A STUDY OF MECHANISMS PRODUCING COMPETENCE AT THE GASTROESOPHAGEAL JUNCTION

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INTRODUCTION

The esophagus is a propulsive tube extending from the pharynx to the stomach. In most mammals, including man, coordinated contractions of the esophagus propel the contents in a caudal direction, whereas during oral transport the esophagus is passive. In ruminants coordinated contractions of the esophagus are responsible for movements of the contents in both directions (1, 2). Between swallows and regurgitations, mechanisms at either end of the tube prevent easy access of air from above and gastric contents from below. At its oral end is the upper esophageal sphincter and at its caudal end is the lower esophageal sphincter (LES).

The upper sphincter is composed of horizontal fibres of the cricopharyngeus muscle which arise from the sides of the cricoid cartilage and pass horizontally backwards to interlace with each other behind the pharyngoesophageal junction (3, 4). This strip of muscle is 1-2 cms. wide and is continuous caudally with the circular muscle layer of the upper esophagus. Both muscles in man and most other mammals are striated and tightly joined to each other. Since the cricopharyngeus muscle in its mean vertical extent measures 1.2 cms. (5) and less than 2 cms. in its widest part, manometric evidence that the elevated resting intraluminal pressure characteristics of the pharyngo-esophageal junction may extend over a distance of up to 4 cms., suggest that both the cricopharyngeus and esophageal muscle contribute to the closing mechanism at this site (6).

The mechanism which separates the gastric and esophageal lumens has aroused great controversy. Despite the failure of anatomists to define the exact site and nature of the gastroesophageal sphincter,

manometric studies in man (7) and experimental animals (8) have demonstrated a zone of elevated pressure at the gastroesophageal junction. This high pressure zone is now accepted as the "physiological sphincter", whose main function appears to be the prevention of reflux of gastric contents into the esophagus.

The role of paraesophageal structures and the position of the high pressure zone in relation to the diaphragm have remained controversial. The crucial mechanism preventing reflux seems to be the competence of the gastroesophageal sphincter. It has been shown that the presence or absence of reflux esophagitis in patients with hiatus hernia depends essentially upon the activity of the lower gastroesophageal sphincter rather than the existence of a hiatus hernia (9). Cohen & Harris (10) reported that the symptoms of reflux esophagitis and the radiological demonstration of a hiatus hernia had no close interdependence.

A number of operations have been devised in an attempt to prevent gastroesophageal reflux in man. The names of Allison, Belsey, Nissen, Collis and Hill (44, 89, 90, 100, 91) are associated with some of the better known procedures. The mechanisms by which these procedures may prevent reflux are less well defined than the physiological mechanisms which maintain competence at the normal gastroesophageal junction.

The majority of experimental studies on the mechanisms for restoring gastroesophageal competence have suffered from lack of a suitable animal model. In one such study a canine model with gastroesophageal reflux was made by excising the gastroesophageal region and anastomosing the esophagus to the gastric fundus (57). Such a model

bears no resemblance to the human situation, where a physiological sphincter exists, but is somehow incapable of preventing reflux. Therefore, the relevance of these experiments to human gastroesophageal reflux remains doubtful. Lind (40) showed that vagotomy performed on the thoracically displaced canine gastroesophageal (GE) junction renders it relatively incompetent and provides a model for the study of gastroesophageal reflux. It is reasonable to assume, then, that if vagotomy were performed on the GE junction before displacing it into the chest, not only would it serve as a model for gastroesophageal reflux but also the effect of thoracic displacement on the GE junction could be studied. The present study was undertaken to investigate the function of the vagotomized GE junction when displaced into the chest, the efficacy of modified Belsey and Allison repairs (56, 44) in restoring normal function, and the mechanisms by which the GE competence may be restored.

REVIEW OF LITERATURE

GASTROESOPHAGEAL JUNCTION

Anatomical studies of the gastroesophageal junction are disappointing in that they fail to define accurately where the esophagus ends and the stomach begins. They also fail to demonstrate an unequivocal muscular arrangement suggesting a sphincter mechanism. A review of terminology is essential at this stage.

The Cardia, as defined by Ingelfinger (1), is the line separating the saccular cavity of the stomach from the tubular lumen of the esophagus. The line is imaginary: there are no general landmarks, and the esophageal muscular coat merges imperceptibly with the gastric muscle. The lateral wall of the esophagus and the medial aspect of the fundus of the stomach meet at an angle known as the Incisura Cardiae or the angle of His (11). The angle is variable: it may be acute (50°) or so obtuse (175° - 180°) as to be non existent (12). Immediately distal to the cardia is the Willis loop (13) or the collar of Helvetius, a bundle of oblique muscle fibres of stomach which loop around the posterior, lateral and anterior aspects of the esophageal outlet as a horseshoe-shaped sling. These muscle fibres are thought to support the angle of His and are regarded by some as a sphincter mechanism between the esophagus and the stomach (14, 15, 16). The junction between the squamous esophageal and glandular gastric mucosa is an irregular line. The mucosal junction bears little relationship to the structure and function of the underlying muscle and does not always lie at the cardia. In a study of 200 cadavers, the mucosal junction was above the incisura cardiae in 47% (17), and a similar relationship was recorded in a smaller group. (18). Laimer described a well-defined esophageal constriction about 2 cms. above the diaphragmatic hiatus and 3 to 4 cms. above the

cardia (19). Similar observations have been made by others, although the details of the length of the constriction and its relationship to the diaphragm are variable, as are the names given to it. Some anatomists accept that a narrowing of the esophageal lumen, up to 3 cms. in length, either at or a few cms. proximal to the diaphragmatic hiatus, is frequently demonstrable in human cadavers. The cause of the narrowing has been reported as localized muscular thickening (22, 23, 24, 16). In 60 to 80 specimens examined by Lerche (16) the muscular wall at the site of constriction appeared thicker than the adjoining esophageal segment. Others have searched unsuccessfully for such a thickening (20, 13). In some animals more pronounced muscular thickenings have been described in the gastroesophageal region. The muscle at the cardia of the bat is five to six times thicker than elsewhere in the stomach (21). It is reasonable to conclude that, in man, unequivocal anatomical evidence for the existence of a lower esophageal sphincter (LES) is lacking.

GASTROESOPHAGEAL COMPETENCE

A number of mechanisms aiding or providing closure of the gastroesophageal junction have been described.

These include:

1. Esophago-gastric angle (angle of His) with its sling fibres (33, 34)
2. Mucosal rosette supported by muscularis mucosae. (36)
3. The right crus of the diaphragm forming a pinchcock (35).
4. An abdominal segment of esophagus (37) and
5. Intrinsic contraction of the gastroesophageal sphincter.

The evidence in support of all the above mechanisms except the last has been challenged by subsequent studies.

1. Esophagogastric angle: Smiddy & Atkinson (33) felt that the flap valve of mucosa, produced by the oblique entry of the esophagus into the stomach, was important in preventing reflux provided the angle (of His) was supported by a sling of oblique gastric muscle fibres, (collar of Helvetius or sling of Willis). There are a number of poorly designed studies to back this concept. Berridge (38), on a radiological study in humans, found little to support the concept of flap valve. In another study in dogs, downward displacement of the gastroesophageal region into the abdomen abolished the acute gastroesophageal angle without either altering the pressure profile of the gastroesophageal sphincter, or causing reflux to occur (39). When the gastroesophageal junction was displaced upward into the chest, the acute angle of His was obliterated but no evidence of gastroesophageal reflux was noted (40). Vandertoll showed that myotomy of the oblique muscle fibres of the stomach forming the loop of Willis did not produce incompetence at the cardia (41). Radiological studies on sliding hiatus hernia in human beings show that reflux occurs only in a small proportion of cases, although the acute angle of His is absent in all of them. On the basis of above observations it would appear that the flap valve of mucosa produced by the angle of His does not play a significant role in producing competence at the cardia.

2. Mucosal Rosette

When the gastro-esophageal region is approached through an esophagoscope, radiating folds of mucosa present the appearance of a rosette. A "rosette" is also seen when the gastroesophageal region is viewed from the gastric side. On the esophageal side the folds are

are lined with stratified squamous epithelium and on the gastric side with columnar epithelium. The muscularis mucosae in this region also appears thicker than usual. The mucosal rosette, supported by thickened muscularis mucosae, gives the impression of a purse-string action on the lumen. However, resection of the mucosal folds in the dog, where they are most prominent, does not result in reflux (42). Further evidence against the importance of the mucosal rosette comes from the observation that in atrophic gastritis in human beings, a condition in which the mucosal rosette is destroyed, there is no gastroesophageal reflux (43). It is likely that this mucosal arrangement is a result (rather than a cause) of tonic contraction of the intrinsic gastro-esophageal sphincter.

3. Diaphragmatic Pinchcock:

Jackson (35) suggested that fibres of the right crus of the diaphragm, which loop around the lower end of the esophagus, may act as a pinchcock preventing reflux of the gastric contents into the esophagus. Allison (49) & Peters (45) supported this hypothesis and emphasized the role of the phrenicoesophageal ligament which was thought to act as a centering mechanism by which the "hiatal noose" compressed the esophagus in the appropriate regions. The phrenicoesophageal ligament, a connective tissue structure, is said to arise from the inferior surface of the diaphragm and the hiatal margin, and is attached to the esophagus, not only at the cardia, but also above it (46, 47, 45). Both the development and the composition of the ligament are quite controversial. Some see the structure as tough and substantial, consisting of elastic, fibrous and fatty tissue with some striated muscle fibres; others suggest it has to "be dissected out with the eye

of faith". Although its very existence has been denied in human beings, there is no doubt it is important in the dog, since division of the phrenicoesophageal ligament in the dog frequently leads to massive herniation of the stomach into the chest (51).

To study the role of the diaphragm, Braasch (48) excised the gastroesophageal junction in dogs and placed the newly created gastro-esophageal junction below the diaphragm in one group and above the diaphragm in another group. He did not find any significant difference in the incidence of post-operative esophagitis or alteration of esophageal function in the two groups: all animals developed reflux and concurrent esophageal ulceration, suggesting that the diaphragm played no role in preventing reflux in this model. Bremner (49), on the other hand, showed that complete division of the phrenicoesophageal ligament in the dog did not produce a significant change in the function of the gastroesophageal junction (40). Unilateral phrenic nerve section is reported neither to affect the function of the cardia during swallowing, nor to permit gastroesophageal reflux (50). In dogs, bilateral phrenic nerve avulsion does not lead to gastroesophageal reflux. The above studies indicate that neither the diaphragm nor the phrenicoesophageal ligament play a significant role in preventing gastroesophageal reflux.

4. Abdominal segment of the esophagus:

Dornhorst (52) suggested that the terminal portion of the esophagus resembled a flaccid tube exposed to the extrinsic pressure of intra-abdominal contents, which maintained its walls in apposition, forming a valve. When intra-abdominal pressure was raised, the increase in pressure was transmitted both to the abdominal segment of the esophagus and to the gastroesophageal sphincter, so that the gradient between the

sphincter and the lower esophagus remained unchanged (26). However, Lind (53) showed that the increase in pressure in the junctional zone was greater than the increase in gastric and esophageal pressures, suggesting contraction of smooth muscle of the GE sphincter. The role of the intra-abdominal esophagus was evaluated in another study in dogs, where the gastroesophageal junctional zone was excised (54). A short intra-abdominal segment of esophagus did not prevent reflux. When a long segment of esophagus (4 cms. or more) was maintained below the diaphragm on the other hand, reflux did not occur, since manual compression of the abdomen failed to produce a simultaneous pressure increase in the thoracic esophagus (in 4 out of 5 animals). It appears, then, that a long segment of intra-abdominal esophagus may help in competence but a short segment plays no significant role. Conversely, in some cases of hiatal hernia where there is no abdominal esophagus, the competence is maintained by the lower esophageal sphincter which responds to abdominal compression (55).

5. Lower Esophageal Sphincter (LES):

Manometric studies have identified a zone of elevated pressure at the gastroesophageal junction (25, 26, 27, 1). Physiological responses of this zone strongly suggest an intrinsic sphincteric mechanism. This zone is now regarded as the intrinsic "physiological sphincter". The high pressure zone in most healthy persons is 2 to 4 cms. long when water-filled, open-tipped tubes are used as pressure sensors (28).

With swallowing the sphincter relaxes and then contracts. Generally, the lower esophageal sphincter relaxes 2 secs. after the upper esophageal sphincter, although in some cases relaxation begins

when the bolus is placed in the mouth, even before swallowing is started. In all cases, however, the lower sphincter relaxes 3 secs. or more before peristaltic contraction reaches it, so that the gastric entrance is open before the bolus arrives. There is a difference between the responses of the upper and lower portions of the gastro-esophageal sphincter (30). In the lower portion, relaxation is followed by restoration of resting tone only, whereas in the upper half relaxation is followed by contraction, producing a rise of pressure well above the resting tone. When the contraction subsides, pressure returns to resting level. This difference in the behavior of the upper and lower parts of the sphincter may be due to difference in the anatomical arrangement, for the sphincter is truly the junction of two organs.

Apart from the area of the high pressure zone, the entire esophagus in the dog consists of striated muscle. Nevertheless, the pressure profile of the esophagus and the lower esophageal sphincter in the dog is almost identical to that seen in human beings (8), in whom the entire lower third of the esophagus is usually smooth muscle. The distribution of skeletal and smooth muscle in the esophagus of cats and monkeys is similar to that in man, and the pressure sequences in these animals are also similar (31, 32).

Control of Lower Esophageal Sphincter Activity:

The resting pressure within the LES is normally higher than that of either the upper stomach or esophagus. This is true even of isolated preparations of the gastroesophageal region (79, 80). The mechanisms responsible for maintaining the high pressure within the LES, as well as its relaxation during deglutition, have not been defined clearly. The first question to be resolved is whether the LES is in a

state of tonic contraction in the absence of esophageal peristalsis. Hellemans and Vantrappen (81) studied this problem by recording the electrical activity from the LES of dogs at rest and during swallowing. They were not able to detect electrical activity in the caudal portion of the LES either at rest or during swallowing. However, brusts of spike activity were found to accompany peristalsis in the body of the esophagus and in the upper portion of the sphincter. More recently Arimori and Code (82) were able to record slow, continuous, phasic electrical activity from the distal segment of the LES, which was nearly always diminished or abolished by swallowing. It is not clear whether these phasic potentials, in the absence of deglutition, represent tonic activity of the LES. Firstly, contractile activity in other parts of the gastrointestinal tract is normally associated with spike potentials rather than with phasic potentials. Secondly, these potentials were recorded at time constants of 0.1 to 0.01 secs., which are 10 to 100 times shorter than the period of the recorded potentials. Such conditions would produce considerable distortion of the potentials picked up by the electrodes. Further studies are needed to clarify the nature and significance of these electrical potentials.

A number of control systems may regulate the activity of the sphincter. These may be classed as (1) Nervous and (2) Hormonal.

Nervous Control: Anatomical studies show that both vagal and sympathetic fibres supply the gastroesophageal junctional zone. The final distribution is so intricate and controversial that functional innervation of this area can hardly be decided by anatomic methods. (58, 59, 60). It is agreed, however, that the vagus has some influence on the lower end of the esophagus and LES. Radical sympathetic

extirpation in the dog had no effect on the lower esophagus or LES (61). In the cat sympathectomy had little effect on esophageal motility but caused marked diminution in the resting pressure of the inferior sphincter (62).

The effects of vagotomy both in the dog and the human being have been studied in some detail in recent years. Bilateral supra-nodosal vagotomy in the dog led to dilatation of the esophagus with absent peristaltic contractions, reduced tone in the lower esophageal sphincter and failure to relax with deglutition (63). In appearance and function the esophagus closely resembled that of human achalasia. Bilateral subhilar vagotomy reduced both the resting tone of the lower esophageal sphincter and the incidence of relaxation in response to deglutition (64). Vagotomy performed through the diaphragm at or just above the gastroesophageal junction reduced the tone of the lower esophageal sphincter but did not affect the ability of the sphincter to contract or relax (65).

The sphincter appears to be a dynamic organ which responds to a number of stimuli. As measured by intraluminal pressure sensors, its tone varies, not only with deglutition but also with alterations in intragastric and intra-abdominal pressure. When intra-abdominal pressure is raised by abdominal compression, the lower esophageal sphincter responds by increasing its tone (53). This response is probably mediated by long vago-vagal reflexes arising in the stomach, since vagotomy at the gastroesophageal junction in the dog abolishes this response (40). A similar response has been shown to accompany an increase in intragastric pressure in the decerebrate cat (83, 84). These observations are further supported by studies in patients who

underwent sub-diaphragmatic vagotomy for peptic ulceration. Following vagotomy there was no significant change in the resting tone of the sphincter, but when subjected to abdominal compression the response of the sphincter was significantly reduced (66). Lind et al (53) regard the response of the sphincter to abdominal compression a better test of its integrity than the study of resting tone.

Subcutaneous injection of atropine in normal human subjects caused a significant reduction in the sphincteric pressure, both under resting conditions and during abdominal compression, without affecting pressures in the stomach or esophagus (78). From these observations it was concluded that a cholinergic mechanism, mediated by vagi, was partly responsible for the resting tone of the sphincter and its response to abdominal compression. After complete sympathectomy and subhilar vagotomy the intrinsic sphincter still retained some tone although it was reduced to the extent expected by vagotomy (61). Esophageal section at various levels produced the same change as expected by concomitant vagotomy at that level. It can, therefore, be established that the intrinsic lower esophageal sphincter has inherent tone which is modified by the vagi and perhaps to a minor extent by the sympathetic nerves. It can relax and contract in coordination with deglutition without the help of extrinsic nerves and thus seems to enjoy a considerable degree of autonomy.

Hormonal Control: Cannon (85) was the first to demonstrate that an increase in gastric acidity promoted closure of the cardia so that higher intragastric pressure was required to produce reflux. Similar results were obtained by Clark and Vane (86) during perfusion of the LES with 0.1 N HCl. In both cases the increase in sphincter tone was

unaffected by bilateral vagotomy, which suggested a hormonal basis for the response. Slightly different results were obtained by Giles et al (69) following irrigation of the cardiac mucosa with HCl in human beings. Although the sphincteric pressure increased in response to gastric acidification, the response was virtually abolished after injection of atropine. This apparent discrepancy may have been resolved by Titchen and Wheeler (84) who showed that increased sphincter tone may be produced by acid perfusion of both the caudal esophagus and the cardiac mucosa. Only the latter is vagally mediated, so that the earlier results may be due to acid reflux from the stomach into the lower esophagus.

Giles also showed that both synthetic and hog gastrin stimulated the sphincter in humans and endogenous gastrin had a similar effect (68). Castell and Harris (70) suggested that gastrin had a direct physiological action on the LES. Endogenous release of gastrin by antral alkalinization increased the sphincter tone while inhibition of endogenous gastrin by acidification of stomach (presumably distal to the cardia) had the opposite effect. Both endogenous and synthetic gastrin have also been shown to stimulate contractions of isolated longitudinal and circular muscle of the lower esophageal sphincter (86). The above studies indicate that in the normal human being gastrin can increase the LES pressure, both directly by acting on the sphincter, and indirectly by stimulating acid secretion.

More recent studies indicate that secretin and CCK also interact with gastrin in regulation of LES competence (87). Secretin inhibits the action of gastrin on the sphincter, probably by competing for the same receptor sites, while CCK appears to potentiate the action of gastrin.

RESTATEMENT OF THE PROBLEM

A critical review of the literature leaves no doubt that the intrinsic lower esophageal sphincter is the chief mechanism which prevents reflux of gastric contents. However, relatively little is known about the mechanisms which normally control its function. Sympathetic control appears insignificant. Vagi may partly control the resting tone of the sphincter and its response to changes in intra-abdominal pressure. Recent studies indicate that gastrin increases the sphincteric pressure, with CCK promoting its action and secretin opposing it.

The etiology of gastroesophageal reflux also remains an enigma. There is some evidence that the lower esophageal sphincter is abnormal in hiatus hernia associated with reflux (55). Resting pressure is usually lower than normal and the sphincter does not contract in response to abdominal compression.

A number of operations have been devised to correct gastro-esophageal reflux in human beings. In many of these operations there is little scientific evidence to support their efficacy, and the failure rate is accordingly high (92, 93). Even those authors who claim a good success rate, however, have only clinical follow-up evidence to testify to the usefulness of their procedures. Experimental studies designed to elucidate the mechanisms which restore competence at the gastro-esophageal junction have been far from satisfactory. In these studies the disease was reproduced in animals, either by myotomy (57) or by complete excision of the lower esophageal sphincter, after which different kinds of repair were performed (67). This situation obviously is very remote from human reflux esophagitis.

Our own animal model, on the other hand, with hiatus hernia and intact though weakened sphincter, whose construction will be described later, bears a fair likeness to the human situation. Using this model, we shall attempt to answer the following questions:

1. Is the function of the lower esophageal sphincter affected by changing its location in relation to the diaphragm?
2. Do reparative procedures such as Belsey and Allison operations (89, 49) restore gastroesophageal competence, and if so, how?

The following mechanisms will be considered:

a) Restoration of a Subdiaphragmatic position of the lower esophageal sphincter.

b) Flap-valve effect of the repair on the GE junction.

c) Increase in the length of the high pressure zone.

d) Improvement in the intrinsic function of the sphincter.

METHOD

METHOD

Twenty-four mongrel dogs, weighing 12 to 22 kgms. were used in this study. They were immunized against Distemper and Rabies, and given a course of an antihelminthic drug. The dogs were trained to lie quietly and to accept the passage of a tube into the stomach during manometric studies, Hollander's test, and cinefluoroscopy. The dogs were weighed at regular intervals and difficulties in swallowing and vomiting, if any, were noted.

The experiments were conducted in the following stages:

STAGE I: Pre-operative Studies

1. Four manometric studies were done on each dog on separate days.
2. Hollander's test was performed to show the integrity of the vagi.
3. Cinefluoroscopic examination of the esophagus and the stomach was performed to confirm the normal appearance and position of the GE junction.
4. Esophagoscopy and esophageal biopsy before surgery.
5. A subdiaphragmatic truncal vagotomy and Heineke-Mikulicz type of pyloroplasty was performed. The excised tissue was examined microscopically to confirm the presence of nerve tissue.

STAGE II: Post-vagotomy Studies

A period of two to four weeks was allowed for recovery from the operation, and the studies were repeated as follows:

1. Four manometric studies on each dog on separate days.
2. Hollander's test to check the completeness of vagotomy.
3. Cinefluoroscopy of gastroesophageal region.
4. Esophagoscopy and biopsy.

A thoracotomy was then performed and the gastroesophageal junction

was displaced four cms. into the mediastinum, thus creating a hiatus hernia.

STAGE III: Post-thoracotomy Studies

Four to six weeks after thoracic displacement of the GE junction the studies were repeated as follows:

1. Four manometric studies on each dog on separate days.
2. Cinefluoroscopic examination.
3. Esophagoscopy and biopsy.

The 24 dogs were then randomly divided into 4 groups of 6 each.

Group A Underwent repair of hiatus hernia by Allison's method (44), which consisted of reduction of the gastroesophageal junction below the diaphragm and crural repair.

Group B Underwent a modified Belsey repair (56).

Group C Underwent a modified Belsey repair, with retention of the gastroesophageal junction in the chest, about 4 cms. above the diaphragm.

Group D Underwent a thoracotomy with dissection and resuture of the stomach in the hiatus, i.e. a sham procedure.

STAGE IV: Post-repair Studies

Four to six weeks after the above operations, all the dogs were studied as follows:

1. Four manometric studies were performed on separate days.
2. Cinefluoroscopic studies of the esophagus and stomach were repeated.
3. If a dog died, autopsy was performed to determine the cause of death, to assess the integrity of repair, and to examine the esophageal mucosa for evidence of esophagitis.

MANOMETRIC STUDIES

An adaptation of the method described by Brody & Quigley (71) was used to measure true intraluminal pressures as an index of motor activity. The open-tip catheter with external transducer system provides a stable device for simultaneous recording of pressures from multiple sites. The strain gauge transducers have a wide range of sensitivity, with a frequency response faithful up to 100 cycles/sec., and the volume/pressure coefficient of 0.0001 mls. per cm. of water pressure or lower. Similar equipment has been used by Ingelfinger, Loher and Lind (72, 73, 55).

Three polyethylene tubes (PE 190, I.D. 0.047", O.D. 0.067"), 170 cms. in length and fused at their tips were used as pressure sensors. Each tube had a single lateral orifice, 2 mms. in diameter placed 5, 10 and 15 cms. from the fused tips. The lumen of each tube immediately distal to the lateral opening was occluded by a small plastic plug. A fourth polyethylene tube (PE 60, I.D. 0.031" O.D. 0.048") fitted with a piano wire was attached to the assembly to facilitate its passage and prevent kinking during the study. The proximal end of each tube was connected through a three-way stop-cock to a strain gauge (Stathan P23 DE), the resistance modulations of which were recorded by a multichannel recorder - Visicorder 1508 (75). The speed of the recording paper was generally 1 mm/sec. to 2 mm/sec., and the sensitivity of the recording was adjusted to represent 10 cms. water pressure/cm. deflection.

Through the three-way stop-cocks, the strain gauge chambers and recording tubes were filled with water at room temperature and infused constantly at the rate of 1.8 mls./min./tube by means of a

Harvard dual reciprocal infusion-withdrawal rump (76). The system was calibrated before and after each test by means of a water manometer connected to the strain gauges through another three-way connector, to record a deflection of 1 cm. with an increase or withdrawal of 10 cms. water pressure from the system. The strain gauges were kept at the level of the lower esophagus to eliminate the effect of hydrostatic pressure.

Each dog was fasted for at least twelve hours prior to each manometric study. The tests were carried out on a specially constructed table which allowed the dog to lie comfortably on its side, with the head extended to facilitate the movement of the tubes. Respiration was monitored by a tubular pneumograph placed around the chest and connected to the recorder through a pressure transducer (PM 5TC). For abdominal compression a pneumatic cuff, connected to a manometer, was placed around the abdomen and secured by a tape. A short padded metal bar with two holes in the middle was fastened between the jaws behind the canine teeth, and then anchored to the table. The polyethylene pressure-tube assembly was passed through a hole in the bar and advanced until all the tubes were in the stomach. After a brief period of adjustment to infusion, a continuous recording was made while the tubes were withdrawn at 1.0 cms. intervals from the stomach into the GE sphincter, and then at 0.5 cms. intervals into the body of the esophagus. At each location the pressures were recorded until they stabilized. When the resting "pull-through" was completed, the tubes were again introduced into the stomach and another recording obtained while the abdominal cuff was inflated to 70 mmHg. pressure.

Measurement of Pressures from the Recordings:

The recordings represented pressures from three tubes, a

pneumograph and an event marker (Fig. 10). The pressures fluctuated with respiratory excursions. With inspiration, a positive or upward deflection was seen when the units were in the abdomen, and a negative or downward deflection when the units were in the chest. The point at which the inspiratory deflection changed from upward to downward, or point of respiratory reversal (PRR) was taken as the level of the effective diaphragmatic hiatus. The pressures were measured from a base line, obtained by infusing the tubes outside the body, with the strain gauges and the tubes positioned at the level of the gastro-esophageal junction. The mean of the end-inspiratory and end-expiratory points was taken as actual pressure. The fundic pressure was taken at a stable point two to three cms. distal to the high pressure zone, and the esophageal pressure at a point three to four cms. proximal to it. The sphincteric pressure, measured at a stable point in the station showing maximum overall pressure, was taken as mean maximal resting pressure (Fig. 10). A sharp and brief elevation of pressure was considered a contraction. All measurements were taken from the most distal tube, the other two tubes being used for reference. If a dog swallowed while the distal tube was in the GE sphincter, enough time was allowed for the pressure to return to the pre-deglutition level before pulling the tube proximally.

HOLLANDER'S (INSULIN) TEST:

The physiological basis of the test is as follows: Insulin-induced hypoglycemia stimulates gastric secretion when the vagal supply to the stomach is intact but fails to do so after complete vagal section. With Hollander's (77) criteria for interpretation of the results, this test was used to determine the completeness of vagotomy.

The pre-operative test was performed on each dog to establish the integrity of the vagi. Two to four weeks after vagotomy the test was repeated to check the completeness of vagotomy.

The fasting dog was positioned on the table in the same way as for a manometric test. A Levine tube was passed into the stomach and its tip was positioned in the antrum. The gastric contents were aspirated continuously with a suction apparatus at a negative pressure of ten to twenty cms. of water. The patency of the tube was constantly checked and maintained. The first sample of gastric juice was collected for half an hour and subsequently four samples were collected, each for 15 minutes. Venous blood was withdrawn initially after one half hour to determine the resting blood glucose level. Soluble Insulin in a dose of 1.2 units/kg. body weight, was then injected intravenously and samples of blood for glucose estimation were collected every fifteen minutes for the next two hours.

All samples of gastric juice were tested for pH, total volume, acid concentration, and acid content calculated in mEq. per volume. Hollander test was considered negative if the volume of gastric juice and acid output did not increase in response to hypoglycemia. If the specimen was insufficient for titration, a drop in pH was taken as evidence of response to hypoglycemia.

CINEFLUOROGRAPHY:

Motor function of the esophagus and the lower esophageal sphincter, and the position of the gastroesophageal junction were studied by fluoroscopy and recorded on a cine film and on single-frame x-ray plates.

The fasting dog was placed on its side on a flat board, gently

restrained and secured to the x-ray table. A sphygmomanometer cuff was loosely fastened around the animal's abdomen. The jaws were fastened to a perforated, padded metal bar and anchored to the table. A Levine tube was passed through the centre hole in the bar and advanced into the upper esophagus. A thin mixture of Barium Sulphate was then injected through the tube in 25 ml. boluses. Peristalsis, with passage of Barium down the esophagus, through the cardia and into the stomach, was observed on the fluoroscopy screen and filmed. After 200 mls. of Barium Sulphate were placed in the stomach the abdomen was compressed to 70 mmHg. pressure by inflating the sphygmomanometer cuff. Spontaneous reflux, if any, was noted. Esophageal peristalsis was then initiated by injecting 20 ml. of air into the upper esophagus, and finally the x-ray table was tilted to 45 degrees head down position in an attempt to induce reflux. Cineradiography and single frame plates provided a permanent record of the pertinent points of the examination.

OPERATIVE PROCEDURES:

The dogs were fasted for at least 18 hours before each operation. Physical examination was carried out and the weight recorded before anaesthesia, which consisted of six per cent Sodium Pentobarbitone (30 mg./Kg.) injected intravenously. Respiration was maintained by a positive pressure respirator, which administered an air-oxygen mixture via an endotracheal tube. An intravenous infusion of normal saline was maintained throughout the operation. The quantity of saline infused during surgery depended upon the amount of blood loss: about 500 mls. were required during vagotomy and pyloroplasty, and 750 to 1000 ml. during the thoracotomy procedures.

Post-operatively, the animal's respirations were assisted by the respirator until it was ventilating adequately spontaneously. It was observed in the operating room till consciousness and the cough reflex returned. The endotracheal tube was removed immediately before transferring the animal to the kennel.

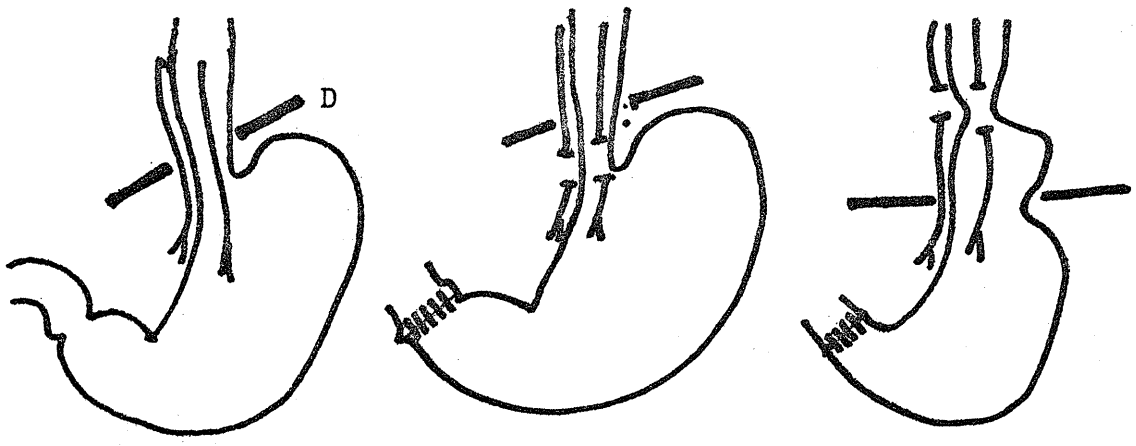
During the first 48 hours after surgery only water was allowed orally. The feeds were gradually thickened with milk and meat until the seventh to tenth day, when normal diet was resumed. No antibiotics were given unless there was definite evidence of infection.

VAGOTOMY AND PYLOROPLASTY:

The dog was anaesthetized and placed on the operating table in the supine position. The site of operation was shaved, washed and painted with alcoholic solution of 0.1% Thimerosal (Ingram & Bell Ltd. Canada).

The abdomen was opened through a midline incision extending from the xiphisternum to an inch below the umbilicus. After general exploration of the abdominal viscera, the esophageal hiatus was displayed. The peritoneum and phrenicoesophageal ligament were incised at the hiatus. The vagi were identified, dissected, and a 2 cms. segment of each nerve was excised from immediately above the gastroesophageal junction. The cut ends of the nerves were either ligated or secured with silver clips. Accessory vagal fibres were sought and divided.

Pyloroplasty was performed by Hieneke-Mikulicz's method. The pylorus was opened by a 4 cms. longitudinal incision, extending for two thirds of its length over the anterior wall of stomach and one third over the anterior duodenal wall. The incision was then sewn transversely using a single layer of interrupted 000 silk sutures.



STAGE I STAGE II STAGE III

Figure 1. Operative Procedures. Diagrams of the Anatomical Situation during the First Three Stages. Stage I, Pre-operative: Gastroesophageal Junction is located below the Diaphragm and the Vagus Nerves are intact. Stage II, Post-vagotomy: the Nerves have been divided at the Gastro-esophageal Junction and a Pyloroplasty has been performed. Stage III, Post-thoracotomy: the vagotomized Gastroesophageal Junction is displaced into the Thorax.

The abdomen was closed by suturing the linea alba and the skin in layers with 00 interrupted silk.

THORACIC DISPLACEMENT OF THE GE JUNCTION:

The anaesthetized animal was positioned on the table on its right side. An incision was made along the left ninth rib extending from the costochondral junction anteriorly to the sacrospinalis muscle posteriorly. After incising the overlying muscles the rib was dissected subperiosteally and the pleural cavity entered through the rib-bed. A self-retaining retractor was used to expose the left pleural cavity.

The pleura over the lower esophagus was incised longitudinally down to the diaphragm and the esophagus was mobilized, preserving the vagi. The gastroesophageal junction was then isolated from the diaphragm by dividing adhesions and the remains of the phrenicoesophageal ligament. With gentle traction on the lower esophagus, the GE junction and proximal portion of the stomach were displaced into the chest. Often it was necessary to divide the upper short gastric arteries and some branches of the left gastric vessels. The gastroesophageal junction was retained in the chest by suturing the diaphragm to the stomach circumferentially 4 cms. below the GE junction. After complete haemostasis a chest tube was inserted through a small incision in the intercostal space immediately below the main incision. The thoracic cavity was closed in two layers by approximating the divided muscles and skin. During recovery from the anaesthesia 20 cms. of water suction was applied to the intercostal tube to remove the air and fluid, and to allow re-expansion of the lung. The intercostal tube was removed after the lungs were completely inflated and the dog was breathing adequately. The animal was then weaned off the respirator and returned to the kennel.

Post-operative management was the same as after vagotomy.

MODIFIED BELSEY REPAIR:

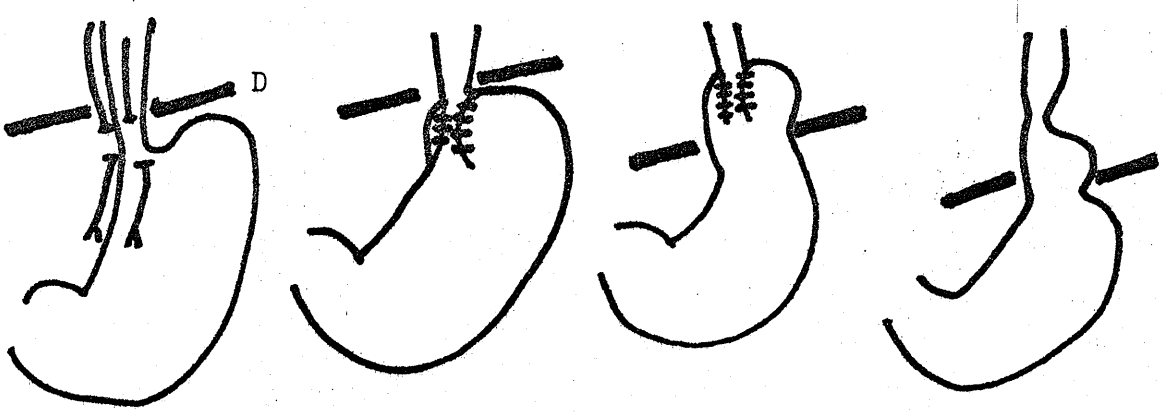
The left pleural cavity was exposed through the tenth rib bed. The size of the previously created hiatus hernia was noted, and the lower esophagus was mobilized. The stomach was then dissected off the diaphragmatic hiatus by dividing fibrous adhesions and silk sutures. The fundus of the stomach was wrapped around three fourths of the circumference of the lower three to four cms. of esophagus. This was then sutured to the esophagus with multiple interrupted sutures in three longitudinal rows (Fig. 2b). Occlusion of the esophageal lumen was prevented by prior insertion of a Levine tube into the esophagus. The gastroesophageal junction was then replaced in the abdomen and anchored to the inferior surface of the diaphragm with two silk sutures. The crura were approximated with interrupted silk sutures to narrow the hiatus to normal size.

MODIFIED BELSEY REPAIR WITH INTRATHORACIC GASTROESOPHAGEAL JUNCTION:

The procedure was as described above except that after repair, the gastroesophageal junction was not returned to a subdiaphragmatic position but retained in the thorax. The body of the stomach was sutured to the diaphragm to prevent further displacement of the gastro-esophageal region. This placed the sphincter with its fundic cuff about four cms. above the diaphragm.

CRURAL REPAIR:

In this group reconstruction of anatomy was similar to that described by Allison (44). The left pleural cavity was opened through the tenth rib bed. The fundus was freed from its attachment to the hiatus, and the lower esophagus and gastroesophageal junction were



GROUP A GROUP B GROUP C GROUP D

Figure 2. Stage IV. Reparative Procedures. Diagrams of Anatomical Situation after Three Types of Repairs and the Sham Operation. Group A, Abdominal Replacement of the GE Junction and Crural Repair. Group B, Abdominal Replacement of the GE Junction and Fundic-wrap. Group C, Fundic-wrap with Retention of the GE Junction in the Thorax. Group D, Sham Operation; the Fundus and the GE Junction are Dissected off the Diaphragm and the Fundus is Resutured to the Diaphragm.

mobilized from the neighbouring structures. The gastroesophageal junction was replaced below the diaphragm and the esophagus sutured to the hiatus about two cms. above the GE junction. The crura were approximated behind the esophagus with interrupted silk sutures placed 0.5 cms. apart. The hiatus was tightened until the tip of the index finger only could be passed alongside the esophagus. The closure of the thoracic cavity and post-operative management were as previously described.

SHAM PROCEDURE:

The left pleural cavity was opened through the tenth rib bed. The fundus of the stomach was completely freed from the hiatus, and the lower esophagus was mobilized. The stomach was then re-sutured to the diaphragm about four cms. distal to the gastroesophageal junction. The thoracotomy closure and the after-care were identical to that of the other repairs.

RESULTS

Twenty-three dogs were studied through all stages of the experimental protocol. Only those dogs who developed gastroesophageal incompetence were retained for study of the different repair procedures. One dog was excluded because of breakdown of the Belsey repair, and was replaced by another dog from the Sham group. After studies had established that no significant change was produced by Sham operation, a Belsey repair was performed, and results of subsequent studies on this dog were included in the Belsey group. The values from this dog are therefore included both in the Sham and in the Belsey groups.

A total of 40 dogs were operated upon, of which 16 died before completion of the study, and were replaced randomly by new animals which were again studied from Stage I.

OPERATIVE MORTALITY

The most common cause of death was massive intrathoracic herniation of the upper abdominal viscera through the esophageal hiatus (Table 1). This usually occurred after subhiatal vagotomy, which involved complete division of the phrenico-esophageal ligament. The ligament is a well-defined structure in the dog, and although not important in maintaining competence at the GE junction (40), it seems to maintain the GE junction below the diaphragm. Three dogs died of distemper, two from anesthetic mishaps, two from infection and three from other causes.

TABLE 1

Analysis of Operative Mortality

Cause of Death	Numbers
Massive intrathoracic herniation of abdominal viscera	6
Distemper	3
Anesthetic deaths	2
Infection	2
Haemorrhage	1
Undetermined	2
Total	16 (40) = 40%

HOLLANDER TEST

Before surgery, integrity of the vagi was demonstrated by a positive Hollander test in all dogs. Following vagotomy, the Hollander test was repeated to confirm the completeness of vagal section. The usual criteria for interpretation of the Hollander test were applied, except when there was contamination of the gastric juice with bile, or where sufficient quantity was not available for titration. Under these circumstances, if there was no significant drop in the pH during the hypoglycemic phase, the test was considered negative.

All vagotomized dogs showed a negative response to hypoglycemia, except Dog 15 where the test was equivocal (Table 2).

TABLE 2

Results of Hollander's Insulin Test in 23 dogs

Group and dog number	Control	Post-Vagotomy
Group A		
11	+	-
31	+	-
15	+	±
12	+	-
19	+	-
18	+	-
Group B		
17	+	-
7	+	-
16	unsatisfactory test	-
3	+	-
25	+	-
24	+	-
Group C		
28	+	-
4	+	-
23	+	-
1	+	-
29	+	-
8	+	-
Group D		
24	+	-
6	+	-
14	+	-
10	+	-
13	+	-
9	+	-

+ Positive - Negative

WEIGHTS

All the animals were weighed at least once at every stage of the study. The animals regurgitated frequently in the post-vagotomy period, but within 2-3 weeks normal eating was restored. Before the next operation most animals had either recovered their normal weight or had gained some weight. Following intrathoracic displacement of the gastroesophageal junction, regurgitation was worse but did not prevent the animals from eating. Most dogs lost weight in the immediate post-operative period but gradually regained it, and weighed close to normal when the hiatus herniae were repaired. Only Dog 24 lost a significant amount of weight from continuous regurgitation through an incompetent LES. However, following a modified Belsey repair, it regained its normal weight. Neither the reparative procedures nor the Sham operations produced a significant change in the weights of the animals (Table 3).

TABLE 3

Weights of the animals in Kilograms at Different Stages of the Study

Group & Dog number	Control	Post-Vagotomy	Post-thoracic displacement	Post-repair
Group A				
11	20.5	20.9	21.0	21.1
31	12.2	12.2	12.5	12.9
15	11.7	11.9	12.0	12.6
12	15.0	16.0		19.1
19	14.8	17.0	16.9	25.2
18	15.5	15.7	14.6	15.8
Group B				
17	14.6	15.1	14.4	19.2
7	12.0		17.8	21.6
16	23.2	23.0		24.7
3	16.1	19.2	18.2	23.5
25	14.6	15.1	15.6	15.2
24	13.3	14.0	10.2	13.7
Group C				
28	20.0	20.7	20.6	23.0
4	13.0	12.9	13.5	15.4
23	16.4	17.4	15.6	14.8
1	20.5	17.1	22.0	22.5
29	21.6	20.9	20.2	
8	23.3	22.11	20.8	23.0
Group D				
24	13.3	14.0	12.8	10.2
6	18.9	22.0	17.8	18.3
14	13.0	13.6	14.0	
10	13.0	14.0	17.0	19.2
9	23.0	20.0	25.0	28.4
13	16.2	15.5	17.0	

At each stage of the study the stomach, gastroesophageal junction and lower esophagus were examined by cinefluorography, manometry, esophagoscopy and esophageal biopsy. The results were as follows:

STAGE I: Control Studies

Cinefluorography

In all dogs the esophagus appeared normal and the GE junction was located below the diaphragm. The peristaltic wave proceeded smoothly down the esophagus till the Barium dropped into the stomach. No gastroesophageal reflux was noted either at rest or during application of 70 mm.Hg. pressure to the abdomen in a head down position (Table 4).

In two animals a knuckle of stomach appeared above the diaphragm on abdominal compression but returned below the diaphragm when the pressure was released. Neither of these dogs showed any evidence of hiatus hernia at vagotomy.

TABLE 4

Results of Cinefluorographic Studies in 23 Dogs

Stages of Study	Spontaneous Reflux	Reflux with Compression	Total Showing Reflux
Control	0	0	0 (23)
Post-vagotomy	6	9	9 (23)
After thoracic displacement of GE junction	16	22	22 (23)*

* One animal missed the study.

Manometry

Three to four manometric records were available from each dog for analysis. The pressure profiles of all dogs revealed a high pressure zone between the stomach and the body of the esophagus. This zone, located partly below the diaphragm, had a mean length of 2.64 ± 0.08 cms. (SE).

The following intraluminal pressures were measured at rest and during abdominal compression:

(i) Mean fundic pressure

(ii) Mean maximal sphincteric pressure

(iii) Mean esophageal pressure

(iv) Stomach-to-sphincter gradient

At rest mean pressures in cms. of water were 6.71 ± 0.29 (SE) in the stomach, 26.68 ± 1.67 in the sphincter, and -0.14 ± 0.2 in the lower esophagus. The application of abdominal compression increased the mean pressures to 20.91 ± 0.96 in the stomach, 54.49 ± 2.48 in the sphincter, and 1.0 ± 0.27 in the lower esophagus (Table 5). The stomach-to-sphincter gradient increased, from 20.04 ± 1.56 cms. of water at rest to 33.44 ± 2.25 under compression, which represents an increase of 13.4 cms. of water ($P < .001$) in the gradient. This change is attributed to an increase in the mean maximal sphincteric pressure, which was greater than the increase in pressure in the stomach, and which is the normal response of the LES to abdominal compression, as described by Lind (53). It is considered a better measure of LES function than the resting pressure.

Esophagoscopy and Esophageal Biopsy

The esophageal mucosa was inspected through an esophagoscope under general anaesthesia and a mucosal biopsy was taken from the area just above the cardia. All animals showed a normal esophageal mucosa, both on

Table 5

Comparison of Mean Intraluminal Pressures in Cms. of Water, Control vs. Vagotomy (24 Dogs)

	CONTROL	SIGNIFICANCE OF DIFFERENCE	VAGOTOMY
Pressures at rest			
Stomach	6.71 ± 0.29	$P<.02$	7.56 ± 0.37
Lower Esophageal Sphincter	26.68 ± 1.67	$P<.01$	21.43 ± 1.14
Esophagus	-0.14 ± 0.21		0.04 ± 0.19
Gradient between Stomach and Sphincter	20.04 ± 1.56		16.62 ± 1.44
Pressures under Compression			
Stomach	20.91 ± 0.96		22.68 ± 1.15
Lower Esophageal Sphincter	54.49 ± 2.48	$P<.001$	37.8 ± 1.55
Esophagus	1.0 ± 0.27		1.33 ± 0.23
Gradient between Stomach and Sphincter	33.44 ± 2.2	$P<.001$	14.95 ± 1.45

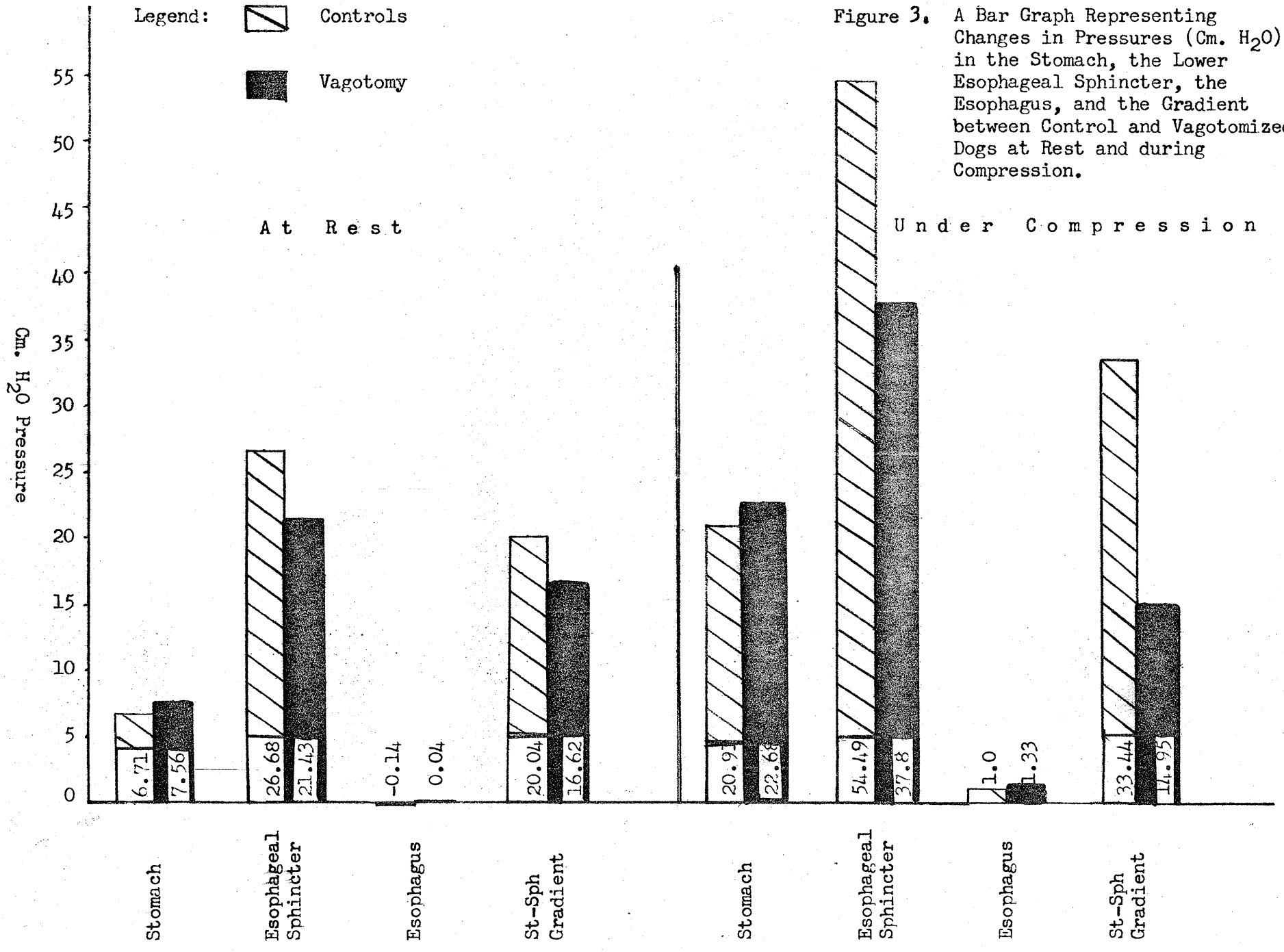


Figure 3. A Bar Graph Representing Changes in Pressures (Cm. H_2O) in the Stomach, the Lower Esophageal Sphincter, the Esophagus, and the Gradient between Control and Vagotomized Dogs at Rest and during Compression.

inspection and on histology.

STAGE II: Post-Vagotomy Studies

Six to eight weeks after vagotomy and pyloroplasty the studies were repeated and the following results were obtained:

Cinefluorography

Twenty-three dogs underwent cinefluorographic examination. In all cases, the esophagus appeared grossly normal with a normally-located gastroesophageal junction. A total of nine dogs showed gastro-esophageal reflux, six spontaneously and three with abdominal compression. When the animals were randomly divided into four groups for the study of reparative procedures, one dog showing gastroesophageal incompetence was in Group A, two in Group B, three in Group C, and three in Group D (Table 12, Appendix).

Manometry

The mean gastric, sphincteric and esophageal pressures, and the stomach-to-sphincter gradient, were measured and compared with pre-operative values. A preliminary analysis of variance showed significant differences between the animals in each group. Therefore each dog was used as its own control for all comparisons. The paired t-test was used for comparison values obtained at each stage.

After vagotomy the mean resting pressures in cms. of water were 7.56 ± 0.37 in the stomach, 21.43 ± 1.14 in the sphincter and 0.04 ± 0.19 in the lower esophagus. On compression the mean pressure in the stomach was 22.68 ± 1.15, in the sphincter 37.84 ± 1.55, and in the esophagus 1.33 ± 0.23. The stomach-to-sphincter gradient was 16.62 ± 1.44 at rest and 14.95 ± 1.45 during compression.

When these values were compared with pre-operative values (Table 5) a significant change was noted in the mean maximal sphincteric pressure, which decreased at rest by 5.25 cms. of water ($P < .001$) and under compression by 16.69 cms. of water ($P < .001$). The decrease in the sphincteric pressure was reflected in stomach-to-sphincter gradient, which also decreased by 3.48 ± 1.14 cms. of water ($P < .001$) at rest and 18.49 ± 2.44 ($P < .001$) during compression. A comparison between post vagotomy resting and compression gradients (16.62 vs. 14.95) indicates complete abolition of the LES response to compression.

The mean length of the lower esophageal sphincter after vagotomy was 2.60 ± 0.09 cms, as compared to 2.64 ± 0.08 before operation; this change was statistically insignificant.

Esophagoscopy and Biopsy

Gross examination of the esophageal mucosa at esophagoscopy failed to show any evidence of esophagitis in any animal, and mucosal biopsies from the lower end of the esophagus were reported normal on microscopic examination.

In summary, subdiaphragmatic vagotomy reduced the mean maximal pressure in the LES, lowered the stomach-to-sphincter gradient both at rest and during compression, and decreased the LES response to compression. Although these changes occurred in all animals, only nine showed gastro-esophageal reflux during cinefluorography, suggesting that the latter was an imperfect measure of GE competence.

The LES pressure and the stomach-to-sphincter gradient showed considerable variation from day to day and responded to a host of different stimuli. It is therefore likely that some dogs demonstrate incompetence only periodically, and that this will be missed unless

unless cinefluorography is done during that period. In any event vagotomy was not enough to produce GE incompetence in all the animals.

STAGE III: Studies after Thoracic Displacement of the GE Junction

Four to six weeks after displacement of the GE junction above the diaphragm, the tests were repeated. By this time the animals were on a normal diet and had almost regained their normal weight. The following results were obtained:

Cinefluorography

All the dogs with one exception underwent cinefluorographic examination at this stage. Supradiaphragmatic displacement of the GE junction and 4 to 6 cms. of stomach was confirmed in all animals. Gastroesophageal reflux was readily demonstrated in all animals, in sixteen spontaneously and in six under abdominal compression.

Manometry

The manometric records showed two high pressure zones three to four cm. apart, with an intervening plateau and often a double respiratory reversal. The distal high pressure zone was short and represented the diaphragmatic hiatus. The proximal high pressure zone, representing the lower esophageal sphincter, had a mean length of 1.87 ± 0.09, significantly less than that at Stage II ($P < .001$).

The resting mean intraluminal pressures in cms. of water were 7.64 ± 0.37 in the stomach, 16.99 ± 0.95 in the lower esophageal sphincter, and 0.23 ± 0.25 in the esophagus. On abdominal compression the mean pressures were 19.45 ± 1.08 in the stomach, 27.74 ± 1.23 in the sphincter and 2.01 ± 0.31 in the lower esophagus. The stomach-to-sphincter gradient was 9.43 ± 0.82 at rest and 8.26 ± 0.99 during compression.

When these values were compared with those after vagotomy the most significant changes were found in the LES pressure, which decreased by 4.42 cms. of water ($P < .001$) at rest and 10.07 ($P < .001$) during abdominal compression, and in the stomach-to-sphincter gradient, which decreased by 4.52 cms. of water ($P < .001$) at rest and 6.69 ($P < .001$) during compression (Table 6). The gastric pressure at rest did not change significantly, but during abdominal compression decreased by 3.23 cms. of water ($P < .01$). The mean esophageal pressure did not change significantly at rest, but during compression increased by 0.68 cms. of water ($P < .05$).

Esophagoscopy and Biopsy

Although all animals demonstrated gastroesophageal reflux during x-ray studies, none showed any evidence of esophagitis on esophagoscopy or mucosal biopsy.

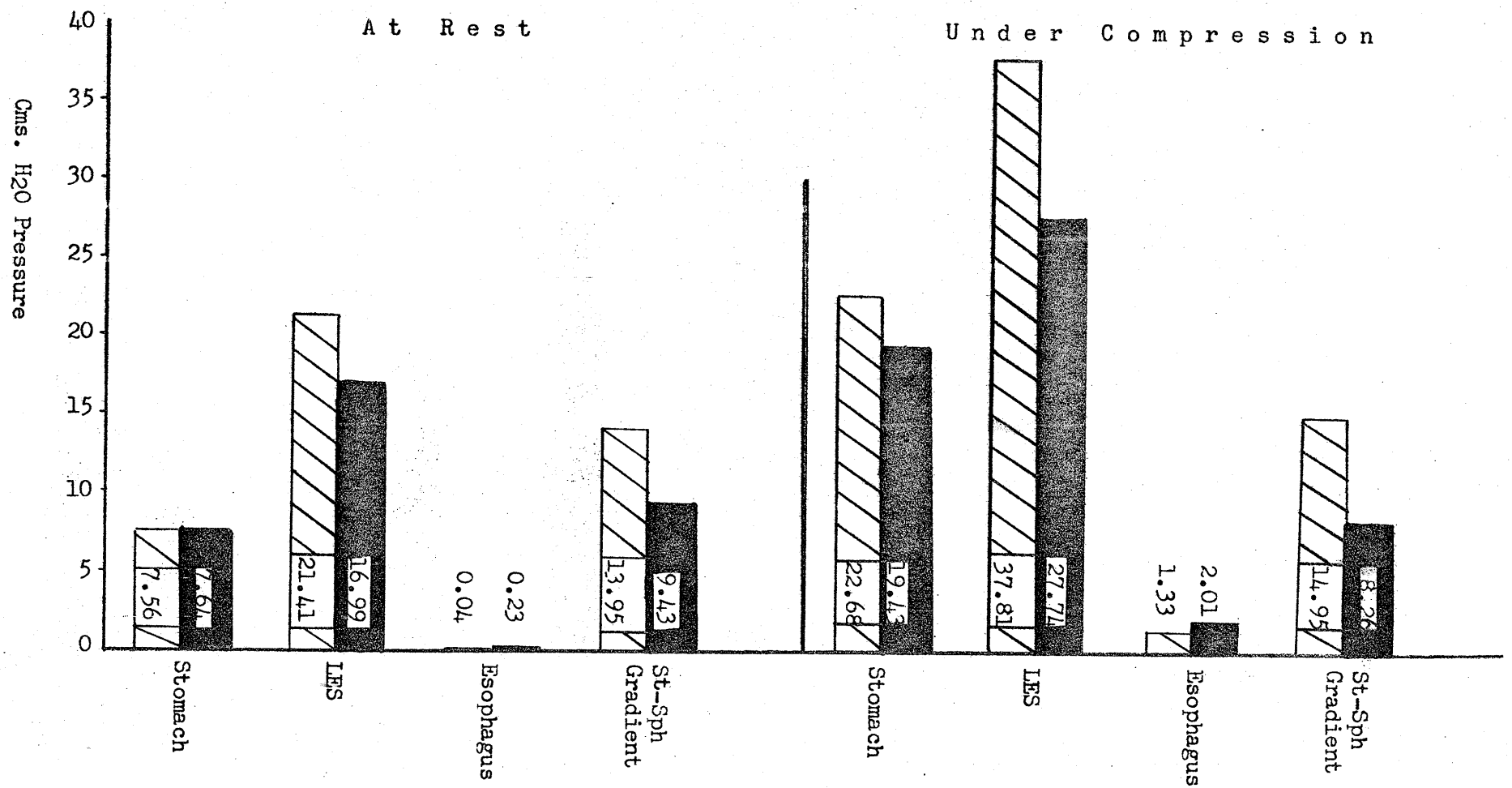
In summary, thoracic displacement of the vagotomized LES resulted in a decrease in its length and a fall in its intraluminal pressure and consequently the stomach-to-sphincter gradient. In all of the 22 dogs studied at this stage, cinefluorography showed gastroesophageal reflux, although none showed any gross or microscopic evidence of esophagitis.

Table 6

Comparison of Mean Intraluminal Pressures in Cms. of Water, Vagotomy vs Thoracic Displacement of Gastroesophageal Junction (24 dogs)

	VAGOTOMY	SIGNIFICANCE OF DIFFERENCE	THORACIC DISPLACEMENT
Pressures at Rest			
Stomach	7.56 ± 0.37		7.64 ± 0.37
Lower esophageal sphincter	21.41 ± 1.15	$P < .001$	16.99 ± 0.95
Esophagus	$0.04 \pm .19$		0.23 ± 0.25
Gradient between stomach and sphincter	13.95 ± 0.97	$P < .001$	9.43 ± 0.82
Pressures under Compression			
Stomach	22.68 ± 1.15	$P < .01$	19.45 ± 1.08
Lower esophageal sphincter	37.81 ± 1.55	$P < .001$	27.74 ± 1.23
Esophagus	1.33 ± 0.23	$P < .05$	2.01 ± 0.31
Gradient between stomach and sphincter	14.95 ± 1.45	$P < .001$	8.26 ± 0.99

Figure 4. A Bar Graph Representing Changes in Pressures (Cm. H_2O) in the Stomach, the Lower Esophageal Sphincter, the Esophagus and the Gradient between Vagotomy and Thoracic Displacement at Rest and During Compression.



STAGE IV: Studies after Restoration of Gastroesophageal Competence

Upon completion of the studies in Stage III, the dogs were randomly assigned to four groups, and each group of six dogs underwent a different procedure. Since there were only 23 dogs available at this stage one dog was used twice, first for sham operation (Group D) and then for Belsey repair (Group B). The results of different reparative procedures were evaluated by comparing the values at Stage III with those at Stage IV for the same six animals.

GROUP A. (Reduction of LES below the diaphragm and crural repair)

Six to eight weeks after Allison repair, cinefluorography and manometry were performed, and the following results were obtained:

Cinefluorography

All animals showed a normally-located GE junction, both at rest and during abdominal compression. Esophageal motor activity was not significantly changed. No reflux was demonstrated in four out of six dogs. Of the two dogs showing GE incompetence, one had shown reflux after vagotomy and was now back to that status, whereas the other dog was obviously worse than at the post-vagotomy stage.

	Number Studied	Spontaneous Reflux	Reflux on Compression	Total Showing Reflux
Before repair	6	4	6	6
After repair	6	2	2	2

Manometry

To demonstrate that the six animals randomly selected for this procedure were not significantly different from the rest of the animals in the experiment, their intraluminal pressures through Stages I to III

were compared using the paired t-test for statistical analysis. The results (Table 7) indicate that the changes in this group followed the same trend as the total animal population in the study. The confidence levels were lower, due to smaller numbers and wide variations between the animals in the group. Vagotomy decreased the LES pressure and the stomach-to-sphincter gradient, both at rest and during abdominal compression. Also, the response of the sphincter to compression was significantly reduced, as judged by the difference in the resting and compression gradients. Displacement of the LES above the diaphragm further reduced the resting sphincteric pressure and the stomach-to-sphincter gradient, but the change was not statistically significant. However during abdominal compression the decrease in the LES pressure and the stomach-to-sphincter gradient were statistically significant ($P < .05$). As expected, the length of the high pressure zone also decreased significantly ($P < .001$).

After thoracic displacement of the vagotomized LES, the mean resting pressures for this group of six dogs were 7.14 ± 0.74 cms. of water in the stomach, 15.62 ± 1.67 in the sphincter, and $0.43 \pm .4$ in the esophagus. The resting stomach-to-sphincter gradient was 8.47 ± 1.47 cms. of water. During abdominal compression the pressures were 18.77 ± 1.74 cms. of water in the stomach, 27.83 ± 1.84 in the sphincter, 1.5 ± 0.36 in the esophagus, with a stomach-to-sphincter gradient of 9.06 ± 2.16. After reduction of the LES below the diaphragm and crural repair (Allison, 44), the mean intraluminal pressures at rest were 8.0 ± 0.85 cms. of water in the stomach, 24.08 ± 2.06 in the sphincter, and 0.94 ± 0.34 in the esophagus, and the resting stomach-to-sphincter gradient was 16.0 ± 2.12. Under abdominal compression the pressures were

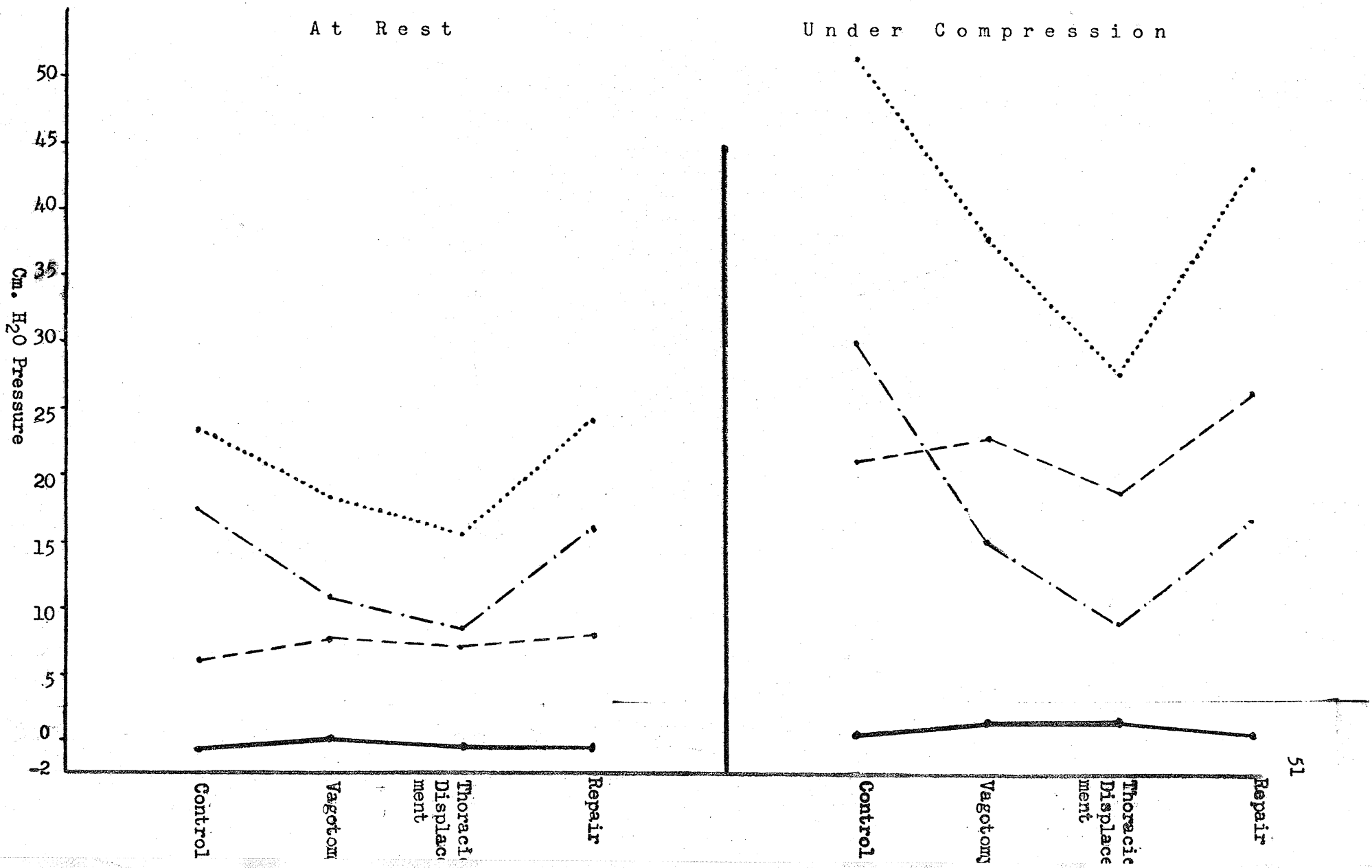
Table 7

Comparison of Mean Intraluminal Pressures in Cms. of Water and Length of the Lower Esophageal Sphincter in Cms., before and after Vagotomy, after Thoracic Displacement, and after Repair in Group A

	Control	Significance	Vagotomy	Significance	Thoracic Displacement	Significance	Repair
Resting Pressures							
Stomach	6.04 ± .42	P<0.1	7.8 ± .69		7.14 ± .74		8.0 ± .85
Sphincter	23.5 ± 2.9	P<.05	18.36 ± 2.19		15.62 ± 1.6	P<.001	24.08 ± 2.06
Esophagus	-0.6 ± .3		.18 ± 0.3		- .43 ± .4		- .9 ± .34
St-Sph Gradient	17.5 ± 3	P<.02	10.98 ± 1.57		8.47 ± 1.47	P<.001	16.0 ± 2.1
Compression Pressures							
Stomach	21.0 ± 1.13		22.87 ± 1.8		18.77 ± 1.7	P<.02	26.16 ± 3.1
Sphincter	51.38 ± 5.4	P<.05	37.9 ± 2.6	P<.01	27.8 ± 1.8	P<.01	43.08 ± 3.3
Esophagus	.5 ± .6		1.5 ± .37		1.5 ± .36		.6 ± .29
St-Sph Gradient	29.9 ± 5.18	P<.05	15.04 ± 1.9	P<.05	8.9 ± 2.16	P<.05	16.79 ± 2.8
Length of LES	2.7 ± .12		2.6 ± .09	P<.001	1.55 ± .14	P<.001	2.68 ± .06

Legend: – – – – Stomach
····· Sphincter
——— Esophagus
—·—·— St-Sph Gradient

Figure 5. A Frequency Distribution Representing Changes in Pressure (Cm H_2O) in the Stomach, the Lower Esophageal Sphincter, the Esophagus, and the Gradient in Control, Vagotomy, Thoracic Displacement and Repair, at Rest and Under Compression in Group A.



26.16 ± 3.15 cms. of water in the stomach, 43.08 ± 3.29 in the sphincter, and 0.62 ± 0.29 in the esophagus, and the stomach-to-sphincter gradient was 16.79 ± 2.8.

After repair significant changes were noted in the resting sphincteric pressure, which increased by 8.45 ± 0.85 cms. of water ($P < .001$), and the stomach-to-sphincter gradient which increased by 7.52 ± 1.07. On compression the significant changes were in the gastric pressure, which increased by 7.39 ± 2.08 ($P < .02$), the sphincter pressure, which increased by 15.25 ± 3.06 ($P < .01$), and the stomach-to-sphincter gradient, which increased 7.73 ± 2.47 ($P < .05$). There was no significant difference between the gradients with and without compression. Another significant change was seen in the length of the high pressure zone, which increased from 1.55 ± 0.14 cms. to 2.68 ± 0.65 ($P < .001$).

The net result of replacement of the LES below the diaphragm was an improvement in its length and an increase in its pressure resulting in an increase in the stomach-to-sphincter gradient. The mean pressures were now fairly close to the post-vagotomy level (Figure 5). Manometric improvement, in the sphincteric pressures and in the gradient, was accompanied by restoration of GE competence radiologically in four of the six animals. This indicates a close correlation between the LES pressure and stomach-to-sphincter gradient and the GE competence.

GROUP B. (Modified Belsey Repair)

The combined effects of restoration of the LES to its normal location and fundic wrap were studied six to eight weeks post-operatively in this group of six dogs. Manometric studies provided the measurements of intraluminal pressures while cinefluorography demonstrated the presence

or absence of gastroesophageal competence.

Cinefluorography

In all animals the GE junction was found below the diaphragm. Peristalsis in the esophagus was slow but did occur in response to each bolus of barium or air. None of the dogs showed reflux, either spontaneously or under compression. Dog 24, who demonstrated gross reflux and weight loss after sham procedure, developed gastroesophageal competence and gained weight following Belsey repair. In some animals there was delayed emptying of the lower esophagus, and two or three air swallows were required before the esophagus was cleared of all barium.

	Number Studied	Spontaneous Reflux	Reflux on Compression	Total Showing Reflux
Before repair	6	4	6	6
After repair	5	0	0	0

Manometry

The effects of vagotomy and thoracic displacement on manometry in Group B animals were also similar to those of the entire animal population in this study (Table 8). Vagotomy reduced the resting sphincteric pressure and the gradient but the change was not statistically significant. However, during compression there was significant decrease in the LES pressure ($P < .05$) and consequently in the stomach-to-sphincter gradient ($P < .05$). The sphincteric response to compression, measured as the difference between resting and compression gradients, was also reduced from 13.27 cms. of water to 4.55 cms. of water. Two dogs demonstrated gastroesophageal reflux at this stage (Table 12, Appendix).

Before repair the mean resting intraluminal pressures in this group were 7.65 ± 0.68 cms. of water in the stomach, 18.49 ± 2.4 in the LES, and 0.62 ± 0.6 in the esophagus, and the resting stomach-to-sphincter gradient was 10.83 ± 1.7. During compression the pressures in cms. of water were 18.86 ± 2.33 in the stomach, 29.07 ± 2.6 in the sphincter, and 2.1 ± 0.99 in the lower esophagus, and the stomach-to-sphincter gradient was 10.2 ± 2.1. After repair the mean intraluminal resting pressures in cms. of water were 8.04 ± 0.97 in the stomach, 27.04 ± 4.1 in the sphincter, and -0.66 ± 0.46 in the esophagus, and the gradient was 19.54 ± 3.68. During abdominal compression the mean pressures in cms. of water were 21.9 ± 1.69 in the stomach, 52.4 ± 2.39 in the sphincter, and 10.99 ± 0.6 in the esophagus, and the stomach-to-sphincter gradient was 30.03 ± 2.9 (Table 8).

Comparison of the values before and after repair shows a significant increase in the LES pressure both at rest ($P < .05$) and during compression ($P < .001$), and a corresponding increase in the stomach-to-sphincter gradient. There was also a significant improvement in the sphincter response to compression; the difference between the resting and compression increased from 4.65 cms. of water before repair to 10.49 cms. of water after the repair. The other significant change was an increase in the length of the high pressure zone from 2.04 ± 0.12 cms. before repair to 3.42 ± 0.15 ($P < .01$) after repair.

Improvement in the LES pressure and the stomach-to-sphincter gradient correlates well with restoration of gastroesophageal competence as demonstrated by cinefluorography. Correlation is better with intraluminal pressures on compression (Fig. 6) than with pressures at rest.

It appears that, as pressures reach the normal range, competence is regained. The other change which may contribute to restoration of GE competence is the increase in length of the high pressure zone to an above-normal value.

Table 8

Comparison of Mean Intraluminal Pressures in Cms. of Water and Length of the Lower Esophageal Sphincter in Cms., before and after Vagotomy, after Thoracic Displacement, and after Repair in Group B

	Control	Significance	Vagotomy	Significance	Thoracic Displacement	Significance	Repair
Resting Pressures							
Stomach	7.47 ± .77		7.6 ± .87		7.65 ± .68		8.04 ± .97
Sphincter	32.14 ± 4.9		24.28 ± 2.5	P<.05	18.49 ± 2.24	P<.05	27.04 ± 4.3
Esophagus	.41 ± .33		.13 ± .6		.62 ± .6		-.66 ± .46
St-Sph Gradient	24.8 ± 4.49		16.6 ± 1.9	P<.05	10.83 ± 1.77	P<.05	19.54 ± 3.68
Compression Pressures							
Stomach	20.8 ± 2.27		22.8 ± 2.8		18.86 ± 2.33		21.9 ± 1.69
Sphincter	59.08 ± 5.9	P<.05	42.38 ± 3.9	P<.02	29.07 ± 2.6	P<.001	52.4 ± 2.39
Esophagus	1.9 ± .44		1.4 ± .62		2.1 ± .99		.99 ± .6
St-Sph Gradient	38.35 ± 5.9	P<.05	21.25 ± 2.45	P<.05	10.2 ± 2.1	P<.01	30.03 ± 2.9
Length of LES	2.8 ± .17		2.8 ± .18	P<.05	2.04 ± .12	P<.01	3.42 ± .15

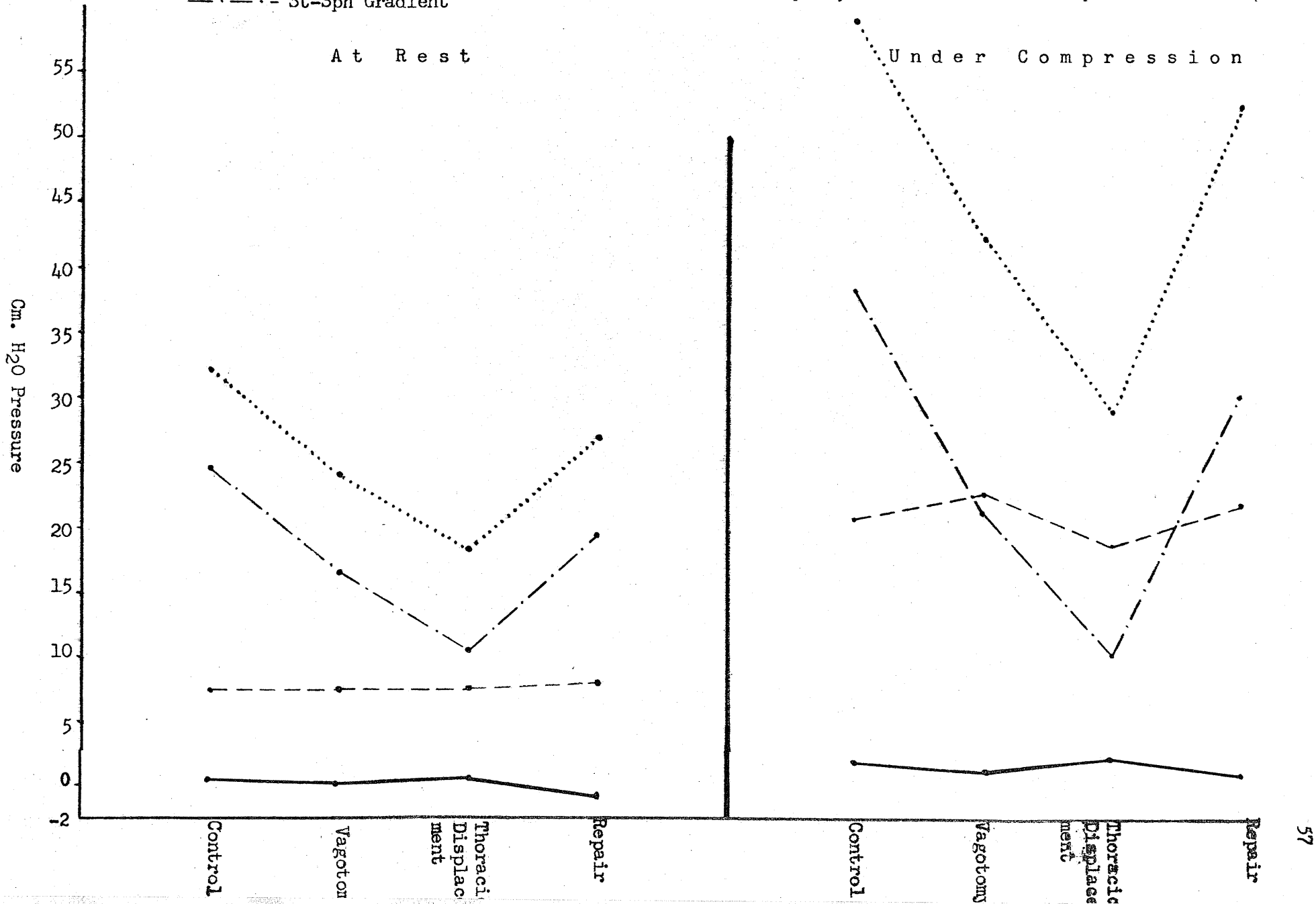


Figure 6. A Frequency Distribution Representing Changes in Pressure (Cm H_2O) in the Stomach, the Lower Esophageal Sphincter, the Esophagus, and the Gradient in Control, Vagotomy, Thoracic Displacement and Repair, at Rest and Under Compression in Group B.

GROUP C (Fundic-wrap on Intrathoracic LES)

In six dogs, all with gastroesophageal incompetence, a modified Belsey type of fundic-wrap was performed, retaining the GE junction 4 cms. above the diaphragm. Following this procedure there was no significant change either in GE competence or in intraluminal pressures.

Cinefluorography

All animals continued to show a portion of suprahiatal stomach, with the GE junction located about 4 cms. above the diaphragm. Motility in the body of the esophagus appeared normal. Five dogs, who showed GE reflux before repair, continued to show reflux. One dog, who was not studied at Stage III, also demonstrated GE reflux after repair.

	Number Studied	Spontaneous Reflux	Reflux on Compression	Total Showing Reflux
Before repair	5	4	5	5
After repair	6	5	5	6

Manometry

Table 9 shows the effect of vagotomy and thoracic displacement of the GE junction on the intraluminal pressures in the animals selected for Group C, and their statistical significance. Vagotomy reduced the resting pressure in the sphincter and the stomach-to-sphincter gradient ($P < .05$). During compression the LES pressure decreased from 49.82 cms. of water to 32.85 ($P < .01$), with a corresponding decrease in the stomach-to-sphincter gradient ($P < .01$). As expected, the response of the LES to compression was also significantly decreased. Thoracic displacement of the LES further reduced its intraluminal pressure and the stomach-to-sphincter gradient, but the changes did not reach statis-

tical significance. However, under abdominal compression both the sphincter pressure and the gradient were significantly reduced ($P < .05$). The length of the high pressure zone also diminished with intrathoracic displacement of the GE junction, from 2.62 ± 0.26 cms. to 1.99 ± 0.19 ($P < .05$). These changes are similar to those seen in the other groups, and indicate that the animals in this group were not entirely different from the rest in their physiological behaviour.

In this group before repair, the man intraluminal pressures in cms. of water under resting conditions were 7.43 ± 0.86 in the stomach, 17.22 ± 2.42 in the sphincter, and 0.07 ± 0.41 in the esophagus, and the stomach-to-sphincter gradients was 10.08 ± 1.85. During abdominal compression the pressures were 18.45 ± 2.1 in the stomach, 25.33 ± 3.11 in the sphincter, and 1.83 ± 0.26 in the esophagus, and the stomach-to-sphincter gradient was 7.17 ± 1.75. Four to six weeks after repair the mean resting pressures in cms. of water were 6.45 ± 1.14 in the stomach, 16.92 ± 2.8 in the LES, and 10.62 ± 0.68 in the esophagus, and the stomach-to-sphincter gradient was 10.47 ± 1.87. During abdominal compression the pressures were 17.13 ± 4.2 in the stomach. 27.87 ± 5.63 in the LES, and 2.54 ± 0.73 in the esophagus, and the stomach-to-sphincter gradient was 10.73 ± 2.37. When these values are compared with those before repair, no statistically significant difference is demonstrated (Table 9).

The length of the high pressure zone, which was 1.94 ± 0.12 cms. before repair, was not significantly changed after repair (Table 33, Appendix).

GROUP D (Sham Procedure)

The six animals in this group were studied before and after the

Table 9

Comparison of Mean Intraluminal Pressures in Cms. of Water and Length of the Lower Esophageal Sphincter in Cms., Before and After Vagotomy, after Thoracic Displacement, and after Repair in Group C

	Control	Significance	Vagotomy	Significance	Thoracic Displacement	Significance	Repair
Resting Pressures							
Stomach	6.61 ± .44		7.63 ± .93		7.43 ± .86		6.45 ± 1.14
Sphincter	24.79 ± 1.6		21.77 ± 1.97		17.22 ± 2.42		16.92 ± 2.82
Esophagus	-0.18 ± .62		0.02 ± .31		0.07 ± .41		0.62 ± .68
St-Sph Gradient	17.80 ± 1.55	$P<.05$	14.16 ± 1.78	$P<.1$	10.08 ± 1.85		10.47 ± 1.87
Compression Pressures							
Stomach	18.74 ± 1.43		20.65 ± 1.46		18.45 ± 2.1		17.13 ± 4.2
Sphincter	49.82 ± 3.66	$P<.01$	32.85 ± 1.81	$P<.1$	25.33 ± 3.11		27.87 ± 5.63
Esophagus	0.79 ± .52		1.11 ± .51		1.83 ± .26		2.54 ± .73
St-Sph Gradient	30.87 ± 3.0	$P<.01$	12.33 ± 1.78	$P<.05$	7.17 ± 1.75		10.73 ± 2.37
Length of LES	2.4 ± .3		2.62 ± .26	$P<.05$	1.99 ± .19		1.94 ± .1

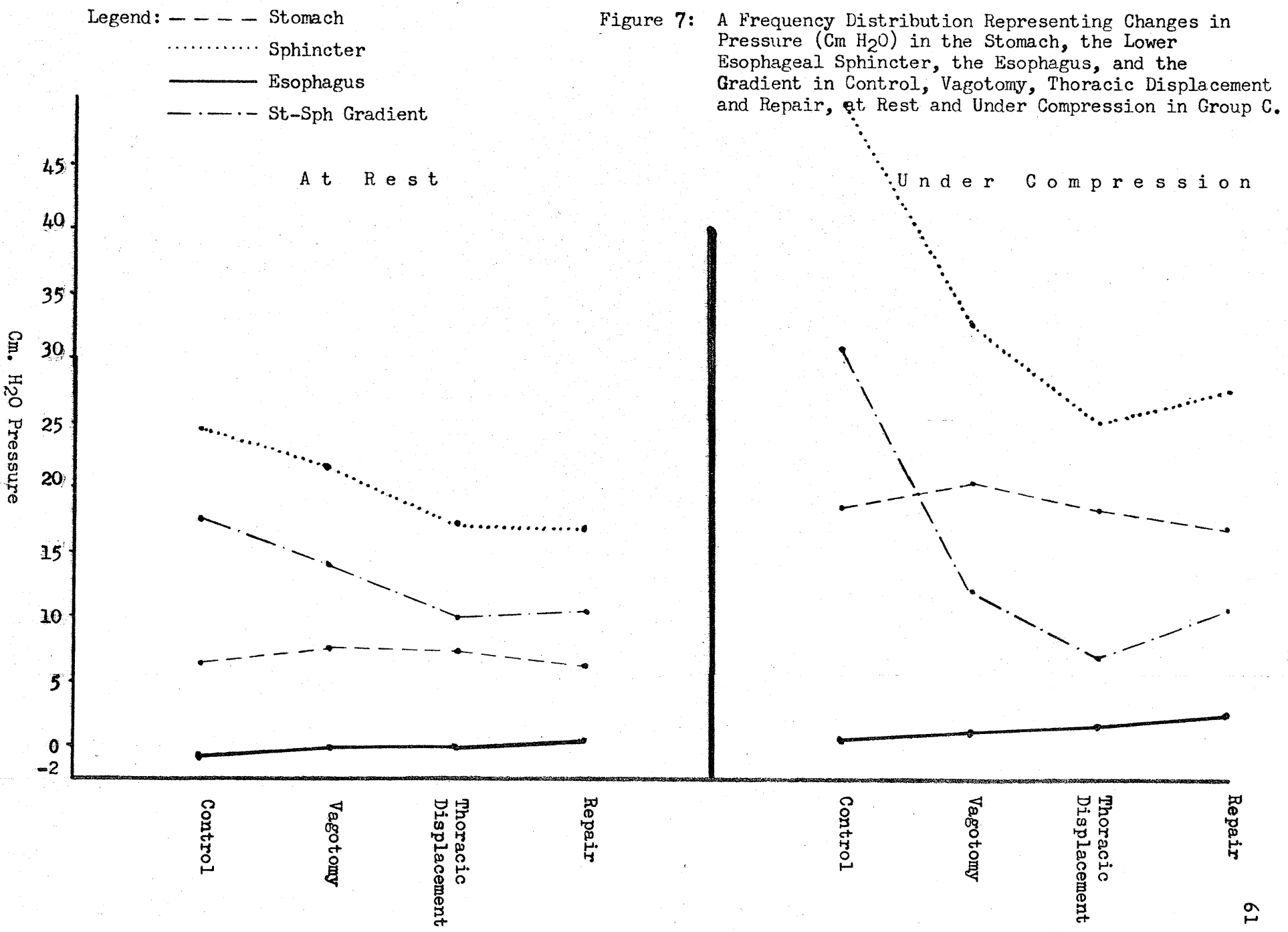


Figure 7: A Frequency Distribution Representing Changes in Pressure (Cm H_2O) in the Stomach, the Lower Esophageal Sphincter, the Esophagus, and the Gradient in Control, Vagotomy, Thoracic Displacement and Repair, at Rest and Under Compression in Group C.

procedure with cinefluorography and manometry and the results of the studies are as follows:

Cinefluorography

Before sham procedure all six animals in this group demonstrated a portion of the stomach above the diaphragm, and gastroesophageal reflux. After Sham procedure five dogs, who underwent cinefluorography, showed gastroesophageal reflux.

	Number Studied	Spontaneous Reflux	Reflux on Compression	Total Showing Reflux
Before operation	6	4	6	6
After operation	5	4	5	5

One dog died before cinefluorography could be performed. Manometric tests on this animal did not show any significant change after the sham procedure.

Manometry

The dogs in this group had also responded to vagotomy and thoracic displacement of the GE junction in the usual manner. Vagotomy reduced the mean resting pressure in the LES from 26.35 $\pm$ 2.6 cms. of water to 21.21 $\pm$ 2.41 ($P < .02$) and the resting stomach-to-sphincter gradient from 19.77 to 14.07 ($P < .01$). Under abdominal compression similar changes were noted. There was no rise in the stomach-to-sphincter gradient on compression, indicating an absence of LES response to abdominal compression. Thoracic displacement of the GE junction did not produce a significant change in the mean intraluminal pressures at rest, but during compression there was a fall in the LES pressure, from 38.08 $\pm$ 3.15 to 28.75 $\pm$ 2.49 cms. of water ($P < 0.05$). Also, the length of

the high pressure zone decreased from 2.38 ± .15 to 2.06 ± .11. All of the above changes are similar to those seen in other groups after vagotomy and thoracic displacement of LES.

Analysis of manometric tests performed after the sham procedure did not show a significant change in the LES function (Table 10). Before sham operation the mean resting pressures in cms. of water were 8.41 ± 0.88 in the stomach, 16.68 ± 1.53 in the LES, and 0.66 ± 0.51 in the esophagus, with a stomach-to-sphincter gradient of 8.25 ± 1.61. Under abdominal compression the pressures were 22.0 ± 2.78 cms. of water in the stomach, 28.75 ± 2.49 in the LES, and 2.56 ± 0.68 in the esophagus, with a gradient of 6.75 ± 2.11. After sham procedure the resting intraluminal pressures were 8.75 ± 0.89 cms. of water in the stomach, 18.33 ± 2.0 in the LES, and -0.27 ± 0.39 in the esophagus, giving a stomach-to-sphincter gradient of 9.58 ± 1.69. Under compression the pressures in cms. of water were 22.79 ± 2.46 in the stomach, 28.66 ± 3.66 in the sphincter, 1.89 ± 0.63 in the esophagus, and a gradient of 6.2 ± 1.87. The length of the high pressure zone was 2.06 ± 0.11 cms. before and 1.76 ± 0.11 after the operation.

A comparison between intraluminal pressures recorded before and after sham operation does not show any significant change, with the exception of esophageal pressure under compression which decreased after sham operation ($P < 0.05$).

Table 10

Comparison of Mean Intraluminal Pressures in Cms. of Water and Length of the Lower Esophageal Sphincter in Cms., before and after Vagotomy, after Thoracic Displacement and after Repair in Group D

	Control	Significance	Vagotomy	Significance	Thoracic Displacement	Significance	Repair
Resting Pressures							
Stomach	6.72 ± .66		7.14 ± .65		8.41 ± .88		8.75 ± .8
Sphincter	26.35 ± 2.6	P<.02	21.21 ± 2.41		16.68 ± 1.53		18.33 ± 2.0
Esophagus	-0.14 ± .33		-0.14 ± .27		0.66 ± .51		-0.27 ± .3
St-Sph Gradient	19.77 ± 2.64	P<.01	14.07 ± 2.18		8.25 ± 1.61		9.58 ± 1.6
Compression Pressures							
Stomach	23.1 ± 2.56		24.39 ± 3.0		22.0 ± 2.78		22.79 ± 2.4
Sphincter	57.69 ± 4.66	P<.05	38.08 ± 3.15	P<.05	28.75 ± 2.49		28.66 ± 3.6
Esophagus	0.66 ± .45		1.32 ± .45		2.56 ± .68	P<.05	1.89 ± .6
St-Sph Gradient.	34.57 ± 3.0	P<.02	13.27 ± 4.22		6.75 ± 2.11		6.2 ± 1.8
Length of LES	2.42 ± .13		2.38 ± .15	P<.05	2.06 ± .11		1.76 ± .1

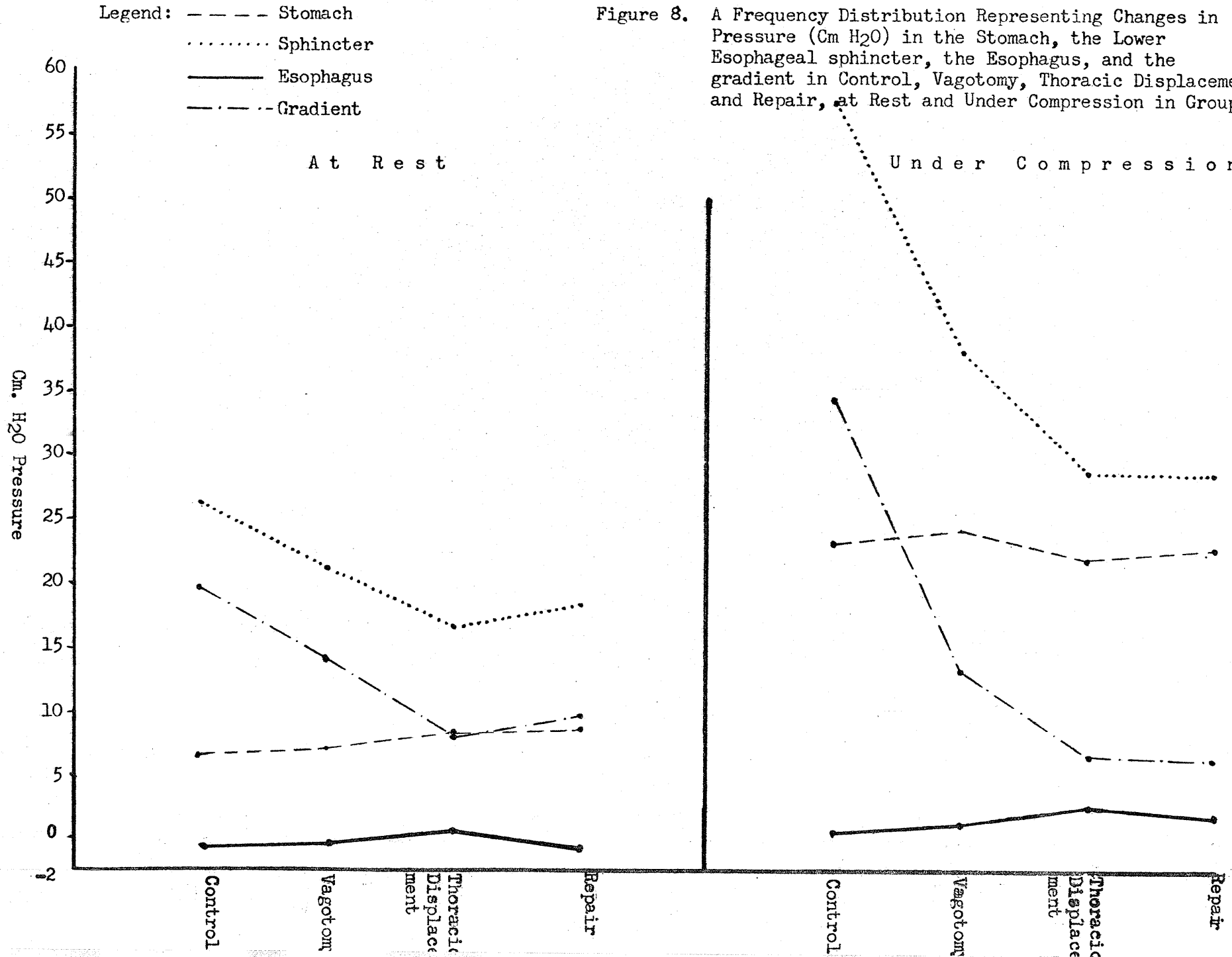


Figure 8. A Frequency Distribution Representing Changes in Pressure (Cm H_2O) in the Stomach, the Lower Esophageal sphincter, the Esophagus, and the gradient in Control, Vagotomy, Thoracic Displacement and Repair, at Rest and Under Compression in Group D.

DISCUSSION

THE MODEL

The animal model used in this study has several shortcomings. Although gastroesophageal incompetence was demonstrated in all animals with a vagotomized GE junction located in the chest, none of them developed esophagitis during the six to eight weeks of subsequent study. Other factors may contribute to the development of esophagitis besides gastroesophageal reflux. In human beings, the duration of gastroesophageal reflux seems an important factor, since occasional heartburn associated with gastroesophageal reflux is a common occurrence, but gross esophagitis is demonstrated only in those with longstanding reflux (95). Perhaps the dog would also develop esophagitis if the esophagus were exposed to acid-pepsin reflux for a period longer than six to eight weeks. There may also be a species difference in the sensitivity of the esophagus to acid-pepsin reflux. Plzak (96) applied artificial gastric juice, made from crystalline pepsin and dilute hydrochloric acid in a jet with constant stream size, to esophageal and duodenal mucosa of an anaesthetized dog. He found that esophageal mucosa ulcerated in 5 minutes and duodenal mucosa in 15-40 minutes. In his study the damage to mucosa was not only from acid-pepsin but also due to the mechanical trauma produced by the jet of acid. Obviously the situation bears no likeness to gastroesophageal reflux in the present model, and the validity of the observations is questionable. In fact the canine esophagus is fairly resistent to normal gastric juice. After resection of the gastroesophageal junction, Anderson (97) had to use histamine stimulation to produce esophagitis in his dogs. None of the unstimulated dogs developed esophagitis. Histamine stimulation in the absence of gastroesophageal reflux also failed to produce esophagitis, although

gastroduodenal inflammation and ulceration were common. These studies would indicate that, in dogs, prolonged exposure to gastric juice rich in acid-pepsin is required to produce esophagitis. In the present canine model, moreover, vagotomy probably protected the esophagus by reducing the acid-pepsin output of the stomach.

During vagotomy and thoracic displacement of the gastroesophageal junction a number of changes took place, all of which could have influenced gastroesophageal competence. To expose the vagi the phrenoesophageal ligament was divided circumferentially, which resulted in some widening of the esophageal hiatus and allowed greater mobility to the gastroesophageal junction. Bremner (49) studied lower esophageal sphincter pressures and gastroesophageal competence before and after complete division of the phrenoesophageal ligament, and concluded that the ligament did not play a significant role in maintaining LES pressure or gastroesophageal competence in the dog. He also concluded that the ligament provided structural support for maintenance of the normal anatomic position of the GE junction. When this support is lost the dog is prone to develop massive herniation through the hiatus, which accounted for the majority of the deaths in this study (Table 1). The above observations suggest that division of the phrenoesophageal ligament did not contribute significantly to the observed changes in the intraluminal pressure and GE competence.

During thoracic displacement the abdominal esophagus and upper part of the stomach were freed from all surrounding structures, including the esophageal hiatus, and the acute angle of His became an obtuse angle. Bremner (49) studied 5 dogs after excision of the diaphragmatic crura and found no evidence either of GE incompetence or significant

change in LES pressure. In a similar study on monkeys no change was found in LES pressure after excision of the left crus of the diaphragm (98). These studies indicate that separation of the gastroesophageal junction from the crura and surrounding structures was not responsible for the changes seen in GE competence and intraluminal pressures.

On the other hand, the shortening of the high pressure zone observed after thoracic displacement of the GE junction was due, at least in part, to separation of the GE junction from the crura. Bremner (49) showed that the length of the high pressure zone recorded at a normally located GE junction includes a crural component, since excision of the crura, with replacement of the GE junction in its normal position, shortens the high pressure zone. The present study is consistent with Bremner's observation: the length of the LES decreased from 2.60 ± 0.09 cms. to 1.87 ± 0.09 ($P < .001$) when the GE junction was displaced from the diaphragmatic crura (Table 11).

TABLE 11

Comparison of Mean Length of the Lower Esophageal Sphincter in cms. in 24 dogs, before Vagotomy, after Vagotomy, and after Thoracic Displacement of the LES.

Control	Significance of Difference	Vagotomy	Significance of Difference	Thoracic Displacement
2.64 ± 0.08		2.60 ± 0.09	$P < .001$	1.87 ± 0.09

It is unlikely that shortening of the high pressure zone leads to gastroesophageal reflux. Bremner (49) did not notice any change in gastroesophageal competence or sphincteric pressure when the GE junction

was located below the diaphragm. Presumably, the LES acts as a barrier to reflux by maintaining its pressure higher than that in the stomach, regardless of its length.

If neither isolation of the junctional zone from diaphragmatic influences, which destroys the angle of His, nor severance of the phrenicoesophageal ligament, which removes the anatomical stability of the LES, can alter sphincteric pressure or lead to GE reflux, how is gastroesophageal incompetence produced in this experimental model? It must be the result of vagotomy and thoracic displacement of the gastroesophageal junction. This study clearly demonstrates that subdiaphragmatic vagotomy decreased the resting tone of the LES and its response to abdominal compression (Fig. 3, Table 5), and supports the observations of Elbute (65) and of Lind (53). The changes in sphincteric function were associated with loss of gastroesophageal competence in nine out of twenty-three dogs (Table 12, Appendix). There are two possible explanations why the rest of the dogs did not demonstrate GE incompetence after vagotomy. Firstly, post-vagotomy cinefluorographic tests to demonstrate gastroesophageal reflux were carried out only once on each animal, over a period of ten to fifteen minutes, which may not have been sufficient to demonstrate reflux. Possibly, more animals would show reflux if the tests were repeated or prolonged. The second plausible explanation is that the decrease in sphincteric pressure and its response to compression was not sufficient to produce gastroesophageal incompetence in some animals.

The scatter-graph (Fig. 9, Appendix) shows a distinct correlation between the stomach-to-sphincter gradient under compression and gastroesophageal reflux. Although all animals at Stage III showed reflux,

with gradients lower than the normal range, there was considerable overlap at Stage II, after vagotomy: a number of animals had gradients close to the lower range of normal, but only a few showed evidence of GE incompetence on x-ray examination. The crucial level of gradient for incompetence seems to be around 10 cms. of water: if the LES cannot maintain a pressure 10 cms. higher than the gastric pressure under abdominal compression, it is likely to be incompetent on cinefluoroscopy.

The effect of thoracic displacement of the vagotomized GE junction was a further reduction in the sphincteric pressure and its response to compression, which was associated with gastroesophageal reflux in all animals tested. The fact that a normal GE sphincter does not become incompetent when displaced above the diaphragm, although its pressure is slightly reduced, was well shown by Lind (40). However, a vagotomized sphincter has a lower resting pressure which, combined with its inability to contract in response to compression, makes it less able to prevent reflux. As opposed to positive intra-abdominal pressure, an intrathoracic sphincter is exposed to negative (subatmospheric) pressure, which further reduces its intraluminal pressure. Thus a combination of vagotomy and thoracic displacement produces a model with GE incompetence.

RESTORATION OF GASTROESOPHAGEAL COMPETENCE

The results clearly show that fundic-wrap, combined with replacement of the GE junction below the diaphragm (56), was the most effective of the three procedures used to restore gastroesophageal competence. None of the five animals tested with cinefluorography showed any evidence of reflux. The sphincteric pressures improved in all six animals, particularily in response to compression (Table 8, Fig. 6).

The Allison procedure (44) came out second best, with restoration of competence in four of the six dogs, and improvement in mean sphincteric pressure and stomach-to-sphincter gradient which were not as marked as in the "modified Belsey" group.

	Belsey Repair	Allison Repair
Mean improvement in resting St-Sph gradient	8.7 ± 2.8	7.52 ± 1.07
Mean improvement in St-Sph gradient on compression	19.83 ± 3.53	7.73 ± 2.4
Mean gradient difference	11.13	0.21

How did the modified Belsey procedure restore competence? Firstly, the lower esophagus and the fundus of the stomach were dissected from surrounding structures. Secondly, the GE junction was replaced below the diaphragm and held there by approximation of the crura, establishing a relatively long intra-abdominal segment of esophagus. Lastly, the gastric fundus was wrapped around the GE junction and lower 4 cms. of the esophagus. Any one or more of these manoeuvres could have played a part in restoring competence.

The results from the sham group indicate that separation of the lower esophagus and the gastroesophageal junction from the neighbouring structures had no effect, either on gastroesophageal competence or on sphincteric pressures (Table 10, Fig. 8). Operative trauma to the junctional zone and its separation from surrounding structures can therefore be excluded as a factor contributing to competence after modified Belsey and Allison repairs.

It was clearly demonstrated in the first part of this study that the vagotomized GE sphincter works better in the abdomen than in

the chest. Therefore, any repair which restores a thoracic LES to an abdominal position should lead to an improvement in its function. This is well shown in the "Allison" group, where four of the six animals regained gastroesophageal competence, and mean intraluminal pressures improved in all animals. Likewise, reduction of the LES below the diaphragm would also be a significant factor in improving sphincteric function in the modified Belsey group.

It has already been shown that the diaphragmatic crura contribute to the length of the high pressure zone at the GE junction, and that removal of this influence shortens the high pressure zone by 0.5 to 1 cm. Bremner (49) has further shown that the crural contribution to the high pressure zone has no effect on GE sphincter function, and the findings of the present study support that observation. Approximation of the crura in the "Allison" group restored the length of the GE sphincter (Table 7). Crural repair then served only to retain the GE junction below the diaphragm both in the "Allison" and in the "modified Belsey" groups.

The only difference between the Allison and the modified Belsey repair was the fundic-wrap in the latter, which not only restored competence in all animals, but also improved sphincteric pressure and its response to compression. The response of the LES to an increase in intragastric pressure seems to represent a genuine increase in tone. It is an intrinsic property of the sphincter itself, apparently unaffected by location above or below the diaphragm, which is probably mediated by the vagi (40, 66, 99). How does the Belsey repair restore this response? There is no evidence to suggest that the vagi regenerated, thus establishing the nerve supply to the LES. Vagal regeneration should

have had a uniform effect, improving the LES response to a rise in intragastric pressure in all groups. This did not occur. Also, vagal regeneration would have taken much longer than 6-8 weeks, which was the time allowed between repairs and post-operative tests in this study. Hollander tests would have provided better evidence of vagal function, but were not carried out after the repairs.

Did the Belsey procedure produce a flap valve at the GE junction? This study indicates that it did not, since mechanical flap valve would be expected to function regardless of the position of the GE junction. In Group C, where fundic-wrap was performed around a thoracically-located GE junction, there was no improvement either in gastroesophageal competence or in sphincteric function. Also, if the fundic-wrap served to "squeeze" the junctional zone mechanically during abdominal compression, there should have been no relaxation of the LES in response to swallowing. Inspection of post-repair manometric records showed that the LES relaxed and contracted in response to swallows, much like a normal sphincter.

Was it downward displacement of the GE junction which restored competence? There is no doubt that fundic-wrap displaced the GE junction 3-4 cms. below the diaphragm. This was accompanied by an increase of 0.5 to 1 cm. in the length of the high pressure zone recorded at this region (Table 8). However, it has already been established that the length of the high pressure zone is not a significant factor in gastroesophageal competence. It would seem that downward displacement of the GE junction improves the intrinsic function of the LES, particularly its response to a rise in intragastric pressure, but it is hard to accept that a mere change in position could restore a neural reflex. Cohen (99) has suggested that the response to compression depends upon the base-line

sphincteric pressure, and that elevation of base-line pressure would improve this response. This obviously does not apply to the vagotomized LES, whose resting (base-line) pressure improved both after Allison repair and after modified Belsey repair, but whose response to compression improved only after the latter.

The presence of a ring of gastric muscle around the LES may in itself support the junctional zone (by supporting its contraction) during a rise in intra-abdominal pressure. It may be possible to show contractile activity of the fundic-wrap itself, and to determine its relationship with LES activity by implanting electrodes in the junctional zone and the fundic-wrap. This was not done in the present study for technical reasons and for fear of interfering with LES function.

In summary, the modified Belsey repair restores competence by improving the intrinsic function of the LES, both its resting pressure and its response to a rise in intragastric pressure. Intrinsic function improves to some extent by relocation of the LES below the diaphragm, and possibly also by further downward displacement. The presence of fundic muscle around the LES may reinforce its contraction through an unknown mechanism.

CONCLUSIONS

The most important mechanism responsible for maintenance of gastroesophageal competence is the intrinsic activity of the lower esophageal sphincter. When intragastric pressure rises, active contraction of the LES increases its intraluminal pressure and prevents gastroesophageal reflux. The fact that vagotomy decreases this response indicates its neural nature through a reflex arc mediated by the vagus.

When the vagotomized lower esophageal sphincter is displaced into the chest, its base-line pressure decreases further. This phenomenon, coupled with a poor response to compression, leads to gastroesophageal reflux. This canine model was used to study the mechanisms which restore competence after modified Belsey and Allison procedures.

The Belsey procedure was effective in restoring gastroesophageal competence in all animals. It improved the base-line LES pressure by bringing it down below the diaphragm. The improvement in its intrinsic response to compression, however, is not clearly understood. There is evidence to indicate that the Belsey repair did not produce a mechanical flap-valve, or lead to regeneration of vagi. The most likely mechanisms which improved the LES response to compression were firstly, displacement of the sphincter to a more inferior location in the abdomen, and secondly, the presence of fundic muscle around the GE junction to reinforce its contraction.

The phrenoesophageal ligament did not play a significant role in gastroesophageal competence, although division of the ligament allowed abnormal mobility to the GE junction, which at times led to herniation of the entire stomach into the chest with a fatal outcome.

The diaphragm contributed to the length of the high pressure zone recorded at the GE junction, but did not contribute to GE competence

Thoracic displacement of the GE junction decreased the length of the high pressure zone, while abdominal replacement restored it.

SUMMARY

Vagotomy at the GE junction and displacement of the junctional zone above the diaphragm produced a model of gastroesophageal incompetence. Vagotomy decreased base-line (resting) LES pressures, and significantly reduced its response to an increase in intragastric pressure. Thoracic displacement further lowered base-line pressures of the vagotomized LES, and gastroesophageal reflux was demonstrated in all the 23 animals studied. Despite demonstrable GE incompetence, none of the animals showed any evidence of esophagitis. The animals with an incompetent LES were treated with Allison and modified Belsey procedures to elucidate the mechanisms which restore competence.

It was demonstrated that the phrenoesophageal ligament, the diaphragmatic crura, and other surrounding structures played no part in restoring GE competence. The crura did add length to the high pressure zone recorded at the GE junction, but did not affect competence. Relocation of the LES below the diaphragm, as done in the Allison procedure, improved the base-line pressure in the LES but did not improve its response to abdominal compression. The improvement in this group was not enough to restore competence in all animals. This observation is compatible with results of the Allison repair in human beings, where it restored GE competence in only 50-60 per cent of the patients (94).

The modified Belsey repair proved to be the most effective procedure in restoring GE competence. It combined the advantages of a restoration of the LES to a subdiaphragmatic position, and a fundic-wrap, which placed the LES further down in the abdomen and probably also reinforced the LES contraction during abdominal compression. The fundic-wrap was ineffective when performed around an intrathoracic GE junction, and therefore probably did not act as a mechanical flap valve.

On the basis of this study, a modified Belsey procedure can be recommended as the method of choice over an Allison repair for restoring gastroesophageal competence in human beings.

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APPENDIX

Table 12

Results of Cinefluorographic Examinations, Controls, after Vagotomy, after Thoracic Displacement of GE Junction, and after Repair

Dog Number and Group	Control		After Vagotomy		After Thoracic Displacement		After Repair	
	Resting	Compression	Resting	Compression	Resting	Compression	Resting	Compression
GROUP A								
11	-	-	-	-	+	+	+	+
31	-	-	-	-	+	+	-	-
15	-	-	-	-	-	+	-	-
12	-	-	-	-	+	+	-	-
19	-	-	-	-	-	+	-	-
18	-	-	+	+	+	+	+	+
GROUP B								
17	-	-	-	-	+	+	-	-
7	-	-	-	-	-	+	-	-
16	-	-	-	-	+	+	-	-
3	-	-	-	+	-	+	not done	
25	-	-	+	+	+	+	-	-
24	-	-	-	-	+	+	-	-
GROUP C								
28	-	-	+	+	+	+	+	+
4	-	-	-	+	+	+	+	-
23	-	-	+	+	+	+	+	+
1	-	-	-	-	+	+	+	+
29	-	-	-	-	not done		-	+
8	-	-	-	-	-	+	+	+
GROUP D								
24	-	-	-	-	+	+	+	+
6	-	-	+	+	-	+	+	+
14	-	-	-	+	-	+	+	+
10	-	-	-	-	+	+	-	+
13	-	-	+	+	+	+	not done	
9	-	-	-	-	+	+	+	+

- no reflux

+ reflux

Table 13

Intraluminal Pressures in Cms. of Water, Group A, Controls

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
31	4.0	10.0	-2.0	6.0	15.0	28.0	-2.0	13.0	7.0
	8.0	18.0	+1.0	10.0	18.0	45.0	-1.0	27.0	17.0
	8.0	22.0	-3.0	14.0	30.0	58.0	0.0	28.0	14.0
	9.0	22.0	0.0	13.0	22.0	34.0	0.0	12.0	-1.0
19	2.0	16.0	-3.0	14.0	13.0	28.0	-2.0	15.0	1.0
	6.0	32.0	+1.0	26.0	17.0	41.0	+3.0	24.0	-2.0
	6.0	27.0	-2.0	21.0	18.0	42.0	+2.0	24.0	3.0
	6.0	20.0	+1.0	14.0	18.0	36.0	+4.0	18.0	4.0
18	6.0	16.0	+0.5	10.0	20.0	36.0	+0.5	16.0	6.0
	8.0	20.0	0.0	12.0	20.0	56.0	+3.0	36.0	24.0
	8.0	13.0	+2.0	5.0	28.0	46.0	+5.0	18.0	13.0
	6.0	22.0	+0.0	16.0	20.0	60.0	0.0	40.0	24.0
15	10.0	20.0	+3.0	10.0	28.0	78.0	+1.0	50.0	40.0
	3.0	18.0	-2.0	15.0	27.0	38.0	0.0	11.0	-4.0
	4.0	11.0	-2.0	7.0	18.0	52.0	+1.0	34.0	27.0
	3.0	21.0	-2.0	18.0	25.0	42.0	-1.5	17.0	-1.0
12	4.0	37.0	-2.0	33.0	21.0	77.7	-2.0	56.7	23.7
	6.0	28.0	-2.0	22.0	23.5	86.5	-1.5	63.0	41.0
	7.5	32.0	-1.0	24.5	26.0	68.0	-2.0	42.0	17.5
	4.0	34.7	-1.5	30.7	19.5	70.3	-1.5	50.8	20.1
11	6.5	38.0	0.0	31.5	16.5	52.5	+2.0	36.0	4.5
	10.0	22.0	+1.0	12.0	23.5	50.0	+1.5	26.5	14.5
	4.0	25.8	-0.5	21.8	16.5	44.0	+3.0	27.5	5.7
	6.0	40.1	-2.0	34.1	20.5	64.8	+1.5	44.3	10.2

Table 14

Intraluminal Pressures in Cm. of Water, Group A, after Vagotomy

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
31	6.0	13.0	+2.0	7.0	18.0	40.0	+3.0	22.0	15.0
	6.0	10.0	-2.0	4.0	17.0	24.0	+1.0	7.0	3.0
	6.0	10.0	-2.0	4.0	20.0	31.0	+2.0	11.0	7.0
	10.0	19.0	-2.0	9.0	23.0	44.0	+1.0	21.0	12.0
19	4.0	20.0	0.0	16.0	24.0	46.0	0.0	22.0	6.0
	5.0	12.0	0.0	7.0	16.0	24.0	+2.0	8.0	1.0
	5.0	17.0	0.0	12.0	27.0	34.0	+2.0	7.0	-5.0
	6.0	14.0	0.0	8.0	26.0	26.0	+2.0	0.0	-8.0
18	9.0	26.0	+2.0	17.0	21.0	43.0	+3.5	22.0	5.0
	8.0	20.0	+2.0	12.0	18.0	32.0	+3.0	14.0	2.0
	8.0	14.0	0.0	6.0	20.0	22.0	+3.0	2.0	-4.0
	5.0	10.0	+1.0	5.0	16.0	24.0	+2.0	8.0	3.0
15	9.0	20.0	+1.5	11.0	38.0	46.0	+3.0	8.0	-3.0
	9.0	20.0	-0.5	11.0	28.0	40.0	-1.0	12.0	1.0
	6.0	14.0	+1.0	8.0	28.0	58.0	+2.0	30.0	22.0
	10.0	14.0	-0.5	4.0	30.0	40.0	-1.0	10.0	6.0
12	10.0	18.0	+0.5	8.0	24.0	40.0	+1.0	16.0	8.0
	10.5	22.0	+0.5	11.5	23.0	36.0	+1.0	13.0	1.5
	8.2	30.3	-1.5	22.1	26.5	52.0	-1.0	25.5	3.4
	8.0	21.5	0.0	13.5	23.5	33.0	0.0	9.5	-4.0
11	14.0	19.0	+0.5	5.0	24.0	41.0	+3.0	17.0	12.0
	7.5	17.0	0.0	9.5	18.0	48.0	1.25	30.0	20.5
	8.0	32.0	+1.0	24.0	19.0	50.0	+1.0	31.0	7.0
	9.0	38.0	+1.0	29.0	21.0	36.0	+2.0	15.0	-14.0

Table 15

Intraluminal Pressures in Cm. of Water, Group A,
after Thoracic Displacement of LES

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
31	8.0	22.0	-1.0	14.0	23.0	28.0	+1.0	5.0	-9.0
	10.0	14.0	-4.0	4.0	18.0	18.0	-2.0	0.0	-4.0
	13.0	16.0	0.0	3.0	30.0	42.0	+3.0	12.0	9.0
	8.0	11.0	-2.0	3.0	20.0	22.0	+2.0	2.0	-1.0
19	5.0	14.0	-2.0	9.0	16.0	32.0	0.0	16.0	7.0
	3.0	12.0	-2.0	9.0	10.0	21.0	+2.0	11.0	2.0
	6.0	9.0	-2.0	3.0	11.0	18.0	+1.5	7.0	4.0
	5.0	14.0	-1.0	9.0	9.0	20.0	+1.5	11.0	2.0
18	5.0	16.0	0.0	11.0	21.0	23.0	-1.5	2.0	-9.0
	9.0	15.0	+3.0	6.0	17.0	20.0	+3.0	3.0	-3.0
	10.0	12.0	0.0	2.0	19.0	28.0	0.0	6.0	+4.0
	9.0	16.0	-2.0	7.0	16.0	22.5	0.0	6.0	-1.0
15	6.0	12.0	+1.0	6.0	26.0	26.0	+1.0	0.0	-6.0
	6.0	12.0	-1.0	6.0	16.0	18.0	+3.0	2.0	-4.2
	5.0	12.0	-1.0	7.0	28.0	40.0	+4.0	12.0	+5.0
	5.5	12.0	-0.5	6.5	23.5	28.0	+2.5	4.5	-2.0
12	8.0	18.0	+1.0	10.0	24.0	40.0	+2.0	16.0	6.0
	8.0	22.0	+0.5	14.0	19.0	39.0	+1.0	20.0	6.0
	7.0	21.0	-1.0	14.0	13.0	30.0	0.0	17.0	3.0
	8.0	32.0	0.0	24.0	20.0	32.0	+2.0	12.0	12.0
11	6.0	9.0	+1.0	3.0	18.0	30.0	2.0	12.0	9.0
	7.0	18.0	+2.5	11.0	17.0	31.0	3.0	14.0	3.0
	7.0	16.0	0.0	9.0	18.0	30.0	3.0	12.0	3.0
	7.0	20.0	0.0	13.0	18.0	28.0	2.0	15.0	2.0

Table 16

Intraluminal Pressures in Cm. of Water, Group A, after Allison Repair

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
31	9.0	26.0	-2.0	17.0	24.0	30.0	-2.0	6.0	-11.0
	12.0	26.0	+2.0	14.0	32.0	42.0	+5.0	10.0	-4.0
	14.0	28.0	+2.0	14.0	34.0	56.0	+2.0	22.0	8.0
	10.0	18.0	-1.0	8.0	33.0	50.0	0.0	17.0	9.0
19	5.0	14.0	-1.5	9.0	19.0	35.0	-2.0	14.0	5.0
	6.0	18.0	-1.5	8.0	23.0	42.0	+2.0	19.0	11.0
	5.0	22.0	-2.0	17.0	18.0	28.0	+1.0	10.0	-7.0
	4.0	20.0	-2.0	16.0	17.0	30.0	-2.0	13.0	-3.0
18	8.0	26.0	-2.0	18.0	25.0	31.0	0.0	6.0	-12.0
	8.0	22.0	-1.0	14.0	27.0	56.0	+2.0	29.0	15.0
	5.0	18.0	-3.0	13.0	25.0	40.0	+1.0	15.0	2.0
	6.0	26.0	-1.5	20.0	33.0	53.0	+3.0	20.0	0.0
15	8.0	25.0	0.0	17.0	39.0	57.0	+3.0	18.0	1.0
	11.0	16.0	0.0	5.0	39.0	45.0	+4.0	6.0	1.0
	7.0	28.0	-3.0	21.0	39.0	47.0	0.0	8.0	-13.0
	8.0	24.0	+2.0	16.0	39.0	60.0	+3.0	21.0	5.0
12	4.0	52.0	-2.0	48.0	14.0	40.0	-1.0	26.0	-22.0
	7.0	24.0	-2.0	17.0	21.0	48.0	+1.0	27.0	10.0
	10.0	29.0	+1.0	19.0	22.0	48.0	0.0	26.0	7.0
	8.0	29.0	+2.0	21.0	21.0	64.0	+1.0	43.0	22.0
11	8.0	21.0	0.0	13.0	17.0	26.0	-1.0	9.0	-4.0
	9.0	14.0	-1.0	5.0	24.0	36.0	-1.0	12.0	7.0
	9.0	36.0	0.0	27.0	22.0	38.0	+1.0	16.0	-11.0
	9.0	16.0	-2.0	7.0	22.0	32.0	+1.0	10.0	3.0

Table 17

Intraluminal Pressures in Cm. of Water, Group B, Controls

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
25	7.0	17.0	-1.0	10.0	15.0	50.0	-1.0	35.0	25.0
	6.0	18.0	-1.0	12.0	18.0	34.0	+3.0	16.0	4.0
	6.0	29.0	+1.0	23.0	15.0	52.0	+2.0	37.0	14.0
	5.0	33.0	-1.5	28.0	16.0	50.0	+2.0	34.0	6.0
3	6.0	34.0	+2.0	28.0	20.0	78.0	+3.0	58.0	30.0
	9.0	26.0	+0.5	17.0	20.0	76.0	+2.0	56.0	39.0
	10.0	47.0	+0.5	37.0	24.0	80.0	+2.0	56.0	19.0
	8.0	38.0	0.0	30.0	18.0	72.0	+2.0	54.0	24.0
7	5.7	23.5	-1.0	17.8	28.0	48.0	+3.0	22.0	4.8
	12.0	24.0	-2.0	12.0	23.0	42.0	+2.0	19.0	7.0
	12.3	35.0	+3.0	22.7	47.0	74.0	+2.0	27.0	4.3
	5.0	12.0	0.0	7.0	26.0	50.0	+4.0	24.0	17.0
17	7.0	24.0	+1.0	17.0	18.0	38.0	+1.0	20.0	3.0
	3.0	24.0	-1.0	21.0	15.0	51.0	+1.0	36.0	15.0
	5.0	26.0	+1.0	21.0	15.0	42.0	0.0	27.0	6.0
	3.0	22.0	-2.0	19.0	14.0	55.0	-2.0	41.0	22.0
24	9.0	32.0	+1.0	23.0	16.0	54.0	+2.0	38.0	15.0
	8.0	30.0	+3.0	22.0	22.0	56.0	+6.0	34.0	12.0
	7.0	27.0	+1.0	20.0	23.0	52.0	+2.0	29.0	9.0
	7.0	30.0	0.0	27.0	22.0	50.0	0.0	28.0	1.0
16	9.0	56.0	+2.5	47.0	22.0	101.0	+3.0	79.0	32.0
	12.0	92.0	+2.5	80.0	23.0	105.0	+3.0	82.0	2.0
	8.5	32.0	0.0	23.5	18.5	50.0	+2.5	31.5	8.0
	9.0	40.0	+0.5	31.0	21.5	58.0	+3.0	37.0	6.0

Table 18

Intraluminal Pressures in Cm. of Water, Group B, after Vagotomy

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
25	10.0	36.0	+2.0	26.0	19.0	24.0	+3.0	5.0	-21.0
	10.0	22.0	-2.0	12.0	24.0	36.0	0.0	12.0	0.0
	9.0	22.0	-2.0	13.0	14.0	28.0	0.0	14.0	1.0
	7.0	22.0	0.0	15.0	17.0	35.0	-1.0	18.0	3.0
3	10.0	26.0	0.0	16.0	25.0	37.0	0.0	12.0	-4.0
	8.0	22.0	+2.0	14.0	36.0	66.0	+2.5	30.0	16.0
	12.0	16.0	+1.0	4.0	31.0	44.0	+2.0	13.0	9.0
	8.0	24.0	-3.0	16.0	28.0	37.0	-1.0	9.0	-7.0
7	14.5	45.0	-2.0	30.5	32.0	58.0	+1.0	26.0	-4.5
	10.0	42.0	+1.5	32.0	24.5	68.6	+2.0	44.1	12.1
	8.0	22.0	-1.0	14.0	27.0	65.6	+1.2	38.6	24.6
	8.5	30.0	-1.0	21.5	32.0	49.0	+1.5	17.0	-4.5
17	5.0	21.0	-2.0	16.0	19.0	34.0	-3.0	15.0	-1.0
	3.0	16.0	-1.0	13.0	18.0	33.0	-1.0	15.0	2.0
	4.0	19.0	-2.0	15.0	16.0	35.0	-2.0	19.0	4.0
	6.0	25.0	-1.0	19.0	18.0	36.0	+1.0	18.0	-1.0
24	8.0	22.0	0.0	14.0	27.0	34.0	+2.5	7.0	-7.0
	6.0	19.0	-2.0	13.0	31.0	52.0	+1.0	21.0	8.0
	7.0	17.0	-1.0	10.0	34.0	39.0	+5.0	5.0	-5.0
	7.0	19.5	-1.0	12.5	30.5	41.0	+1.3	11.1	-1.4
16	8.0	30.0	+4.0	22.0	14.0	32.0	+4.0	18.0	-4.0
	8.0	26.0	+2.0	18.0	14.0	34.0	+3.0	20.0	2.0
	10.0	34.0	+2.0	24.0	12.0	40.0	+3.0	28.0	4.0
	8.0	25.0	+3.0	17.0	13.0	40.0	+3.0	27.0	10.0

Table 19

Intraluminal Pressures in Cm. of Water, Group B, after Thoracic Displacement of LES

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
25	10.0	36.0	+2.0	26.0	19.0	24.0	+3.0	5.0	21.0
	10.0	22.0	-2.0	12.0	24.0	36.0	0.0	12.0	0.0
	9.0	22.0	-2.0	13.0	14.0	28.0	0.0	14.0	1.0
	7.0	22.0	0.0	15.0	17.0	35.0	-1.0	18.0	3.0
3	7.0	16.0	+2.0	9.0	20.0	36.0	+3.0	16.0	7.0
	8.0	16.0	-1.0	8.0	30.0	46.0	+1.0	16.0	8.0
	6.0	16.0	+1.0	10.0	26.0	28.0	+2.0	2.0	-8.0
	6.0	16.0	-1.0	10.0	25.0	31.0	+1.5	6.0	-4.0
7	9.0	28.0	+1.0	19.0	28.0	34.0	+3.0	6.0	-13.0
	6.0	9.0	0.0	3.0	23.0	33.0	+0.5	10.0	7.0
	9.0	22.0	+2.0	13.0	23.0	40.0	+3.0	17.0	4.0
	9.0	31.0	+2.0	22.0	31.0	32.0	+1.0	1.0	-21.0
17	5.0	10.0	-2.0	5.0	15.0	25.0	-2.0	10.0	5.0
	6.0	20.0	-2.0	14.0	14.0	34.0	-2.0	20.0	6.0
	3.0	10.0	0.0	7.0	13.0	16.0	+1.5	3.0	-4.0
	5.0	9.0	-1.5	4.0	11.0	23.0	-2.0	12.0	8.0
24	7.0	15.0	-1.0	8.0	16.0	20.0	+6.0	4.0	-4.0
	12.0	19.0	+2.0	7.0	19.0	22.0	+4.0	3.0	-4.0
	6.0	10.0	+2.0	4.0	16.0	16.0	+6.0	0.0	-4.0
	7.0	8.0	0.0	1.0	16.0	16.0	+6.0	0.0	-1.0
16	8.0	20.0	+3.0	12.0	13.0	30.0	+4.0	17.0	5.0
	7.0	12.0	+4.0	5.0	12.0	36.0	+4.0	24.0	19.0
	12.0	28.0	+4.0	16.0	14.0	31.0	+6.0	17.0	1.0
	10.0	27.0	+2.5	17.0	14.0	26.0	+3.5	12.0	-5.0

Table 20

Intraluminal Pressures in Cm. of Water, Group B,
after Fundic-Wrap (Modified Belsey Repair)

Dog Number	Resting				Compression				
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	St-Sph Gradient Difference
25	8.0	30.0	+2.0	22.0	17.0	60.0	+2.0	43.0	21.0
	10.0	54.0	0.0	44.0	20.0	60.0	0.0	40.0	-4.0
	15.0	46.0	-2.0	31.0	24.0	67.0	-1.0	43.0	12.0
	4.0	54.0	+1.0	50.0	19.0	58.0	-1.0	39.0	-11.0
3	6.0	23.0	-2.0	17.0	26.0	70.0	+2.0	44.0	27.0
	6.0	25.0	-2.0	19.0	28.0	56.0	+3.0	28.0	9.0
	6.0	22.0	0.0	16.0	20.0	40.0	+3.0	20.0	4.0
	8.0	16.0	+1.0	8.0	28.0	68.0	+4.0	40.0	32.0
7	10.0	19.0	+1.5	9.0	28.0	44.0	0.0	16.0	7.0
	9.0	20.0	-1.0	11.0	28.0	36.0	+1.0	8.0	-3.0
	9.0	32.0	-1.0	23.0	25.0	60.0	0.0	35.0	12.0
	9.0	26.0	+1.5	17.0	26.0	50.0	+1.0	24.0	7.0
17	5.0	12.0	-1.0	7.0	15.0	42.0	-1.0	27.0	20.0
	4.0	12.0	-1.0	8.0	18.0	50.0	+1.0	32.0	24.0
	4.0	17.0	-2.0	13.0	19.0	60.0	-2.5	41.0	28.0
	4.0	18.0	-2.0	14.0	15.0	44.0	-2.5	29.0	15.0
24	6.0	30.0	-4.0	24.0	18.0	44.0	-2.0	26.0	2.0
	7.0	20.0	-2.0	13.0	22.0	50.0	+2.0	28.0	15.0
	11.0	23.0	-2.0	12.0	24.0	63.0	+2.0	39.0	27.0
	8.0	26.0	-2.0	18.0	22.0	40.0	+4.0	18.0	0.0
16	11.0	42.0	0.0	31.0	25.0	56.0	+3.0	31.0	0.0
	10.0	32.0	0.0	22.0	22.0	52.0	+3.0	30.0	8.0
	12.0	30.0	0.0	18.0	26.0	50.0	+1.0	24.0	6.0
	11.0	20.0	+1.0	9.0	22.0	38.0	+2.0	16.0	7.0

Table 21

Intraluminal Pressures in Cm. of Water, Group C, Controls

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
4	8.0	32.0	-1.0	24.0	26.0	58.0	0.0	32.0	8.0
	4.0	17.0	0.0	13.0	19.0	64.0	+1.0	45.0	32.0
	5.0	27.0	0.0	22.0	19.0	62.0	+1.0	43.0	21.0
	8.0	35.0	0.0	27.0	24.0	60.0	0.0	36.0	9.0
8	4.0	19.0	0.0	15.0	11.5	52.0	-1.0	40.5	25.5
	8.0	35.0	+2.0	27.0	18.0	46.0	+2.0	28.0	1.0
	8.0	26.0	+4.5	18.0	18.0	39.0	+4.5	21.0	3.0
	6.7	26.7	+3.0	20.0	15.8	46.0	+2.5	30.5	10.5
29	8.0	21.0	-2.0	13.0	24.0	38.0	+2.0	14.0	1.0
	6.0	19.0	-3.0	7.0	21.0	49.0	-2.0	28.0	21.0
	5.0	22.0	-2.0	17.0	26.0	52.0	-2.0	26.0	9.0
	10.0	22.0	-2.0	12.0	17.0	37.0	0.0	20.0	8.0
23	4.0	34.0	-3.0	30.0	13.0	45.0	-2.0	32.0	2.0
	4.0	16.0	-1.0	12.0	16.0	66.0	0.0	50.0	38.0
	8.0	19.0	+1.5	11.0	18.0	52.0	0.0	34.0	23.0
	4.0	16.0	-1.0	12.0	11.0	27.0	-1.5	16.0	4.0
28	5.0	20.0	-1.0	15.0	17.0	36.0	+2.0	19.0	4.0
	5.0	21.0	0.0	16.0	15.0	38.0	+3.0	23.0	7.0
	7.0	23.0	-2.0	16.0	14.0	39.0	+1.0	25.0	9.0
	8.0	22.0	+1.0	14.0	20.0	46.0	+2.0	26.0	12.0
1	9.0	44.0	+1.0	35.0	23.0	75.0	+1.5	52.0	17.0
	9.5	18.0	0.0	8.5	19.5	55.0	0.0	35.5	27.0
	6.5	14.0	0.0	7.5	20.3	48.0	+2.0	28.0	20.5
	8.0	43.3	+0.5	35.3	24.0	65.1	+3.0	41.1	5.8

Table 22

Intraluminal Pressures in Cm. of Water, Group C, after Vagotomy

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
4	5.0	26.0	+1.5	21.0	23.0	30.0	+1.5	7.0	-14.0
	2.0	10.0	-2.0	8.0	14.0	28.0	+2.0	14.0	6.0
	5.0	26.0	+0.5	21.0	16.0	30.0	+2.5	14.0	-7.0
	5.0	34.0	-1.0	29.0	16.0	38.0	+4.0	22.0	-7.0
8	8.0	24.5	+2.0	16.5	23.5	38.0	+2.0	14.5	-2.0
	9.3	29.7	+1.5	20.4	22.7	29.0	+1.0	6.3	-14.1
	14.5	35.0	+2.0	20.5	31.0	39.5	+2.0	8.5	-12.0
	11.2	31.0	+0.7	19.8	26.0	27.2	+1.7	1.2	-18.6
29	6.0	22.0	-2.0	16.0	24.0	26.0	-2.0	2.0	-14.0
	8.0	15.0	+1.0	7.0	22.0	34.0	-2.0	12.0	5.0
	8.0	16.0	-1.0	8.0	21.0	30.0	-1.0	9.0	1.0
	7.0	13.0	+1.0	6.0	18.0	23.0	+1.0	5.0	-1.0
23	6.0	13.0	0.0	7.0	18.0	21.0	+2.0	3.0	-4.0
	6.0	12.0	0.0	6.0	15.0	18.0	-1.0	7.0	1.0
	6.0	12.0	-2.0	6.0	19.0	40.0	+2.0	21.0	15.0
	7.0	36.0	0.0	29.0	20.0	36.0	+1.0	16.0	-13.0
28	8.0	17.0	+1.0	9.0	16.0	40.0	+3.0	24.0	15.0
	7.0	26.0	0.0	19.0	20.0	26.0	+2.0	6.0	-13.0
	12.0	18.0	-1.0	6.0	20.0	42.0	+2.0	22.0	16.0
	6.0	26.0	0.0	20.0	15.0	32.0	+1.0	17.0	-3.0
1	11.0	24.0	-1.7	13.5	22.5	31.5	+2.0	9.0	-4.5
	6.0	18.5	-1.0	12.5	17.0	37.0	0.0	20.0	7.5
	10.0	19.3	0.0	9.3	28.0	44.0	0.0	16.0	6.7
	8.5	18.0	+1.0	9.5	28.0	48.3	0.0	20.3	-7.7

Table 23

Intraluminal Pressures in Cm. of Water, Group C,
after Thoracic Displacement of LES

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
4	8.0	30.0	0.0	22.0	24.0	42.0	+2.0	18.0	-4.0
	8.0	34.0	+3.0	26.0	21.0	32.0	+3.0	11.0	-15.0
	14.0	20.0	0.0	6.0	28.0	36.0	0.0	8.0	2.0
8	10.0	19.0	+3.0	9.0	17.0	22.0	+3.0	5.0	-4.0
	8.0	10.0	+2.0	2.0	12.0	18.0	+3.0	6.0	4.0
	6.0	14.0	+1.5	8.0	12.0	16.0	+1.0	4.0	-4.0
	4.0	12.0	-0.5	8.0	13.0	18.0	-0.5	5.0	-3.0
29	11.0	17.0	0.0	6.0	24.0	26.0	+4.0	2.0	-4.0
	8.0	14.0	-2.0	6.0	24.0	26.0	+2.0	2.0	-4.0
	5.0	8.0	-3.0	3.0	20.0	24.0	+1.0	4.0	1.0
	6.0	14.0	-1.0	8.0	19.0	22.0	+1.0	3.0	-5.0
23	6.0	18.0	+1.0	12.0	15.0	22.0	+2.0	7.0	-5.0
	6.0	18.0	0.0	12.0	17.0	34.0	+3.0	17.0	5.0
	8.0	22.0	-1.0	14.0	20.0	38.0	+2.0	18.0	4.0
	6.0	20.0	0.0	16.0	16.0	22.0	+3.0	6.0	-10.0
28	7.0	10.0	+1.0	3.0	14.0	18.0	+2.0	4.0	1.0
	5.0	10.0	+1.0	5.0	11.0	22.0	+2.0	11.0	6.0
	2.0	12.0	-2.0	10.0	11.0	6.0	+1.0	-5.0	-15.0
	3.0	15.0	+2.0	12.0	14.0	16.0	+3.0	2.0	-10.0
1	10.0	19.0	+1.0	9.0	22.0	26.0	+2.5	4.0	-5.0
	10.0	19.8	+0.5	9.8	24.0	26.3	+2.0	2.3	-7.5
	7.5	21.2	-2.0	13.7	17.5	32.0	-1.5	15.0	1.3
	8.0	13.5	-1.2	5.5	16.5	27.3	-0.5	10.8	5.3

Table 24

Intraluminal Pressures in Cm. of Water, Group C,
after Belsey Repair in the Chest

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
4	11.0	24.0	+2.0	13.0	39.0	50.0	+4.0	11.0	-2.0
	11.0	30.0	0.0	19.0	38.0	64.0	+2.0	28.0	9.0
	11.0	30.0	-2.0	19.0	36.0	50.0	+3.0	14.0	-5.0
	14.0	35.0	+3.0	21.0	38.0	52.0	+4.0	14.0	-7.0
8	5.0	10.0	+3.0	5.0	16.0	28.0	+5.0	12.0	7.0
	2.0	10.0	+2.0	8.0	6.0	19.0	+4.0	13.0	5.0
	5.0	10.0	+4.0	5.0	8.0	20.0	+4.0	12.0	7.0
	8.0	24.0	+2.0	16.0	18.0	30.0	+4.0	12.0	-4.0
29	8.0	12.0	-2.0	4.0	14.0	26.0	+2.0	12.0	8.0
	6.0	14.0	-2.0	8.0	17.0	23.0	0.0	6.0	-2.0
	3.0	13.0	-3.0	10.0	13.0	21.0	0.0	8.0	-2.0
	12.0	22.0	+2.0	10.0	23.0	34.0	+2.0	11.0	1.0
23	7.0	18.0	0.0	11.0	4.0	12.0	0.0	8.0	-3.0
	2.0	12.0	+2.0	10.0	14.0	12.0	+4.0	-2.0	-12.0
	10.0	14.0	-1.0	4.0	16.0	18.0	+4.0	2.0	-2.0
	4.0	10.0	-2.0	6.0	12.0	20.0	0.0	8.0	2.0
28	5.0	14.0	+3.0	9.0	16.0	18.0	+5.0	2.0	-7.0
	3.0	8.0	+2.0	5.0	12.0	20.0	+4.0	8.0	3.0
	4.0	9.0	+2.0	5.0	12.0	16.0	+5.0	4.0	-1.0
	4.0	10.3	+3.0	6.3	13.3	18.0	+4.5	4.7	-1.6
1	4.0	13.0	+2.0	9.0	14.0	34.0	+2.0	20.0	11.0
	8.0	16.0	-1.0	8.0	12.0	24.0	0.0	12.0	4.0
	2.0	8.0	-2.0	6.0	4.0	12.0	0.0	8.0	2.0

Table 25

Intraluminal Pressures in Cm. of Water, Group D, Controls

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
14	7.0	14.0	+1.0	7.0	25.0	53.0	+2.0	28.0	21.0
	6.0	18.0	-1.0	12.0	23.0	50.0	+1.0	27.0	15.0
	7.0	18.0	0.0	11.0	34.0	46.0	+2.0	12.0	1.0
	7.0	28.0	+0.5	21.0	26.0	60.0	-2.0	34.0	13.0
13	5.0	32.0	+1.0	27.0	19.0	40.0	+1.5	21.0	-6.0
	5.0	30.0	0.0	25.0	22.0	64.0	+1.0	42.0	17.0
	2.0	32.0	-2.0	30.0	14.0	64.0	-1.0	50.0	20.0
	3.0	26.0	-2.0	23.0	10.0	36.0	-2.0	26.0	3.0
9	7.5	26.0	-2.0	18.5	17.0	58.0	0.0	41.0	22.5
	7.5	48.2	-0.5	40.7	26.0	70.0	-0.5	44.0	3.3
	9.0	34.0	-0.5	25.0	24.0	88.0	0.0	64.0	39.0
	7.5	16.0	-1.5	8.5	25.0	44.6	-2.0	19.0	10.5
6	7.0	14.0	0.0	7.0	18.0	40.0	+1.0	22.0	15.0
	4.0	14.0	-1.0	10.0	17.0	52.0	-1.0	35.0	25.0
	7.0	16.0	-0.5	9.0	19.0	42.0	0.0	23.0	14.0
	7.0	23.0	+1.5	16.0	19.0	55.0	+1.0	36.0	20.0
10	9.5	14.7	-1.5	5.2	35.0	72.0	+1.0	37.0	31.8
	6.0	34.5	0.0	28.5	38.0	90.0	0.0	52.0	23.5
	6.0	46.0	-1.5	40.0	32.5	64.0	+1.0	31.5	-8.5
	11.0	29.3	+2.0	18.3	28.0	84.0	+3.0	56.0	37.7
24	9.0	32.0	+1.0	23.0	16.0	54.0	+2.0	38.0	15.0
	8.0	30.0	+3.0	22.0	22.0	56.0	+6.0	34.0	12.0
	7.0	27.0	+1.0	20.0	23.0	52.0	+2.0	29.0	9.0
	7.0	30.0	0.0	27.0	22.0	50.0	0.0	28.0	1.0

Table 26

Intraluminal Pressures in Cm. of Water, Group D, after Vagotomy

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
14	9.0	22.0	-1.0	13.0	34.0	42.0	+2.0	8.0	-5.0
	11.0	23.0	0.0	12.0	35.0	51.0	+1.0	16.0	4.0
	9.0	14.0	0.0	5.0	26.0	30.0	+1.5	4.0	-1.0
	7.0	15.0	0.0	8.0	26.0	32.0	0.0	6.0	-2.0
13	6.0	30.0	-2.5	24.0	11.0	40.0	0.0	29.0	5.0
	6.0	28.0	+1.5	22.0	21.0	51.0	+3.0	30.0	8.0
	4.0	20.0	+2.0	16.0	16.0	58.0	+4.0	42.0	26.0
	5.0	14.0	-1.0	9.0	17.0	50.0	+3.0	33.0	24.0
9	6.5	38.0	+0.5	31.5	23.5	39.0	+0.5	15.5	-16.0
	8.0	26.2	0.0	18.2	28.5	39.0	-1.0	10.5	-7.7
	9.0	21.0	0.0	12.0	24.0	30.0	0.0	6.0	-6.0
	10.5	33.0	+1.0	22.5	30.0	40.0	+1.0	10.0	-12.5
6	6.0	14.0	+1.0	8.0	15.0	23.0	+1.0	8.0	0.0
	7.0	10.0	-1.0	3.0	20.0	31.0	-1.0	11.0	8.0
	4.0	15.0	-2.0	11.0	7.0	26.0	-1.0	19.0	8.0
	4.0	10.0	-1.0	6.0	14.0	25.0	+1.0	11.0	5.0
10	7.5	20.7	0.0	13.2	33.0	42.0	+0.5	9.0	-4.2
	9.5	23.3	+0.5	13.8	31.0	32.5	+2.0	1.5	-12.3
	8.0	18.5	-0.5	10.5	33.0	34.0	+1.5	1.0	-9.5
	6.5	36.0	+3.0	29.5	28.0	32.0	+3.0	4.0	-25.5
24	8.0	22.0	0.0	14.0	27.0	34.0	+2.5	7.0	-7.0
	6.0	19.0	-2.0	13.0	31.0	52.0	+1.0	21.0	8.0
	7.0	17.0	-1.0	10.0	34.0	39.0	+5.0	5.0	-5.0
	7.0	19.5	-1.0	12.5	30.5	41.6	+1.3	11.1	-1.4

Table 27

Intraluminal Pressures in Cm. of Water, Group D,
after Thoracic Displacement of LES

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
14	5.0	6.0	+1.5	1.0	33.0	32.0	+1.0	-1.0	-1.0
	5.0	7.0	+3.0	2.0	18.0	20.0	+3.0	2.0	0.0
	12.0	16.0	+3.0	4.0	23.0	26.0	+6.0	3.0	-1.0
	12.0	24.0	+3.0	12.0	37.0	38.0	+2.0	1.0	-11.0
13	10.0	18.0	+2.0	8.0	20.0	30.0	+1.5	10.0	2.0
	7.0	27.0	-1.0	20.0	21.0	42.0	0.0	21.0	1.0
	5.0	15.0	-2.0	10.0	21.0	29.0	+1.0	8.0	-2.0
	10.0	31.0	0.0	21.0	25.0	32.0	+2.5	7.0	-14.0
9	11.0	15.0	-2.0	4.0	25.0	32.0	+2.0	7.0	3.0
	11.0	18.0	-1.0	7.0	27.0	34.0	-1.0	7.0	0.0
	12.0	20.0	0.0	8.0	31.0	39.0	+1.5	8.0	0.0
	14.0	24.0	-1.0	10.0	24.0	34.0	+2.0	10.0	0.0
6	6.0	20.0	+1.0	14.0	12.0	30.0	+3.0	18.0	4.0
	6.0	20.0	+3.0	14.0	12.0	19.0	+3.0	7.0	-7.0
	4.0	10.0	0.0	6.0	10.0	26.0	+3.0	16.0	10.0
	5.5	16.5	+1.5	11.0	11.0	25.0	+3.0	14.0	3.0
10	9.0	16.0	-0.5	7.0	34.0	36.0	-1.0	2.0	-5.0
	8.0	14.0	-1.5	6.0	28.0	35.0	+2.0	7.0	1.0
	9.0	11.0	+2.0	2.0	21.0	23.0	+3.0	2.0	0.0
	9.0	20.0	+2.0	11.0	28.0	34.0	+2.0	6.0	-5.0
24	7.0	15.0	-1.0	8.0	16.0	20.0	+6.0	4.0	-4.0
	12.0	19.0	+2.0	7.0	19.0	22.0	+4.0	3.0	-4.0
	6.0	10.0	+2.0	4.0	16.0	16.0	+6.0	0.0	-4.0
	7.0	8.0	0.0	1.0	16.0	16.0	+4.0	0.0	-1.0

Table 28

Intraluminal Pressures in Cm. of Water, Group D, after Sham Repair

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
14	11.0	28.0	+1.0	17.0	28.0	30.0	+3.0	2.0	-15.0
	14.0	34.0	-1.0	20.0	26.0	28.0	+2.0	2.0	-18.0
	11.0	26.0	-1.0	15.0	34.0	36.0	+3.0	2.0	-13.0
	8.0	14.0	-1.0	6.0	19.0	16.0	+4.0	-3.0	
13	6.0	18.0	-3.0	12.0	17.0	28.0	-2.5	11.0	-1.0
	8.0	23.0	0.0	15.0	18.0	25.0	+1.0	7.0	-8.0
	7.0	15.0	0.0	8.0	20.0	32.0	+2.0	12.0	4.0
	9.0	33.0	-1.0	24.0	20.0	35.0	-5.0	15.0	-9.0
9	14.0	24.0	-1.0	10.0	24.0	34.0	+2.0	10.0	0.0
	11.0	15.0	-2.0	4.0	25.0	32.0	+2.0	7.0	3.0
	11.0	18.0	-1.0	7.0	27.0	34.0	-1.0	7.0	0.0
	12.0	20.0	0.0	8.0	31.0	39.0	+1.5	8.0	0.0
6	8.0	13.0	0.0	5.0	21.0	24.0	+2.5	3.0	-2.0
	7.0	11.0	+1.5	4.0	14.0	16.0	+2.5	2.0	-2.0
	6.0	14.0	+1.5	8.0	12.0	22.0	+1.0	10.0	2.0
	9.0	17.0	+3.0	8.0	16.0	20.0	+3.0	4.0	-4.0
10	7.0	19.0	0.0	12.0	32.0	38.0	+1.5	6.0	-6.0
	6.0	18.0	-1.5	12.0	32.0	41.0	+1.0	9.0	-3.0
	8.0	16.0	0.0	8.0	35.0	48.0	0.0	13.0	5.0
	10.0	16.0	+2.0	6.0	24.0	40.0	0.0	16.0	10.0
24	8.0	9.0	+1.0	1.0	20.0	20.0	+10.0	0.0	-1.0
	6.0	15.0	+1.0	9.0	16.0	18.0	+7.0	2.0	-7.0
	8.0	14.0	-2.0	6.0	15.0	15.0	+2.0	0.0	-6.0
	5.0	10.0	-3.0	5.0	13.0	17.0	-2.0	4.0	-1.0

Table 29

Mean Intraluminal Pressure in Cms. of Water, Group A

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
Control									
11	6.62	31.47	-0.37	24.85	19.25	52.82	+2.0	33.5	8.65
12	5.37	32.92	-1.62	27.55	22.5	75.5	-1.75	53.12	25.57
15	5.0	17.5	-0.75	12.5	24.5	52.5	+0.12	28.0	15.5
18	7.0	17.75	+0.62	10.75	22.0	49.5	+2.12	27.5	16.75
19	5.0	23.75	-0.75	18.75	16.5	36.75	+1.75	20.25	-1.0
31	7.25	18.0	-1.0	10.75	21.25	41.25	-0.75	20.0	9.25
After Vagotomy									
11	9.62	26.5	+0.62	16.87	20.5	43.75	1.81	23.25	6.38
12	9.17	22.95	-0.12	13.77	24.25	40.25	+0.25	16.0	2.23
15	8.5	17.0	+0.37	8.5	31.0	46.0	+0.75	15.0	6.5
18	7.5	17.5	+1.25	10.0	18.75	30.25	+2.87	11.5	1.5
19	5.0	13.25	0.0	10.75	23.25	32.5	+1.5	9.25	-1.5
31	7.0	13.0	-1.0	6.0	19.5	34.75	+1.75	15.25	9.25
After Thoracic Displacement									
11	6.75	15.75	+0.87	9.0	17.75	29.75	+2.5	13.25	4.25
12	7.75	23.25	+0.12	15.5	19.0	35.25	+1.25	16.25	0.75
15	5.62	12.0	-0.37	6.37	23.37	28.0	+2.62	4.0	-2.37
18	8.25	14.75	+0.25	6.5	18.25	23.75	+0.37	4.25	-2.25
19	4.75	12.25	-1.75	7.5	11.5	22.75	+1.25	11.0	3.5
31	9.75	15.75	-1.75	6.0	22.75	27.5	+1.0	4.75	-1.25
After Allison Repair									
11	8.75	21.75	-0.75	13.0	21.25	33.0	0.0	11.75	-1.25
12	7.75	33.5	-1.25	26.25	19.5	50.0	+0.25	30.5	4.25
15	8.5	23.25	-0.25	14.75	38.75	52.25	+1.0	13.25	-1.5
18	6.75	23.0	-1.87	16.25	27.5	45.0	+1.5	17.5	1.25
19	5.0	18.5	-1.75	12.5	19.25	33.75	-0.25	14.0	1.5
31	11.25	24.5	+0.25	13.25	30.75	44.5	+1.25	13.75	0.5

Table 30

Mean Intraluminal Pressure in Cms. of Water, Group B

Dog No.	Resting				Compression				
	Stomach	Sphincter	Esophagus	St-Soh Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	St-Sph Gradient Difference
Control									
25	6.0	24.25	-0.62	18.25	16.0	46.5	+1.5	30.5	12.25
3	8.25	36.25	+0.75	28.0	20.5	76.5	+2.25	56.0	28.0
7	8.75	23.62	0.0	14.87	31.0	53.5	+2.75	23.0	8.13
17	4.5	24.0	-0.25	19.5	15.5	46.5	0.0	31.0	10.5
24	7.75	29.75	+1.25	23.0	20.75	53.0	+2.5	32.25	9.25
16	9.62	55.0	+1.37	45.37	21.12	78.5	+2.87	57.37	12.0
After Vagotomy									
25	6.32	20.65	+1.0	14.32	19.0	35.65	+1.65	16.65	2.33
3	9.5	22.0	+0.18	12.5	30.0	46.0	+0.87	16.0	3.5
7	10.25	34.75	-0.62	24.5	28.87	60.0	+1.42	31.4	6.9
17	4.5	20.25	-1.5	15.75	17.75	34.5	-1.25	16.75	1.0
24	7.0	19.37	-1.0	12.37	28.12	41.65	+2.45	11.0	-1.37
16	8.5	28.7	+2.75	20.2	13.2	36.5	+3.25	23.2	3.0
After Thoracic Displacement of LES									
25	9.0	25.5	-0.5	16.5	18.5	30.75	+0.5	12.25	-4.25
3	6.75	16.0	+0.25	9.25	25.25	35.25	+1.87	10.0	0.75
7	8.25	22.5	+1.25	14.25	26.25	34.75	+1.87	8.5	-5.75
17	4.75	12.25	-1.37	7.5	13.25	24.5	-1.12	11.2	3.7
24	8.0	13.0	+0.75	5.0	16.75	18.5	+5.5	1.75	-3.25
16	9.2	21.7	+3.37	12.5	13.2	30.7	+4.37	17.5	5.0
After Belsey Repair									
25	9.25	46.0	+0.25	36.75	17.5	61.25	0.0	41.25	4.5
3	6.5	21.5	-0.75	18.25	25.5	58.5	+3.0	33.0	14.75
7	9.25	24.25	+0.25	15.0	26.75	47.5	+0.5	20.75	-6.0
17	4.25	14.75	-1.5	10.5	16.75	49.0	-1.25	32.25	21.75
24	8.0	24.75	-2.5	16.75	21.5	49.25	+1.5	27.75	11.0
16	11.0	31.0	+0.25	20.0	23.7	49.0	+2.2	25.2	5.2

Table 31

Mean Intraluminal Pressures in Cms. of Water, Group C

Dog Number	Resting				Compression				St-Sph Gradient Difference
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	
Control									
4	6.25	27.7	-0.25	21.5	22.0	61.0	+0.5	39.0	17.5
8	6.67	26.67	+2.37	20.5	15.75	45.82	+2.0	30.0	10.0
29	7.25	21.0	-2.25	13.75	22.0	44.0	-0.5	22.0	8.25
23	5.0	21.25	-0.87	16.25	14.5	47.5	-0.87	32.0	15.75
28	6.25	21.5	-0.5	15.25	16.5	39.75	+2.0	23.25	8.0
1	8.25	29.82	+0.37	21.57	21.7	60.85	+1.62	39.02	17.45
After Vagotomy									
4	4.25	24.0	-0.25	19.75	17.25	31.5	+2.5	14.25	5.5
8	10.95	30.05	+1.55	19.3	25.8	33.42	+1.67	7.5	-11.8
29	7.25	16.5	-0.25	9.25	21.25	28.25	-1.0	7.0	-2.25
23	6.25	18.25	-0.5	12.0	18.0	28.75	+1.0	11.75	-0.25
28	8.25	21.75	0.0	13.5	17.75	35.0	+2.0	17.25	3.75
1	8.87	20.07	-0.42	11.2	23.87	40.2	+0.5	16.25	5.05
Following Thoracic Displacement of LES									
4	10.0	28.0	+0.37	18.0	24.3	36.6	+2.25	12.3	-5.7
8	7.0	13.75	+1.5	6.75	13.5	18.5	+1.62	5.0	-1.75
29	7.5	13.25	-1.5	5.75	21.75	24.5	+2.0	2.75	-3.0
23	6.5	18.25	0.0	13.5	17.0	29.0	+2.5	12.0	-1.5
28	4.25	11.75	+0.5	7.5	12.5	15.5	+2.0	3.0	4.5
1	8.87	18.37	-0.42	9.5	20.0	27.9	+0.62	8.02	-1.48
After Intrathoracic Belsey Repair									
4	11.75	29.75	+0.75	18.0	37.75	54.0	+3.25	16.25	-1.75
8	5.0	13.5	+2.75	8.5	12.0	24.25	+4.25	12.25	3.75
29	7.25	15.25	-1.25	8.0	16.75	26.0	+1.0	9.25	1.25
23	5.75	13.5	-0.25	7.75	11.5	15.5	+2.0	4.0	-3.75
28	4.0	10.32	+2.5	6.32	13.32	18.0	+4.62	4.67	-1.65
1	5.0	19.2	-0.75	14.25	11.5	29.5	+0.12	18.0	3.75

Table 32

Mean Intraluminal Pressures in Cms. of Water, Group D

Dog Number	Resting				Compression				
	Stomach	Sphincter	Esophagus	St-Sph Gradient	Stomach	Sphincter	Esophagus	St-Sph Gradient	St-Sph Gradient Difference
Control									
14	6.75	19.5	+0.12	12.75	37.0	52.25	+0.75	25.25	12.5
13	3.75	30.0	-0.75	26.25	16.25	51.0	-0.12	34.75	8.5
9	7.87	31.0	-1.12	23.17	23.0	65.15	-0.62	42.1	18.83
6	6.25	16.75	-0.12	10.5	18.25	47.25	+0.25	29.0	19.5
10	8.0	31.12	-0.25	23.0	33.37	77.5	+1.25	44.12	21.12
24	7.75	29.75	+1.25	23.0	20.75	53.0	+2.5	32.25	9.25
After Vagotomy									
14	9.0	18.5	-0.25	9.5	30.25	38.75	+1.12	8.5	-1.0
13	5.25	23.0	0.0	17.75	16.25	49.75	+2.5	33.5	15.75
9	8.5	29.55	0.37	21.05	26.5	37.0	+0.12	10.5	-9.55
6	5.25	12.25	-0.75	7.0	14.0	26.25	0.0	12.25	5.0
10	7.87	24.62	+0.75	16.75	31.25	35.12	+1.75	3.87	-12.88
24	7.0	19.37	-1.0	12.37	28.12	41.65	+2.45	11.0	-1.37
After Thoracic Displacement of LES									
14	8.5	13.25	+2.62	4.75	27.75	29.0	+3.0	1.25	-3.5
13	8.0	22.75	-0.25	14.75	21.75	33.25	+1.25	11.5	-3.25
9	12.0	19.25	-1.0	7.25	26.75	34.75	+1.12	8.0	0.75
6	5.25	16.25	+1.37	11.25	11.25	25.0	+3.0	13.75	2.50
10	8.75	15.25	+0.5	6.5	27.75	32.0	+1.5	4.25	-2.25
24	8.0	13.0	+0.75	5.0	16.75	18.5	+5.5	1.75	-3.25
After Sham Repair									
14	11.0	25.5	-0.5	14.5	26.75	27.5	+3.0	0.75	-13.75
13	7.5	22.25	-1.0	14.75	18.75	30.0	+0.12	11.25	-3.5
9	12.0	19.25	-1.0	7.25	26.75	34.75	+1.12	8.0	0.75
6	7.5	13.75	+1.5	6.25	17.75	20.5	+2.25	4.75	-1.50
10	7.75	17.25	+0.12	9.5	30.75	41.75	+0.62	11.0	1.5
24	6.75	12.0	-0.75	5.25	16.0	17.5	+4.25	1.5	-3.75

Table 33

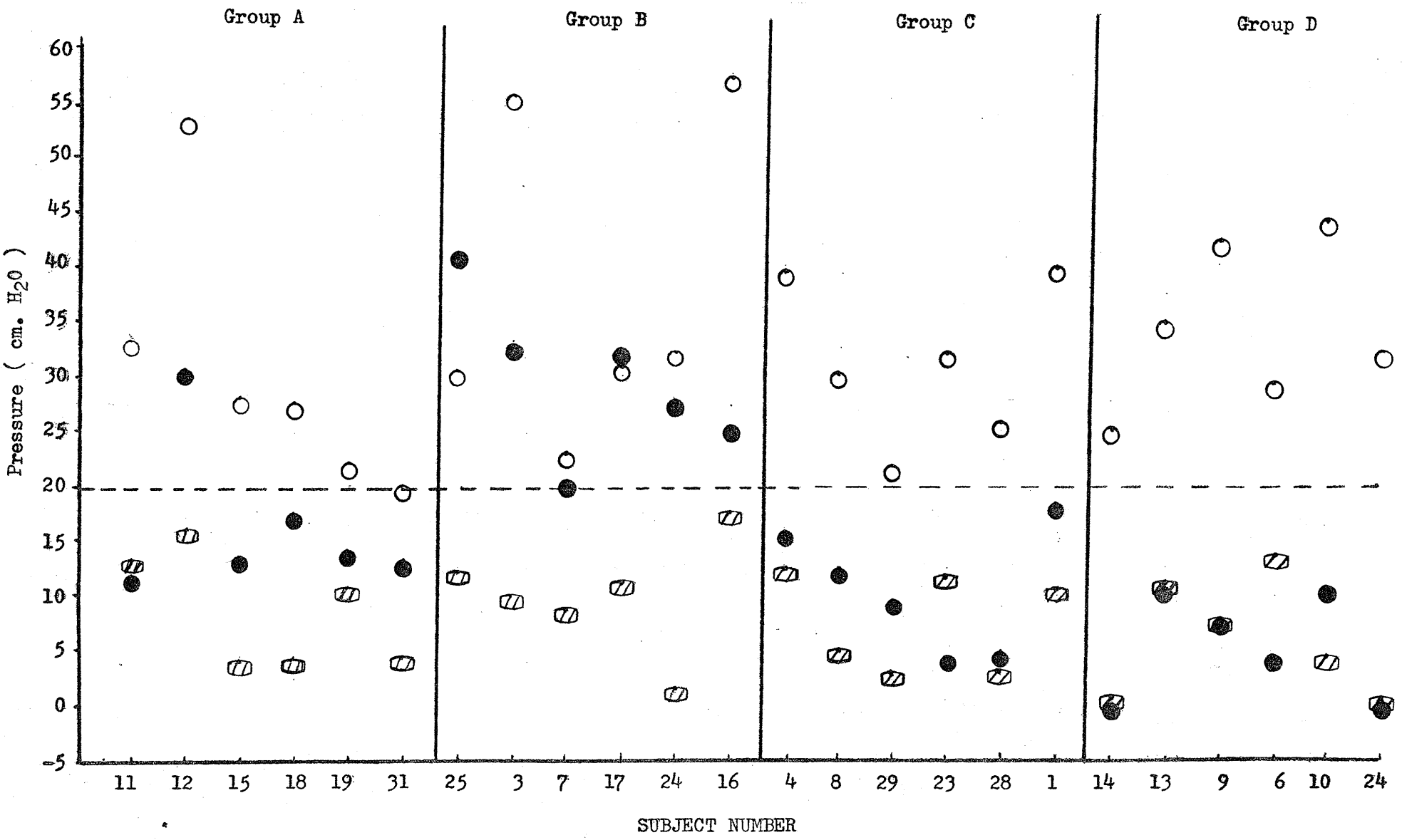
Mean Length of the High Pressure Zone in Cms., Control, after Vagotomy, after Thoracic Displacement of the GE Junction, and after Repair in 24 Dogs

Dog Number	Control	After Vagotomy	After Thoracic Displacement	After Repair
11	3.12	2.77	2.05	2.67
12	2.85	2.87	1.67	2.82
15	3.1	2.25	1.02	2.57
18	2.57	2.6	1.37	2.8
19	2.5	2.67	1.7	2.8
31	2.45	2.57	1.55	2.42
4	2.85	2.75	1.62	1.82
8	2.45	2.2	1.5	1.5
29	2.5	2.65	2.5	2.32
23	1.95	1.87	2.0	2.2
28	2.35	2.55	1.7	2.07
1	3.22	3.75	2.65	1.75
25	2.72	2.8	2.12	3.7
3	2.65	2.55	2.5	3.12
7	3.0	2.75	1.7	3.3
17	2.47	2.75	2.12	3.45
5/24	2.5	2.3	2.12	3.0
16	3.62	3.65	1.67	4.0
14	1.8	1.8	1.6	1.75
13	2.42	2.42	2.2	1.77
9	2.8	2.8	2.3	2.3
6	2.4	2.23	2.27	1.5
10	2.62	2.7	1.87	1.77
24	2.5	2.3	2.12	1.5

CODE:

○ Control
● Repair
▨ Thoracic Displacement

the stomach-to-sphincter gradient on compression (cms. water) as a function of control, thoracic displacement, and repair in 24 dogs.



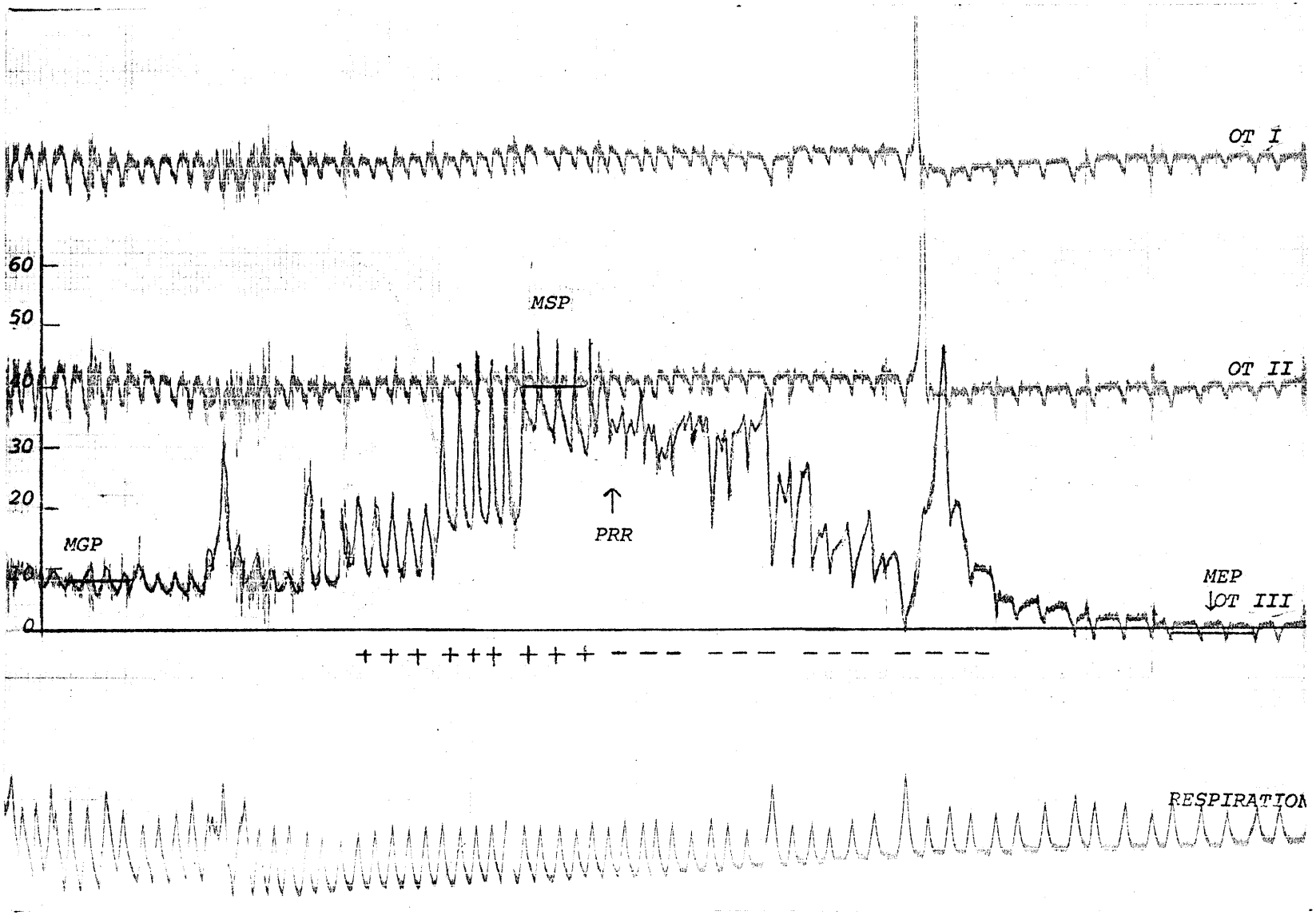


Figure 10. *Resting pressure profile of the canine gastroesophageal junction recorded by three tubes. MGP = Mean Gastric Pressure, MSP = Mean Sphincter Pressure, MEP = Mean Esophageal Presssure, PRR = Point of Respiratory Reversal.*