric acid secretions were studied, serum gastrin levels determined, cinefluoroscopy of the stomach performed, and autopsies done. Gastric acid secretion levels and motility measurements were not different significantly among groups. Postprandial serum gastrin levels were higher significantly in dogs fed once daily than in those fed three times daily. Autopsies revealed that dogs fed commercial dry dog food once daily had enlarged stomachs when compared to the other three groups.

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Introduction

In 1974 Van Kruiningen *et al*[1] suggested a correlation between diet and feeding patterns and the occurrence of canine acute gastric dilatation (AGD). The AGD was described as multifactorial in origin, occurring through the interaction of the susceptible individual, the abnormal stomach, readily fermentable feeds,[2,3] a gas-producing gastric flora,[2,3] and other precipitating elements.[1]

A review of the literature[4] reporting the food habits of feral carnivores makes it clear that the staple diet of *Canidae* living in a natural setting includes carcasses of other animals, carrion, fruits, and grasses. Natural diets are high in animal protein and animal roughage (poorly digestible parts of animal carcasses, *eg*—bone, cartilage, scales, fin, fur, feather, tendon, and teeth) and low in carbohydrates and caloric density (the fat content of the flesh of wild animals is 5%). Feral carnivores undoubtedly eat several times daily (nightly), catching "as catch can," with periods of rest or fruitless scavenging in between. Stomach analyses reveal that carnivores masticate their prey minimally and prefer to swallow large bolets, *ie*—portions of carcasses with indigestible elements included.

When dogs are housed under controlled diurnal circumstances and permitted to create their own eating patterns, they eat three times daily, nocturnally, and nibble in between.[5]

Domestication imposes diets on the dog which are lower in protein and roughage and higher in carbohydrates and caloric density. The natural feeding pattern is replaced by a once-a-day routine designed for convenience. With this contrast in mind, and its possible significance in AGD, a diet mimicking that of the feral canid was developed, and the effects of diets and feeding frequency on gastric function were compared.

Materials and Methods

Eight Irish setters were obtained for research at 12 weeks of age. They were housed separately in chain-link units approximately 1.4 m by 1.8 m in size. Floor surface was concrete and no bedding was provided. (They were offered fiberglass beds but would not use them.) The pens were washed once daily and vacuumed dry. Excrement was removed at frequent intervals throughout the day. Water was available *ad libitum*.

Each of the experimental animals was vaccinated against canine distemper, infectious canine hepatitis, and leptospirosis at 12 and 16 weeks of age. Each was given anthelmintics to eliminate ascarids, hookworms, and whipworms.

During two weeks of acclimation, the animals were fed a commercial dry puppy food. After initial weight comparisons, the dogs were assigned to different feed regimens [Table 1]. Two were fed commercial dry dog food (CD)[a] once daily; two were fed a meat-and-bone (MB) ration once daily; two were fed CD three times daily (at 8-hour intervals); and two were fed MB three times daily (at 8-hour intervals). The MB diet was designed to mimic that of similar-size feral carnivores eating in the wild[4] (composition described in Tables 2 and 3). Whole chicken parts fed raw, with bone and skin included, constituted 60% of the ration, and ground horsemeat constituted 29.9%. The amount fed was based on National Research Council[6] caloric requirements which were calculated from weekly weights. The dogs were given the opportunity to eat each ration for a one-hour period, after which remaining food was removed and weighed. Six months into the study, one of the dogs being fed CD once daily developed AGD, died, and was replaced.

Gastric acid secretion levels were studied in each dog after the animal had been maintained exclusively on one of the four feed regimens for at least one year. The dog was fasted for 24 hours prior to the study. Pentobarbital was administered at 28 mg/kg body weight and then given to effect. Anesthesia was induced to a surgical plane and an endotracheal tube was inserted.

The animal was positioned in left lateral recumbency and a Sovereign polyvinyl stomach tube[b] [Figure 1A], wetted inside and out, was lubricated with Lube jelly[c] and inserted down the esophagus and just through the cardia. (This large-bore tube provided a conduit for the more flexible 16-gauge stomach tube described below, which cannot otherwise be passed through the collapsed esophagus of the anesthetized animal.) The polyvinyl tube was marked approximately 59 cm from the gastric end, and this mark served as a reference point for depth of insertion. In the 25 kg Irish setter, the mark was aligned with the canine teeth and then inserted an additional 1 to 2 cm as needed. A 60 cc catheter-tipped syringe was used to force 100 to 150 ml of air down the tube ensuring that the body of the stomach was opened.

Then a 16-gauge Argyle Levin-type stomach tube[b] [Figure 1B] with approximately 8.6 gm bullet-shaped lead weight attached to the end was wetted, lubricated with Lube jelly,[c] and passed through the larger polyvinyl tube and 10 cm beyond into the body of the stomach. The Levin tube was marked approximately 70 cm from the gastric end, a mark which was aligned with the proximal opening of the Sovereign polyvinyl tube. The most proximal of the four distal fenestrations was closed with transparent tape.

The dog was rotated into ventrodorsal position and a radiograph was taken to verify the placement of the tube. The dog's shoulders, neck, and head were elevated slightly to preclude reflux of secretions into the larger polyvinyl tube. The stomach was rinsed four or five times with 50 ml amounts of water, followed by 30 ml boluses of air. The water was completely retrievable when the dog and tubes were positioned properly.

Collections of basal gastric secretions were taken every 15 minutes for one hour. The dog was held in ventrodorsal or slight left ventrooblique position throughout the study. Collection was aided by gentle rocking of the dog from ventrodorsal to left ventrooblique position and by manual compression of the abdomen dorsally and cranially under the rib cage. The Levin tube was moved in and out 1 to 2 cm as needed. Whenever gastric

Table 1

Experimental Design

Regimen	Feedings per 24 Hours: 1	3
Commercial dry food	15A,* male 22, male	16, female 17, male
Meat-and-bone ration	18, female 19, female	20, female 21, male

*Dog number

mucosa occluded the fenestrations, air was inserted to reopen the channel. Thirty milliliter boluses of air were inserted and boluses of secretions from 1 to 20 ml were aspirated until no more could be collected.

After the four 15-minute basal collections were taken, pentagastrin[d] (12 µg/kg body weight) was injected subcutaneously. Additional collections were taken at 15-minute intervals for two hours. At the conclusion of the study, 30 ml of water was inserted and retrieved to verify again the proper positioning of the tubes.

The 15-minute collection samples were bottled separately. The sample volume was measured and each sample was titrated with 0.1 N NaOH to a pH of 7.0 as indicated by pH meter. Secretion was ex-

Table 2

Chemical Composition of Commercial Dry and Meat/Bone Rations

	Wet Weight Basis (as fed)		Dry Weight Basis	
	Commercial Dry*	Meat/Bone	Commercial Dry*	Meat/Bone
Protein (%)	21.0	17.9	23.9	41.3
Fat (%)	8.0	9.7	9.1	22.4
Carbohydrates (%)	45.0-50.0	2.0	39.6-44.0	5.0
Fiber (%)	4.5	0.3	5.1	0.7
Water (%)	12.0	56.7	—	—
Kcal/gm	3.5	1.4	4.0	3.2

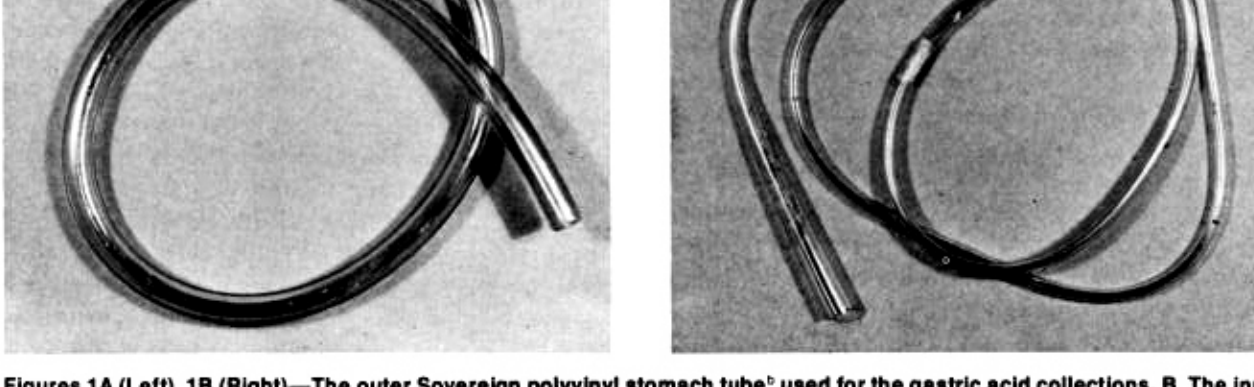
*Purina Dog Chow, Ralston Purina Co., St. Louis, MO

Table 3

Ingredients of Commercial Dry and Meat/Bone Rations

Commercial Dry*	Meat and Bone*
Ground yellow corn	Whole chicken parts, including skin and bone, *ie*—canner fowl 60%)
Soybean meal	
Corn gluten feed	
Ground wheat	Ground horse meat (29.9%)
Meat and bone meal	Ground whole apple (9.7%)
Animal fat preserved with BHA	Bran (0.4%)
Corn gluten meal	Vitamins (A, D, and E)†
Vitamin and mineral sources	Trace minerals

*Listed in order of decreasing amounts.
†Added to equal levels in commercial dry ration.



Figures 1A (Left), 1B (Right)—The outer Sovereign polyvinyl stomach tube[b] used for the gastric acid collections. B. The inner 16-gauge Argyle Levin-type stomach tube[b] with lead weights attached to the end used for the gastric acid collections.

Table 4

Pentagastrin-Induced Acid Secretion

Dog No.	Food Regimen	BAO	MAO	PAO
		(mEq per Hour)		
15A	CD1	0.168	18.32	20.92
22	CD1	0.333	24.37	24.70
18	MB1	0.003	15.59	17.20
16	CD3	0.000	22.41	23.32
17	CD3	0.007	25.13	26.64
20	MB3	0.005	25.69	31.46
21	MB3	0.011	21.83	24.38

Table 5

Pentagastrin-Induced Acid Secretion

Dog No.	Food Regimen	BAO	MAO	PAO
		(mEq per Hour/kg Body Weight)		
15A	CD1	0.00771	0.840	0.960
22	CD1	0.01261	0.923	0.937
18	MB1	0.00012	0.624	0.688
16	CD3	0.00000	1.170	1.220
17	CD3	0.00024	0.850	0.902
20	MB3	0.00022	1.130	1.390
21	MB3	0.00031	0.608	0.679

pressed as mEq of acid. The basal acid output (BAO) was recorded (mEq per hour) and the maximum acid output (MAO) and peak acid output (PAO) were calculated. The MAO was taken as the four highest acid measurements (mEq per hour). The PAO was taken as the two highest acid measurements times two (mEq per hour).

Postprandial serum gastrin levels were measured after the dogs had been maintained on one of the four feed regimens for at least one year. Postprandial samples were taken at 30 and 60 minutes and sent to an outside reference laboratory.[e] The levels were determined by radioimmunoassay and reported in pg/ml.

Cinefluoroscopy studies were done after the dogs had been on one of the four feed regimens for at least one year. The dogs were fasted from 16 to 25 hours prior to the study. Each dog was fed, or force-fed, its normal feed mixed with barium contrast medium immediately before filming was begun. The unanesthetized, untranquilized dog was placed in sternal recumbency. The X-ray beam was positioned at 90° to the table top. Gastric movements were recorded on film at intervals for 8 to 10 minutes and the number of antral contractions per minute was counted. The dog was returned to its cage for 20 to 40 minutes after which fluoroscopy was resumed. Again, gastric movements were recorded and the number of antral contractions per minute was counted. At a later time, stomach width and length measurements were made from the films taken and were used to calculate volume in milliliters for each animal.

At approximately two years of age, each dog was euthanized. This was carried out two hours after a last complete meal and autopsy followed immediately.[7] The body weight was recorded. All organs were examined, with emphasis on the stomach. Gastric parameters including empty stomach weight, filled and empty greater and lesser curvature lengths, and pyloric and cardiac dimensions were noted. The stomach weight as a percentage of body weight was calculated. The gastric contents were examined and weighed and the food volume determined.

Results

The dogs accepted their respective rations and feeding schedules and grew well. After several weeks some of the dogs appeared disinterested with their unchanging rations, but soon thereafter resumed regular daily feeding.

After six months of study, one dog in the CD1 group (No. 15) developed AGD several hours after its meal. The stomach was emptied by stomach tube, and the dog was treated medically. Recovery was uneventful. Two weeks later, the dog developed AGD for the second time following its meal. Again the dog was treated medically and responded. The dog resumed eating the next day, feed was increased slowly, and further recovery was uneventful. Six weeks after the second episode the dog developed AGD again. A stomach tube could not be passed beyond the cardia, so the dog was taken to a local veterinary hospital where a 360° gastric volvulus with a splenic torsion was corrected surgically. The dog appeared to recover well postoperatively, resumed eating, and continued on the study. One month postsurgery the dog was found bloated and prostrate, suffering from AGD and shock. A stomach tube was passed, the stomach was emptied, and the dog was treated medically. The dog appeared to respond well to medical

Figure 2—The effect of once daily feeding of commercial dry dog food (CD1) or meat/bone (MB1) on pentagastrin-induced gastric acid secretion.

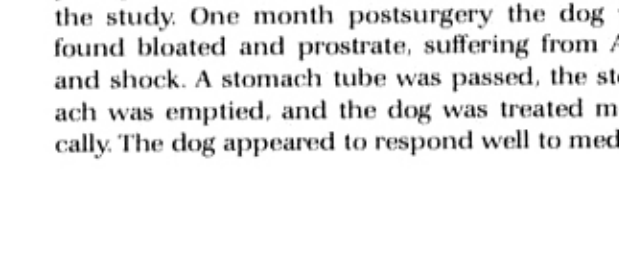


Figure 3—The effect of three daily feedings of commercial dry dog food (CD3) or meat/bone (MB3) on pentagastrin-induced gastric acid secretion.

therapy, but five days later AGD occurred again. The dog was treated medically, failed to respond, died, and was autopsied. A postmortem diagnosis of multiple gastric ulcers, one of which had perforated, was made.

For the purpose of maintaining the integrity of the study, a replacement dog (No. 15A) was purchased (12 weeks of age), conditioned as the originals had been, and placed in the CD1 group.

The remainder of the dogs continued in the study uneventfully with the following exception. Dog No. 21 of the MB3 group showed intermittent anorexia and vomiting for two weeks after having been in the study for 23 months. Studies of gastric function were conducted only after each animal had been on its respective ration for more than 12 months. Dog No. 19 died unexpectedly after 19 months in the study from postanesthetic hypothermia following gastric acid secretion studies.

The results of the gastric acid secretion studies are presented in Tables 4 and 5. Individual responses are plotted in Figures 2 and 3. The BAO was negligible in all dogs. The MAO and MAO/kg body weight were similar among the four groups. Acid secretion peaked initially at 45 to 75 minutes after pentagastrin stimulation. The PAO and PAO/kg body weight were equally similar. No differences were apparent between CD and MB groups regardless of frequency of feeding, nor between dogs fed once and three times daily regardless of ration.

The postprandial serum gastrin measurements are presented in Table 6. The values for all four groups were within the range of normal values.[8-15] Differences in serum gastrin levels were apparent between the dogs fed once daily and those fed three times daily when examined by the two-tailed t-test for uneven sample sizes at 30 minutes ($p<0.01$) and at 60 minutes ($p<0.025$). Animals fed once daily had higher postprandial gastrin responses than those fed three times daily. No differences in serum gastrin levels were noted between the two ration groups.

Cinefluoroscopy showed that the CD1 regimen of feeding caused much greater distention of the stomach than the other regimens; however, motility differences were not discernible. Movements of the fundus and body were insignificant and no differences in antral contraction patterns could be seen, either first hand or later in reviewing the motion films. All seven dogs had surprisingly similar rates of antral contractions, four to five per minute. Attempts to calculate gastric volumes from measurements taken from the films proved unrewarding.

Gastric dimensions taken at the time of autopsy, two hours following a complete meal, revealed a significantly greater postprandial food

Table 6

Postprandial Serum Gastrin Measurements

Dog No.	Food Regimen	30 Mins	60 Mins
		(pg/ml)	
15A	CD1	144	113
22	CD1	119	158
18	MB1	146	193
16	CD3	80	108
17	CD3	97	92
20	MB3	80	92
21	MB3	69	75

Table 7

Gastric Dimensions Taken at Autopsy Two Hours Following a Complete Meal

Dog No.	Food Regimen	Body Weight (kg)	Empty Stomach Weight (gm)	Stomach Weight/ Body Weight (%)	Greater Curvature (food-filled) (cm)	Food Weight (gm)	Food Volume (ml)
15A	CD1	20.9	198.6	0.95	40.7	909	1000
22	CD1	24.5	252.8	1.03	45.7	1100	1050
18	MB1	25.9	146.0	0.56	36.0	697	650
16	CD3	17.7	154.7	0.87	34.3	390	400
17	CD3	27.3	194.0	0.71	38.4	401	375
20	MB3	21.4	143.0	0.66	29.2	334	—
21	MB3	36.4	205.8	0.56	34.5	407	360

residue volume and weight, greater length of greater curvature, and greater gastric weight in dogs fed CD once a day compared with those fed MB once daily, or those fed MB or CD three times daily [Table 7]. Dogs fed the CD1 ration had the heaviest stomachs, as expressed as a percentage of body weight, and those fed MB3 had the lightest. There were no differences in lesser curve, pyloric or cardiac dimensions, and weights of pancreas and salivary glands.

Discussion

All of the experimental animals fared well on their respective rations. One of the four receiving meat and bone ration containing uncooked chicken bones had a period of partial anorexia and intermittent vomiting which may have been related to gastritis. The dog recovered after minor medical treatment. All four dogs readily consumed the bones in their rations. It is interesting to note that whenever radiographs were made, usually after a 24-hour fast, bones were not demonstrable in the stomachs, an indication of the capacity of the dog to digest such material.

One of the four dogs receiving commercial dry dog food, in particular one which was being fed once daily, developed AGD and died. The occurrence of AGD in one of two dogs being fed in this manner does not establish a cause-and-effect relationship between diet and disease. Nevertheless, the coincidence is striking and consistent with the authors' understanding of the role of ration and meal frequency in the pathogenesis of AGD.[1]

No evidence was found of a diet- or meal frequency-induced influence on gastric acid secretion that might have a role in AGD. Gastric acid output was similar from all the dogs following maximum stimulation with pentagastrin. The technique employed was adapted from that in use in man.[16,17] Preparatory to the collections made in these Irish setters, the method was tested repeatedly in mixed breeds, and the effects of two anesthetics (pentobarbital and methoxyflurane)[17-24] and several different pentagastrin doses (6, 8, and 12 µg/kg body weight) were compared. This study represents the first time pentagastrin-stimulated acid secretion has been measured in the dog with an intact stomach. Comparisons with other studies in the literature are fraught with difficulty because of the differences in animal size, gastric surgical preparation, stimuli applied, and methods of measurements.[15,24-33]

Since the authors established that under the test circumstances the dogs produced adequate acid, it appeared important to establish if postprandial serum gastrin levels occurred equally among the various groups. All of the dogs had normal postprandial responses, and the dogs fed once a day had higher gastrin levels, presumably in response to greater gastric distention.

Although one might have expected different gastric motility patterns among the various groups of dogs, none was discernible by cinefluoroscopy. Postprandially, the stomachs of dogs fed commercial dry dog food once daily were larger than those of the other three groups, and this observation correlated well with postmortem findings. When dogs are required to consume their entire daily caloric needs in the form of one meal of commercial dry dog food, a great postprandial gastric volume results. This was indicated by the greater intragastric food volume and weight measured at autopsy two

hours after eating [Table 7]. It also was reflected in the length of the greater curvature of the two-hour postprandial stomach. Rats which have been intermittently fasted and then allowed to engorge develop enlarged, heavier stomachs than those fed *ad libitum*.[34] The dogs in the present study which were forced to fast for 23 hours daily and then allowed to engorge on commercial dry dog food had heavier stomachs (0.95% and 1.03% of body weight) than those consuming meat and bones once daily (0.56%) or those consuming either ration three times daily (0.56% to 0.87%). Cinefluoroscopy showed that once-a-day feeding on commercial dry dog food produced immediate postprandial gastric enlargement. Autopsy demonstrated that the enlargement persisted at least two hours after eating, and postmortem weights of empty stomachs indicated that a chronic change in gastric anatomy had occurred. It is reasonable to presume that there may be an associated change in migrating myoelectric complexes and gastric emptying.[35-40]

Canine acute gastric dilatation is a disease of domestication, occurring most frequently in our best-cared-for purebred stock and least frequently in dogs that run free and have an opportunity to scavenge. Canids in the wild consume diets high in animal protein and roughage (not plant fiber but indigestible or poorly digestible parts of animal carcasses) and low in carbohydrates and caloric density. In contrast, our kenneled domestic canids are forced to consume a diet low in protein and roughage and high in carbohydrates and caloric density. Furthermore, such commercially prepared rations are finely ground, superheated, and dextrinized, rendering them "readily digestible" and fermentable. When the dog is fed such preparations as its exclusive diet once daily, a large volume is consumed and swells postprandially when water or gastric secretions are added. This is in contrast to what occurs in the postprandial digestion of meat and bone; the latter are reduced readily in volume by the digestive capabilities of the canine stomach. It should be pointed out that the rate of gastric emptying is inversely proportional to caloric density;[35,41] it has been clearly established in several species that the presence of indigestible roughage is important for normal gastric motility. The obligate role of roughage in the ruminant is well established,[42,43] and chickens deprived of roughage develop dilated, atonic stomachs.[44-46]

Dogs which have died of AGD often have an entire meal still present in the stomach 12 to 18 hours after eating, a clear indication of delayed gastric emptying. The authors' findings demonstrate enlarged, heavy stomachs in dogs fed commercial dry dog food once daily. This would suggest that these represent stomachs predisposed to AGD.

In previous studies the authors have demonstrated the presence of a fermentative gastric flora, *Clostridium perfringens*, in 70% of dogs,[3] and have suggested that the highly processed soybean meal and cereal grain carbohydrates in commercial dry dog foods represent a substrate for fermentation.[2] Rogolsky and Van Kruiningen[2] showed that lactic acid levels were nine times greater in stomachs from cases of AGD than from controls, that there was a substantial increase in gastric CO_2 in AGD, and that fermentation rates were three times as rapid in contents from AGD cases.

The most compelling evidence of the roles of *Clostridium perfringens* and a fermentable substrate comes from the recent work of Bennett *et al*[47] with monkeys and Stein *et al*[48] with marmosets. In the former study, *C. perfringens* was demonstrated in the gastric contents of 21 of 24 monkeys with AGD (a disease identical to that of the dog), in only two of 18 samples from normal animals, and in five of 11 lots of feed (a cereal soybean-base dry product). The latter report documented the occurrence of 29 cases of AGD in marmosets over a five-week period following therapy with gentamicin and furoxone; *C. perfringens* type A was demonstrated in the gastric contents of all 25 animals that were autopsied.

The authors feel that the data presented here adds to the incrimination of commercial dry diets and feeding frequency in the cause of AGD. The possible sequence of events in AGD may be as follows: 1) Once-a-day feeding of a commercial dry dog food yields gastric enlargement [Figure 4] and

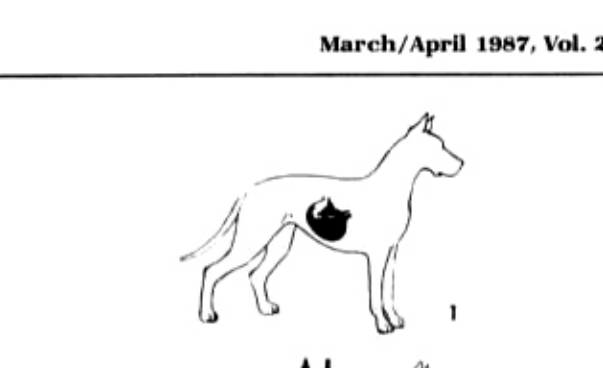


Figure 4—Radiograph of the canine stomach severely distended following once-a-day-feeding of commercial dry dog food.

a characteristic flora. 2) Repeated once-daily enlargement of the stomach results in a large, heavy organ with deranged migrating myoelectric complexes or emptying. 3) Delayed emptying or failure for eructation, in the presence of a fermentable substrate and clostridia, yields gaseous gastric distention. 4) Repetition of these events or contribution by another factor, such as heavy exercise, increased ambient temperature, and overconsumption of water yields acute gastric dilatation, with or without volvulus [Figure 5]. An understanding of this possible sequence should allow for interruption in this pattern and ultimate prevention.

[a]Ralston Purina Co., St. Louis, MO
[b]Sherwood Medical Industries, Inc., St. Louis, MO
[c]Elanco Products Co., Indianapolis, IN
[d]Peptavlon, Ayerst Laboratories, New York, NY
[e]Vet Path Animal Reference Laboratory, Teterboro, NJ

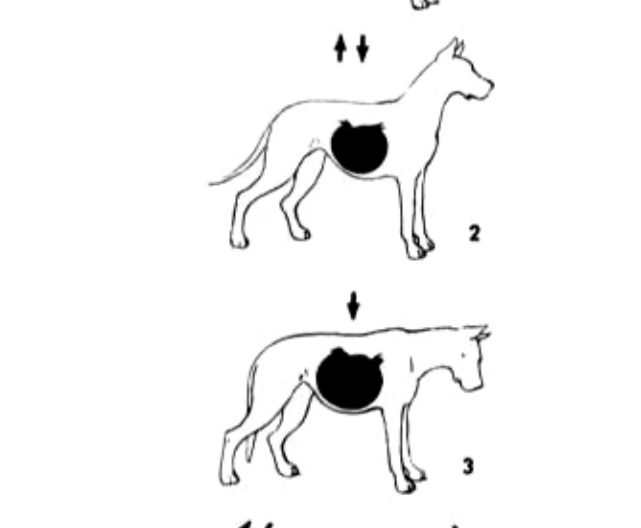


Figure 5—Diagrammatic representation of the pathogenesis of acute gastric dilatation.
1. **Normally proportioned stomach in a dog fed modest-size meals (blackened area) several times daily.**
2. **Chronically enlarged stomach resulting from once daily feeding of commercial dry dog food.**
3. **Irreversibly chronically enlarged stomach, functionally deranged, sometimes producing prodromal signs following meals.**

4a. Acute gastric dilatation.
4b. Acute gastric dilatation with volvulus.

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