African Journal of Biotechnology Vol. 9 (30), pp. 4818-4821, 26 July, 2010
Available online at http://www.academicjournals.org/AJB
DOI: 10.5897/AJB09.1493
ISSN 1684–5315 © 2010 Academic Journals

Full Length Research Paper

Failure of experimental acute intra-gastric gas and fluid accumulation to produce gastric dilatation-volvulus in unanaesthetized dogs

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Accepted 5 April, 2010

The stomachs of 6 mongrel dogs were cannulated with a simple assemblage of cannula and sphygmomanometer for direct and rapid administration of gas and combination of gas and fluid, respectively, to investigate the role of intra-gastric accumulation of gas and fluid in the stomach in development of acute gastric dilatation-volvulus in the animals. In all the experiments, the stomach of each dog was fully distended at a consistent intra-gastric pressure of 31.73 ± 0.43 mmHg. The mean intra-gastric pressure of the empty stomach in fasted unanaesthetized animals was 4.17 ± 0.63 mmHg, while in animals that received 500 ml of water, the mean intra-gastric pressure was 14.2 ± 2.4 mmHg. The animals successfully evacuated the distended stomach at the peak of the intra-gastric pressure by eructation and/or vomition in 1.31 ± 0.43 min. There were no clinical and radiological evidence of the classical acute gastric dilatation-volvulus syndrome in any of the animals. The experiments failed to show that rapid accumulation of gas and fluid in the stomach primarily caused the condition in the clinically normal animals. There was apparent intra-gastric pressure threshold at which the cardiac orifice of the stomach of the animals opened for evacuation. The assemblage was functionally efficient for direct and rapid administration of gas and fluid into the stomach and simultaneous measurement of intra-gastric pressure in unanaesthetized dogs.

Key words: Experimental, gas, fluid, accumulation, gastric dilatation-volvulus, dogs.

INTRODUCTION

Although the pathophysiology and clinical management of acute gastric dilatation-volvulus in dog and man have been widely studied and fairly understood, the aetiology and pathogenesis remain largely unknown (Morris et al., 1947; Vankruiningen et al., 1974; Todoroff, 1979; Lantz et al., 1992., Brockman., 1995). Among several factors, presence of a large volume of gas and fluid in the stomach and inability of the affected animal or person to evacuate the acutely distended stomach by vomiting and/or eructation are consistently associated with the development of the diseases (Wingfield et al., 1976; Strombeck et al., 1989; Strombeck and Guilford., Simpson 2005). Efforts to reproduce the condition experimentally have not been successful. However, indirect gas distention of the stomach of anaesthesized experimental animals via oesophageal tubes with intra-gastric balloon and tube has been used in studying the pathophysiology of the disease (Engler et al., 1967; Vankruiningen et al., 1974; Wingfield et al., 1976; Strombeck et al., 1989).In the experiments reported here, attempts were made to reproduce the condition, by stimulating aerophagia, through rapid and direct administration of gas and fluid into the stomachs of unanaesthesized dogs via a simple gastric cannula.

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MATERIALS AND METHODS

Ethnical committee approval was sought and obtained from the experimental animal unit of the faculty of veterinary medicine before the commencement of the investigation.

Six adult mongrel dogs; 3 males and 3 females, obtained for the local dog market and weighing between 15 and 21 kg body weight, were used for the study. The animals were kept in the departmental kennel for four weeks to acclimatize and were maintained on a

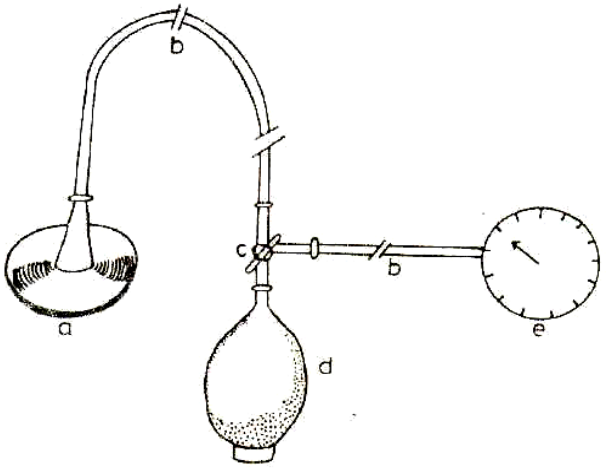

Figure 1. Gastric cannula assemblage. (a) Cannula, (b) Silastic tubing, (c) 3-Way-valve adaptor, (d) Bulb inflator and (e) Aerobic manometer.

Figure 2. Lateral radiograph of the abdomen at the peak of intragastric pressure.

standard diet and water provided *ad libitum* through out the period of study. Baseline evaluations include physical examination, complete blood count (CBC), serum biochemistry analysis, urinalysis and fecal examination for parasitic ova. All results were negative or within established reference ranges. The gastric cannula was made from the base of the inflation nozzle of the arm band of a clinical sphygmomanometer (Boyles USA) and a 75 cm long silastic tubing of 3.0 mm inner diameter. The cannula connected by a 3-way adaptor (JMS inc) to a bulb inflator and an aerobic manometer graduated in millimeter mercury, constituted the assemblage for direct administration of fluid and gas into the stomach of the experimental animals and measurement of intra-gastric pressure (Figure 1).

The stomach in each animal was approached through a ventral median laparotomy under general anaesthesia. The dogs were premedicated with intramuscular injections of xylazine hydrochloride (xylaxine®, Kepro, Holland) and Atropine (Non - proprietary) at the dose rate of 1.0 and 0.02 $mgKg^{-1}$ body weight, respectively. Anaesthesia was induced and maintained using 6% sodium pento-barbitone (Sagatal® Kyron Laboratories) at the dose rate of 10 $mgKg^{-1}$ body weight. The stomach in each animal was approached through a ventral medium laparotomy as described by Chambers (1979). The base of the cannula was inserted into the lumen of the stomach through a gastrotomy incision made at about the midpoint between the pylorus and the cardia on the long axis and between the greater and the lesser curvatures of the stomach respectively. The arm of the cannula was brought out of the stomach through a stab incision made about 2.0 cm caudal to gastrotomy incision. The base of the cannula was sutured to the wall of the stomach with a non-absorbable 1/0 propylene (Ethicon inc) suture material, while the neck of the cannula was secured to the gastric wall with a purse-string suture of the same material. The distal end of the cannula was passed through a subcutaneous tunnel created with a pair of long straight artery forceps on the lateral body wall and exteriorized anterior-dorsally through a stab cutaneous incision between the 11^{th} and 12^{th} ribs. Care was taken not to penetrate into the thoracic cavity in the process. About 7.5 cm of the distal end of the cannula was exposed on the body surface. The cutaneous stab incision was closed on the cannula with a purse-string suture of 2/0 vetafil (Ethicon inc) material. All the animals received intramuscular injection of Dichlofenac (Wuham Pharmaceutical, China) at $5mgkg^{-1}$, post operation. The wounds were allowed to heal for three weeks before the experiments progressed further.

With the gastric cannula connected to the assemblage, the intragastric pressure of the unanaesthetized animals was recorded with the manometer after a 24-h fast. The stomach was inflated by manual pumping of the bulb, while the maximum intragastric pressure and the duration it was retained before eructation and relief of ensuing gastric-distention were recorded three times at hourly intervals for each animal. The lateral and ventro dorsal radiographs of the abdomen of the animals were taken at the peak of the intragastric pressure. One week later, the stomach of each animal was inflated after 500 ml of tap water has been directly administered into the stomach via the gastric cannula. The peak intragastric pressure was recorded. The experiments were repeated three times in each of the animal at hourly intervals. The cannula was left in the animals for 6 months after which the animals were sacrificed and examined for any untoward effects of the materials on the viscera and adjacent tissues.

RESULTS

The mean intragastric pressure in the unanaesthetized fasted animals before direct administration of gas and/or fluid was 4.17 ± 0.63 mmHg, while it rose to 14.2 ± 2.4 mmHg, when 500 ml of tap water was administered through the cannula into the stomach. The stomach was progressively distended as gas was directly administered through the cannula into the stomach. Peak intra-gastric pressure of 31.45 ± 0.71 mmHg was recorded and retained for a mean period 1.31 ± 0.43 min before eructation occurred and gastric distention was relieved. Following direct administration of 500 ml of tap water and immediate gas inflation of the stomach the mean intragastric pressure recorded was 31.73 ± 0.43 mmHg. The peak intragastric pressure was retained for a mean period of 1.33 ± 0.11 min before the animals vomited, relieving the stomach of pressure. The radiographs of the lateral abdomen of the animals showed extensive dilatation of the stomach at the peak of intragastric pressure (Figure 2). Gas silhouette was most pronounced in the proximal jejunum, while the image of the posterior

Table 1. Means and standard deviations of intragastric pressure and duration of gastric distention under various conditions of the stomach.

Animal	Sex	Body weight (Kg)	Means of peak intragastric pressure (mmHg) and duration of gastric distention (min)						Development of acute GD-V*
			Fasted stomach (mmHg)	500 ml water engorged stomach (mmHg)	Gas distended Stomach (mmHg)	Duration of retention of distention (min)	500 ml water and gas distended stomach (mmHg)	Duration of retention of distention (min)	
ES0026	Male	15.7	4.33	15.30	30.89	1.56	31.00	1.33	-ve
ES0086	Male	20.0	4.67	16.00	31.56	1.22	31.67	1.33	-ve
ES0142	Male	18.7	5.82	14.67	31.67	1.33	31.56	1.22	-ve
ES0041	Female	9.80	3.33	10.86	30.67	1.22	32.78	1.56	-ve
ES0138	Female	20.2	3.67	13.53	32.44	1.22	31.00	1.33	-ve
ES0082	Female	20.5	3.33	15.60	30.67	1.22	32.28	1.35	-ve
Mean ± SD			4.17 ± 0.63	14.45 ± 0.24	31.45 ± 0.71	1.31±0.43	31.73 ± 0.43	1.35 ± 0.11	

*GD-V = Gastric dilatation-volvulus; SD = standard deviation; - ve = negative.

digestive tract was consistent with that of the fasted animals without gastric gas distention. None of the experimental animals showed any clinical evidence of acute gastric dilatation-volvulus after evacuation of the distended stomach.

Controlled direct and rapid distention of the stomach in dogs using the assemblage was easy and repeatable. The animals tolerated the cannula. Post mortem examinations did not reveal any evidence of pathological effects of the cannula on the gastro-intestinal tract and/or any other tissue in any of the animals after 6 months. There was no adhesion of tissues that could have caused restriction to the movement of the stomach. Table 1 shows the recorded observations in the experiments.

DISCUSSION

In all the animals and experiments, maximum gastric distention was produced for brief periods, but classical volvulus did not develop. Mechanically, the direct and rapid accumulation of gas and fluid in the stomach of the experimental animals in these studies simulated aerophagia, which has been incriminated in the aetiopathogenesis of the disease in man and other animals. It however failed to reproduce the condition in the normal unanaesthetized animals. It is not clear meanwhile from these experiments, whether the failure was due to the absence of yet unidentified pathological and/or genetic factors that must accompany aerophagia or intragastric accumulation of gas in the experimental animals. Mentioned among such factors is excessive exercise before feeding and the breed predisposition of deep chested dogs. The mongrel dogs used in these experiments did not have such predispositions.

The consistent peak of intragastric pressure recorded in all the animals before the evacuation of the stomach by eructation or vomiting is noteworthy. It seems to suggest that there might be a threshold of intragastric pressure necessary for the opening of certain, but yet unidentified, valvular systems of the gastric cardia in dogs. Two such structures with valvular actions; the gastro-oesophageal sphincter and the valve of His, respectively, have been described in dogs (Mann and Shorter, 1964; Merckley et al., 1976). Relationship between the valves and intragastric pressure has been determined (Diamant and Akin, 1972; Strombeck, 1979; Strombeck et al., 1989; Strumbech and Guilford 1990). Other antireflux mechanisms such as the oblique angle of the gastroesophageal junction, the intra-abdominal esophagus, the diaphragmatic crura and the fundic pressure have also been reported to prevent gastroesophageal reflux (Strombeck and Guilford, 1990; Brockman., 1995). Dysfunction could prevent normal eructation of gastric gas. This probably may have accounted for our unsuccessful attempt to produce gastric dilatation volvulus. The prevalence of gastric dilatation volvulus in large, deep-chested breeds also suggest possible dysfunction of normal anatomic arrangements designed to prevent gastroesophageal reflux (Simpson, 2005). Thus, the size and breed of dogs used in this study may have

excluded such risk factors. Matthiesen (1993) implicated delayed gastric emptying and outflow obstruction associated with compression of the pylorus and duodenum due to partial gastric volvulus, as additional risk factor for gastric dilatation volvulus. The intragastric pressure recorded with the assemblage in the animals used in these experiments was similar to that reported elsewhere (Sukla et al., 1968; Strombeck et al., 1989). Our inability to produce gastric volvulus may be attributed to the multiple nature of the risk factors involved in the gastroesophageal of the condition. However, the apparatus used was found to be reliably suitable for direct administration of gas and fluid into the stomach and simultaneous measurements of intragastric pressure in the unanaesthesized animals. The result or the post-morthern examination after six months of cannulation, indicated that there were no untoward effects of the foreign body in the experimental animals and thus confirmed that the animals could tolerate the cannula for a long time.

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