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Gastroesophageal junction : a study utilization intraluminal pressure measurements in patients with hiatus hernia

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THE GASTROESOPHAGEAL JUNCTION:
A Study Utilizing Intraluminal Pressure Measurements in Patients With Hiatus Hernia.

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INTRODUCTION

The study of the upper gastrointestinal tract is now over two thousand years old. The author estimates that seventy-five percent of the knowledge of this region has been obtained since 1950 because of advances in investigative technique.

However, much remains to be learned about the physiology, function, and pathology of this area. Perhaps of all the sections of the upper alimentary tract, the closing mechanism at the gastroesophageal junction has raised and still raises the most questions and controversies, especially in regard to its deranged function in the clinical disorder of hiatus hernia.

One method by which the distal esophageal sphincter mechanism may be studied is through the use of intraluminal pressure recording techniques.[7,17,18,22,25] In an attempt to gain further information about the motor function of the gastroesophageal junction, nine normal patients and three patients with hiatus hernia were studied using intraluminal pressure recording catheters.

REVIEW OF LITERATURE

The first recorded interest in the upper gastrointestinal tract was shown by Hippocrates (400-500 B.C.), who fed animals colored liquids and immediately slit their throats in an attempt to discover the function of the esophagus.[36] Investigative technique subsequently progressed to the use of a water manometer attached to a rubber catheter which inserted into a gastric fistula (1877).[36] Then balloons were attached to the catheter (1883).[36] Cannon's use of the fluoroscope in this field (1896) was refined by the use of radioopaque balloons (1915).[22] Next, a rubber nipple was added to the open tip catheter, now being passed <u>per os</u>, to prevent catheter obstruction (1928).[36] Fluid manometer use was discontinued in favor of the use of optical and electrical manometers; and flushing systems were used to prevent catheter obstruction (1947, 1951).[7] Pull through techniques (1957)[6] and fluorocine-matography (1958)[42] were next used. Most recently, radiotelemetry capsules have recorded intraluminal pressure <u>in situ</u> (1958).[17] It has been through the evolution of these techniques that the knowledge of the upper gastrointestinal tract has progressed.

Hippocrates' original work led him to conclude that a portion of the liquid swallowed by the animal entered the lung and filtered to the pericardium, where it cooled the heart while the remaining portion was exhaled as a vapor.[36]

This conception of swallowing liquids persisted until 1747 when Albinus, in De Glutitione, showed that ingested fluids do not enter the respiratory tract.[36]

Following Morgagni's 1769 recognition and classification of diaphragmatic and hiatus hernia, the first truly scientific interest in the gastroesophageal junction commenced.

By 1833, discussion of and interest in the gastroesophageal junction prompted Magendii to present his conception of the cardiac rosette, a folding of the lower esophageal mucosa which serves, he thought, as a valve preventing gastric reflux into the esophagus.[36]

The following year, Braune postulated the presence of a mucosal flap valve barrier to reflux at the gastroesophageal junction;[43] and, in 1906, Sauerbach and Von Hacker suggested a diaphragmatic pinchcock mechanism to present gastric reflux.[22] It was their thought that the diaphragm as it contracts, producing inspiration, pinches the esophagus closed.

Lendrum[30] in 1937 continued the controversy of the cardio-esophageal region and denied that there was a distinct ring of muscle to prevent reflux, as most previous investigators had thought.

Lerche's book, 1950, The Esophagus and Pharynx in Action,[31] correlating anatomy and function, has received wide acceptance to the present time. Figure I shows diagramatically his conception, with minor modifications, of the gastroesophageal junction.

By 1952, the frequent use of the balloon and catheter pressure recording methods raised the question as to the value of comparisons between results of the two methods. Thus, Hightower,[22] comparing these methods, found the catheter method to be superior to the balloon method, even though the records showed striking agreement. In the esophagus, pressure transducers showed significantly higher mean pressures than the balloon system for all waves. This was considered to be due to the subatmospheric basal pressure present in the esophagus: -5.5 cm. water, compared with a pressure of 6.2 to 10.4 cm. water in the rest of the gastrointestinal tract. The wave durations, incidence, and rates were found to be the same regardless of the system used. He also found the cardiovascular pulsations and the respiratory excursions (2.6 and 6.5 cm. water, respectively) to be the greatest in the esophagus.

The same year, Code, Hightower, and Morlock[2] found three types of esophageal waves: A primary wave with deglutition; a secondary wave as a result of distention; and a tertiary wave. They discovered that the rate of propagation of the primary wave of the esophagus decreases as the wave passes aborally.

In 1953, Sanchez et al[36] discovered that, in the upper seven-eighths of the esophagus, the swallowing complex consisted

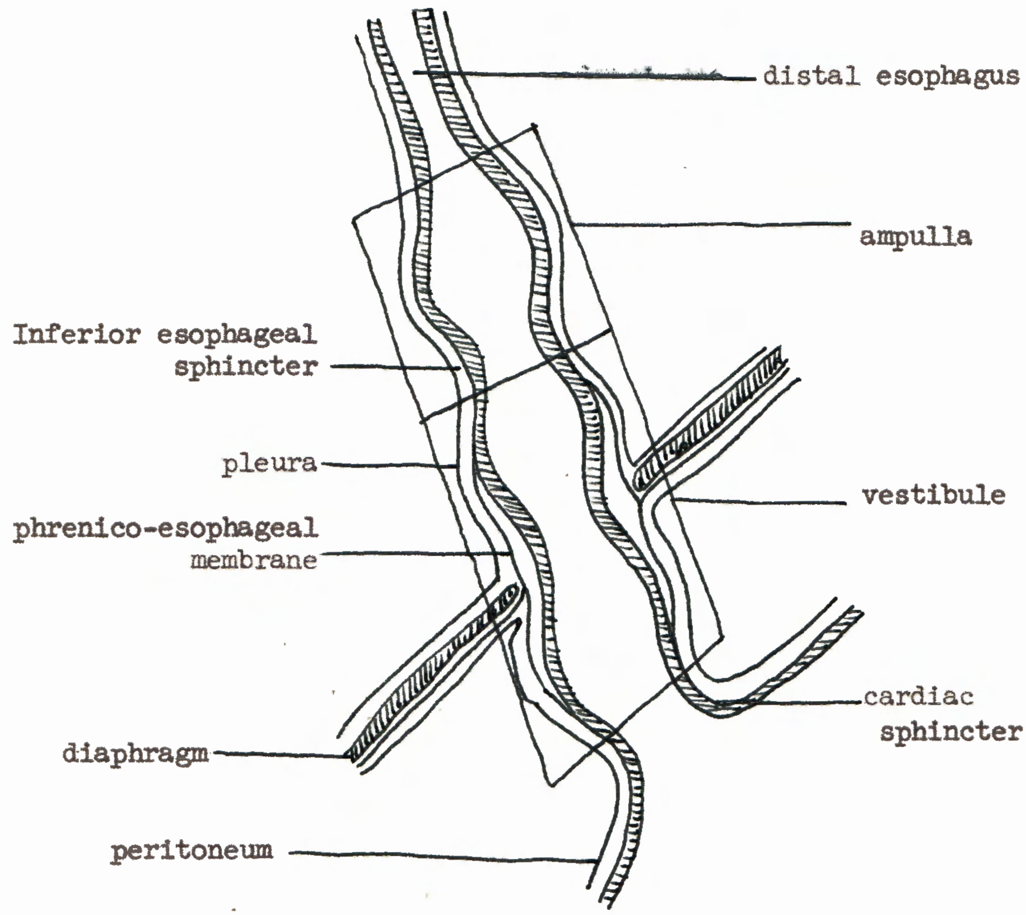


Figure I. Anatomy of the gastroesophageal junction modified after Lerche[31] and Zdanski.[48]

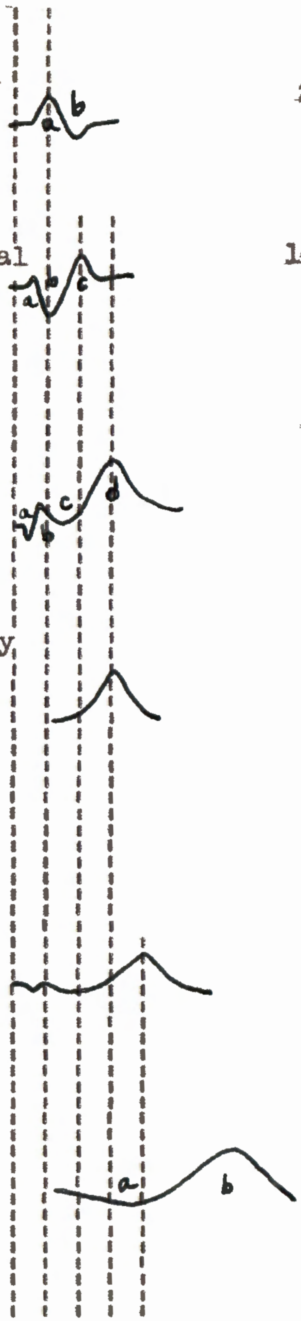
of two major components: An immediate slight elevation of pressure corresponding to the injection of oral contents into the esophagus, and a later, higher pressure wave produced by peristaltic contraction of the lumen. During rapid repeated swallowing, the primary wave was inhibited often until the last swallow.

In the distal esophagus, two different types of swallowing complexes were found. These occurred in the anatomical regions labeled by Lerche as the ampulla and vestibule. In the ampulla, the pressure created by peristalsis falls off more gradually than in the upper esophagus. The initial pressure rise following immediately upon swallowing is not transmitted into the vestibule, and the usual peristaltic pressure wave does not occur in this area (see Figure II).

In 1954, Dornhorst, Harrison, and Pierce[15] described the characteristics of normal esophageal pulsive waves. In their study, the cardia was considered to be a valve by the action of the muscularis mucosa on the mucosa. They claimed their work refuted the diaphragmatic pinchcock mechanism for reflux prevention originally suggested by Sauerbauch and Von Hacker in 1906.[22]

In Weintraub's 1954 discussion of diaphragmatic hernia and review of the literature,[44] Weintraub quoted Allison (1954) as believing that the reflux preventing mechanism at the gastroesophageal junction is dependent on the acute angle at which the esophagus enters the stomach, on the compression of the esophagus

Figure II. Deglutition complexes of the esophagus modified after Sanchez[36] and Hightower.[23,24]



	Basal Pres. (mm Hg)	Amplitude (mm Hg)	Duration (sec.)	Cause
Pharynx	2.0 to 7.5	a. 35 to 75 b. -3 to -6	a. 0.5 to 0.8 b. 0.1 to 0.2	a. primary wave b. passing bolus
Pharyngeo-esophageal junction	14.5 to 22.0	a. 22 to 37 b. neg. to 0 c. 52 to 67	a. 0.1 to 0.3 b. 0.7 to 1.0 c. 4 to 5	a. increase in tone with rise of larynx b. relaxation of muscles c. primary wave
Esophagus primary wave	-5.5	a. - b. 7 to 11 c. - d. 52 to 74	2 to 6	a. rise of larynx b. injection of fluid c. approaching wave d. primary wave
secondary wave		greater than 52	?	local distention
tertiary wave		seen in middle-aged people; are irreg. local, non-peristaltic. causes "curling" & corkscrew configuration.		
Ampulla			elongated	
Vestibule	7.9 to 2.6	a. - b. 22 to 33	greater than 7	a. decreased tone b. primary wave

by the right crus of the diaphragm, and on the intrinsic action of the circular muscle fibers of the esophagus and the oblique fibers of the stomach.

Creamer[12] the following year found that esophageal reflux could be recognized from pressure changes, for the wave was a simple one leaving the base pressure higher until the reflux was expelled by a secondary wave back to the stomach. This reflux occurred only during inspiration, started at the beginning of inspiration, and increased with the increase in intragastric pressure. The respiratory variations in hiatus hernias were noted to be commonly a bifid positive wave with inspiration, though commonly the pattern closely followed that of the intragastric pressure swing, depending upon the competency of the cardia.

Reflux, he noted, occurred more commonly in some positions than in others, although a small change in posture was not accompanied by any appreciable change in the pressure gradient across the cardia. This led him to conclude that "probably in patients with esophageal reflux the competence of the cardia depends on its relative anatomical shape."[12]

Lyon et al[32] concluded that, in the face of little support for the diaphragmatic pinchcock theory and in the face of the pressures involved in the stomach, an intrinsic sphincter could not alone be responsible for the prevention of reflux. Therefore, an intrinsic sphincter, together with a valve mechanism, seemed most likely responsible for reflux prevention.

Botha, Astley, and Carre[6] in 1957, with their pull-through techniques, discerned three types of increased pressure zone withdrawal curves proximal to the cardioesophageal junction which had no relation to respiration. They were called the "saw toothed," the "plateau," and the "step" curves (see Figure III). The total length of this high pressure segment was an estimated mean 2.6 cm. From their observation, it seemed that an important intrinsic sphincteric mechanism existed at the lower end of the esophagus.

Marchand[33] studied the forces productive of reflux and hiatal herniation and concluded that the intragastric pressure was more important than the intraperitoneal pressure in the production of reflux. He also noted that the hiatus normally moves an average of 2 cm. downward during deep inspiration.

Texter, Smith, and Barborka[40] recorded abnormal complexes from the immediate supradiaphragmatic area in patients with hiatus hernia. The alterations consisted of a decrease in amplitude; a prolongation of duration; and, in some instances, no complexes could be recorded. Because similar findings were recorded in a variety of disorders, they suggested that perhaps the disorders have a common physiological basis in a disturbed motor function.

Atkinson et al[4], 1957, pointed out that there was no corre-

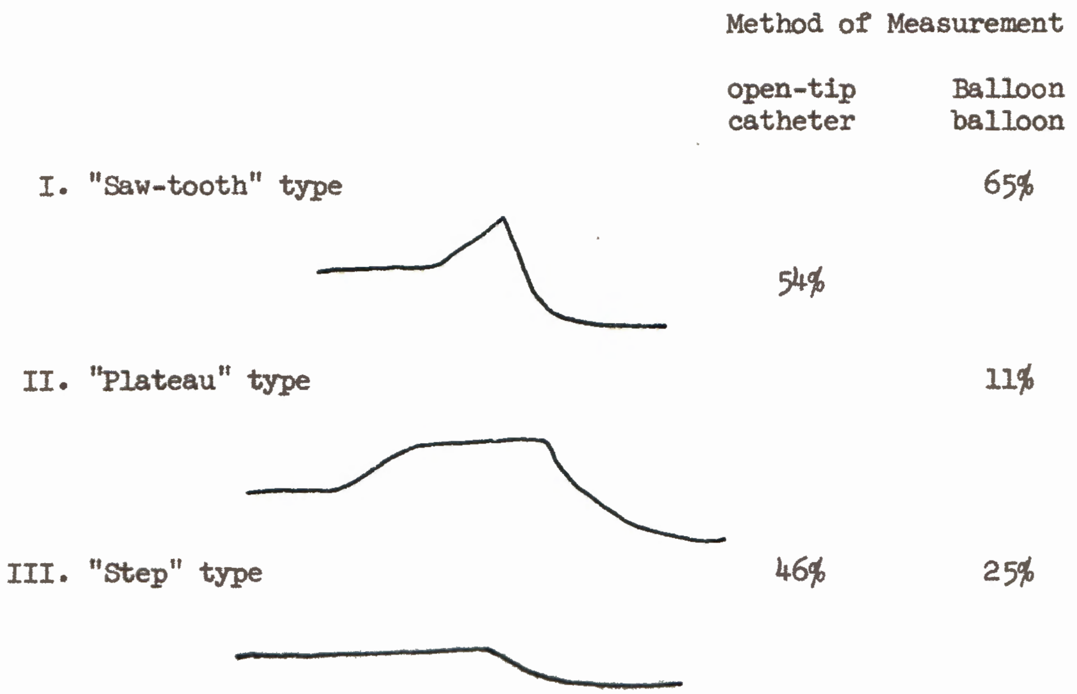


Figure III. Main types of withdrawal curves after Botha et al,[6] bearing no relation to respiratory excursions.

lation between the severity of reflux and degree of hiatus hernia. They noted the sphincteric pressure in the distal esophagus to be a mean of 9 cm. water (6-15 cm. H_2O) in patients 20-30 years old and that the high pressure zone was 1-3 cm. in length. Also, they discovered that in patients with a fixed hernia, the length of hernia on x-ray film corresponded well to the distance above the diaphragm over which the subdiaphragmatic pressure was maintained. Likewise, if the subdiaphragmatic pressure was maintained at a steady level for several cm. above the diaphragm, a hiatus hernia must be present.

Atkinson et al found that they could measure the barrier to reflux by subtracting the pressure in the hernia sac from the maximum pressure in the sphincteric zone. If the difference was 3 cm. or less, reflux was likely to occur. If the difference was 7 cm. or more, there was no reflux. This they then related to sphincter tone, stating that if the tone of the intrinsic sphincter remains normal, the patient with hiatus hernia is unlikely to have symptoms of gastroesophageal reflux; but, if the tone of this sphincter is poor, reflux is likely to develop.[4] They also found no evidence of a diaphragmatic pinchcock and no evidence to support the importance of the esophageal angle of entry.

Inglefinger[26] reviewed exhaustively the literature on esophageal motility. Following are his remarks concerning the gastroesophageal junction:

> "Although no anatomic sphincter is found in the distal 2-5 cm. of the esophagus, and some careful studies have failed to detect a distinctive motility in the area, the bulk of the evidence favors the existence of a lower esophageal sphincter. The sphincteric zone is segmental and usually lies within or immediately above the diaphragmatic hiatus. It is not certain, however, that its anatomic position is fixed; the level of maximum sphincteric activity perhaps shifts position under the narrow conditions of esophageal, gastric and diaphragmatic function. The force of sphincteric contraction, as expressed by intraluminal pressure and resistance to flow, is variable and sometimes quite feeble. Even if small in absolute terms, however, the contribution of the sphincter to the total barrier preventing gastroesophageal reflux may be crucial to the competence of that barrier.
>
> "Within 1.5-2.5 seconds of swallowing or stimulus that elicit secondary peristalsis the lower esophageal sphincter relaxes, presumably in response to the initial inhibitory phase of primary and secondary peristalsis. It is not necessary for a bolus to approach the sphincter to have it relax and a detectable peristaltic contraction is not always an obligatory antecedent. To what extent sphincteric relaxation is achieved without an antecedent peristaltic impulse is, however, uncertain. Obviously it must occur at times, particularly in association with vomiting, eructation, and regurgitation.
>
> "Following the phase of relaxation induced by swallowing or esophageal distention, the sphincter undergoes a contraction exceeding in force its resting tonicity. This contraction represents the effect of the contractile phase of the peristaltic wave on vestibule motility."[26]

Vantrappen _et al_[42] observed the high pressure zone in normal subjects which bridges the level of the diaphragm and the pressure zone in patients with hiatus hernia. They confirmed the presence of a physiologic sphincter and attempted to explain the behavior of this zone in relation to respiration, noting that with inspiration the intrathoracic pressure decreases and results in a decrease of the pressure in the supradiaphragmatic segment of the high pressure zone. The resulting pressure is then higher than in the rest of the esophagus because of wall contraction, but lower than in the infradiaphragmatic segment as a result of the increase in the intraabdominal pressure secondary to inspiration. With expiration, the reverse happens, causing an increase in pressure in the supradiaphragmatic segment of the high pressure zone, whereas the infradiaphragmatic segment remains less affected because of less respiratory pressure variations in the abdomen. They cite as evidence for this that in patients with hiatus hernia the entire pressure zone is in the thorax and respiration changes the pressure of the entire zone.

This led Vantrappen et al finally to the conclusion that, when the normal diaphragmatic esophageal relationship is maintained, the diaphragm permits the infradiaphragmatic segment of the high pressure zone to escape the influence of the negative thoracic pressure during inspiration and thereby maintain a pressure higher than that of the fundic pressure, thus constituting a barrier against reflux.

In 1959, Zdanski[48] noted that the emptying of the contents of the ampulla was inhibited and delayed by deep inspiration which produced a constriction against the tightly contracted cardiac antrum. They noted that this constriction persisted until expiration led to relaxation both of the constriction and of the antrum and thus allowed the rapid passage of contrast medium into the stomach.

Creamer, Harrison, and Pierce[13] defined the gastroesophageal sphincter as the zone of increased pressure found in the distal esophagus and stated that it had no radiological counterpart. They define the "Pressure barrier" as the place where respiratory pressure tracings reverse from the negative pressure on inspiration in the esophagus to the positive pressure on inspiration in the abdomen. This point has been called the pressure inversion point (PIP) or the point of respiration reversal (PRR). Measuring the infradiaphragmatic portion of the esophagus, they found with open-tip catheters that it was 2.5 cm. in mean length compared to 2.0 cm. mean length by x-ray.

Further, Creamer et al noted that the opening and closing of the abdominal esophagus had the characteristics of a valve preventing reflux as well as propagation of material to the stomach, as noted by Zdanski.[48] Without the pressure sphincter, this alone was not enough to prevent reflux, should intragastric pressure exceed intraabdominal pressure. This gave them the following explanation for incompetence occurring with hiatus hernia:

> " . . . if the abdominal gullet is herniated into the chest the valvular action is lost. Competence will then depend upon the ability of the sphincteric area to allow the mucosal folds to plug the cardiac orifice,

and a good correlation between sphincteric tone and esophagitis in hiatus hernia has been demonstrated by Atkinson and others."[13] (See Figure IV.)

Kelley et al,[27] in 1960, found that a small amount of positive pressure remains interposed between the stomach and the esophagus even during periods of "sphincter" relaxation. This showed that relaxation of the vestibule is relative and that, even when relaxed, it might act in some way to prevent reflux.

Wolf[46] pointed out that increased pressure zone *per se* can only withstand pressures of 6-16 cm. water, while reflux does not occur until pressure is in the range of 100 cm. water. He then applied the "Law of Laplace" in an attempt to show that another factor is present to prevent reflux: To initiate flow through the gastroesophageal junction, the pressure in the ampulla must be greater than the pressure in the abdominal esophagus, which in turn must be greater than the gastric pressure, and the volume of the abdomen must increase. Conversely, in order for reflux to occur, the pressure in the stomach must exceed the pressure in the abdominal esophagus, which must, in turn, exceed the pressure of the ampulla. He concludes, therefore, that there must be an abdominal esophageal mechanism preventing reflux. In this he supports Creamer *et al.*[13] (See Figure V.) In further studies on reflux, Wolf also supported the findings of other authors[33,34,38,45,46] with regards to the effect of intraabdominal and intragastric pressures on reflux production.

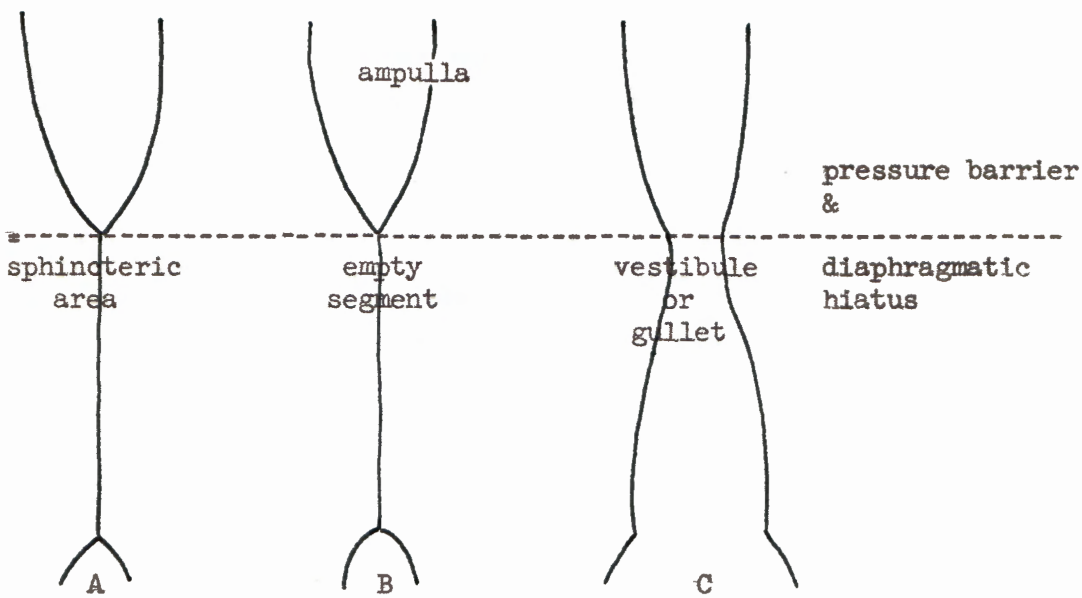


Figure IV. Action of the abdominal gullet after Creamer *et al*[13]
(A) empty esophagus (B) esophagus full with closed gullet on inspiration (C) esophagus full with dilated gullet on inspiration.

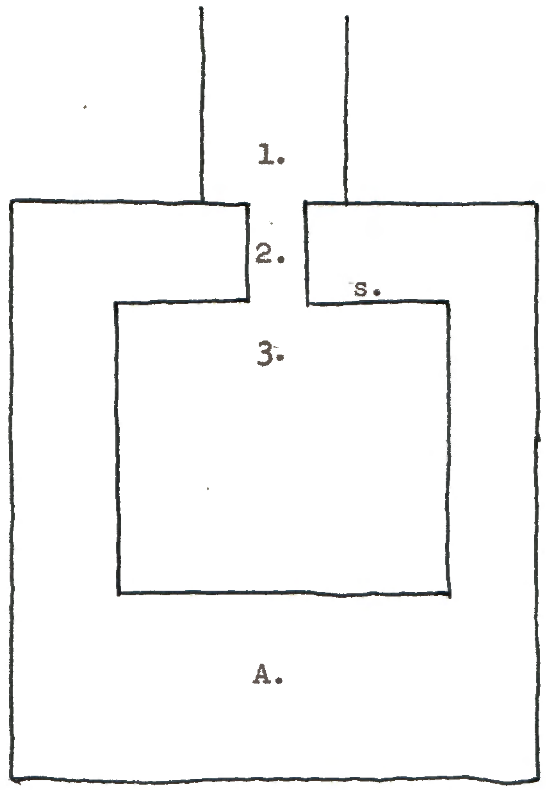


Figure V. Applied Law of Laplace after Wolf.[46] To initiate flow through 2: (1.) P_1 must be greater than P_2 which must be greater than P_3. (2.) P_2 must be greater than P_s. (3.) the volume of A must increase.

Citing Creamer's work[13] as indicating that the physiological hiatus coincides well with the anatomical hiatus, in that both are located at the upper margin of the empty sac bridging the diaphragm on x-ray, Wolf and Cohen[47,10] noted that the lower end of the phrenic ampulla serves as a useful landmark to indicate the position of the hiatus. They also found that the vestibule, by measurement, extends above the junction of the ampulla and the narrowed segment (see Figure VI).

Wolf and Cohen,[47,10] in addition, found in some patients with sliding hiatus hernias, presumably those with an extraordinarily wide hiatus, that there may be considerable difficulty in recognizing a unique position for the PIP and that a variety of types of intraluminal pressure curves may be obtained from the herniated portion of the stomach.

These investigators measured the high pressure zone as being a mean 3 cm. long, with a 2 cm. junctional segment. Defining the PIP as a site at which a biphasic pressure response was obtained accompanying a single inspiration or at which a completely positive inspiratory response abruptly changed to a completely negative one as the recording device was continually pulled cephalad, they assumed the PIP represented the demarcation between the intrathoracic and intraabdominal pressures and resulted from the downward excursion of the hiatus during inspiration. They therefore took the PIP to indicate the level of the hiatus and believed

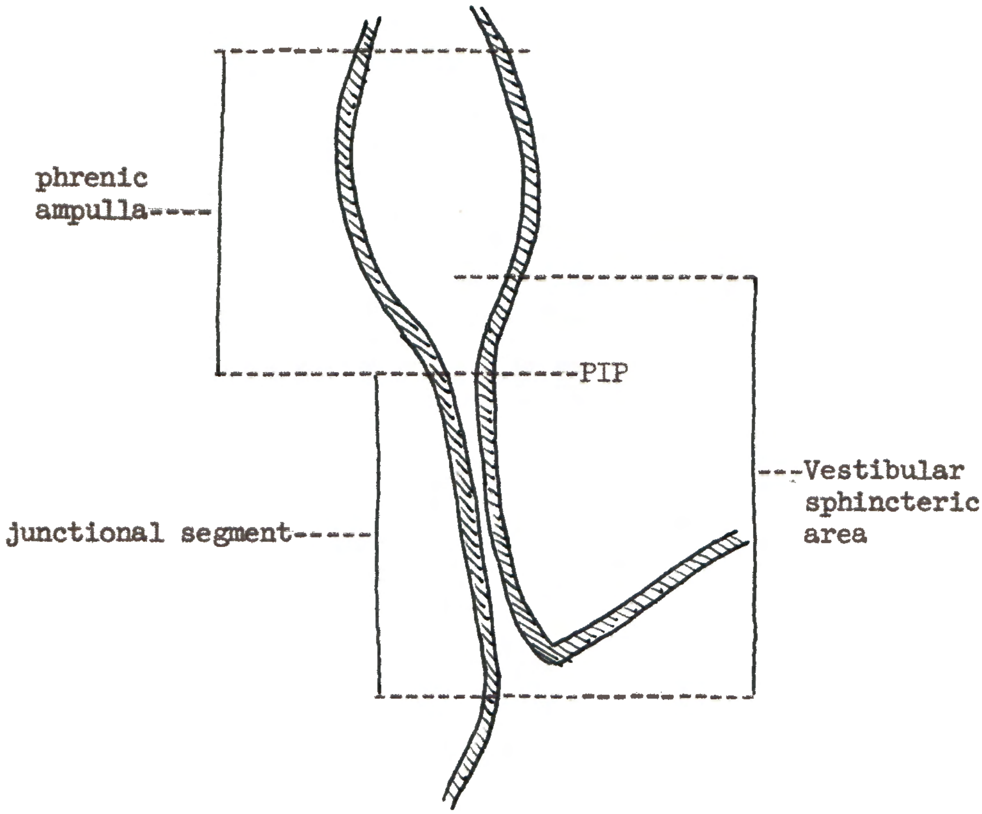


Figure VI. Functional anatomy of the gastroesophageal junction, after Cohen and Wolf.[10,47]

that the decrease in intrathoracic pressure during inspiration produced the distention of the immediate suprahiatal esophagus and the creation of the phrenic ampulla. This is in full agreement with Creamer _et al._[13]

Late in 1962, Code _et al_[9] stated that they believed that esophageal motility tests could be used to detect hiatal hernias, where, as before, reflux could be detected with pressure measuring equipment.

The pressure recording criteria for the diagnosis of hiatus hernia were based on phenomena also noted in patients with hiatus hernia by other authors[6,10,12,33,47] and are as follows:

A. Two points each to -

1. A double reversal of the respiratory excursions.
2. Two pecks of pressure.
3. Increased length of the pressure zone.
4. A plateau configuration of pressure.
5. Indications of sliding at the hiatus:
    a. Irregularity in lower margin of the zone.
    b. Excessive swings of pressure with breathing or swallowing.

B. One point each to -

1. Altered motor activity with deglutition.
2. Exceedingly high or low junctional pressure.

A score of four or more points derived from these criteria indicates that the patient has a hiatus hernia. (See Figure VII.)

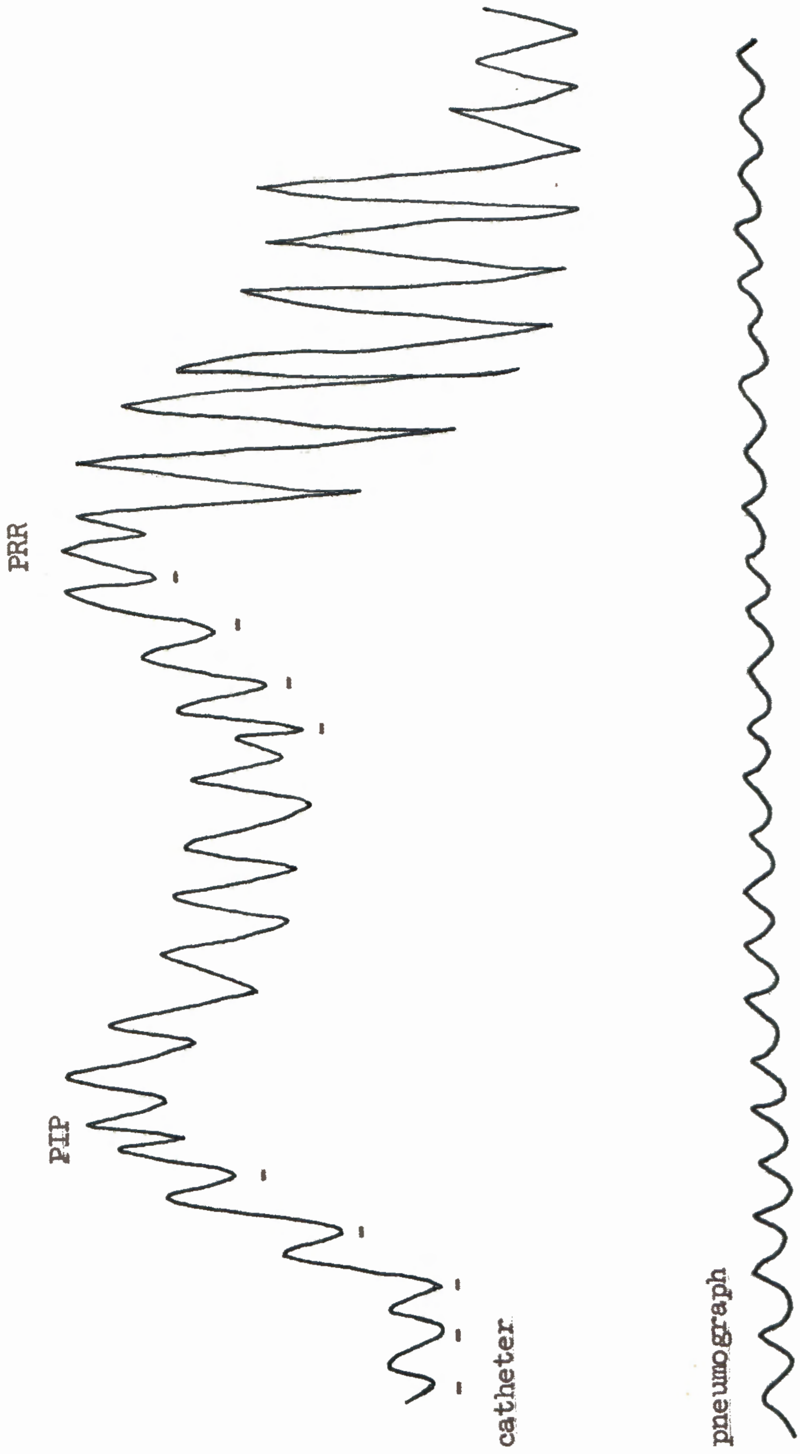


Figure VII. Pressure profile during catheter withdrawal after Code et al[9] showing an increased length of the zone, a double respiratory reversal (at PIP and at PRR), a plateau, two pressure humps (the lower at PIP, the upper at the true sphincter), large changes in pressure with breathing, and excessive pressure. The numerical evaluation is nine.

APPARATUS, METHODS, SUBJECTS

An open-tip catheter method for intraluminal pressure measurement was used similar to that originally described by Brody and Quigley.[7] The catheters used were Intramedic polyethylene tubing size P.E.-190* with an internal diameter of 0.047 inches and an outside diameter of 0.067 inches. Three approximately three-foot lengths of tubing were taped together in such a manner that the tips were 3 cm. apart. These tubes were passed into the stomach per os and then attached to pressure transducers. (See Photo I.)

Four Statham Physiological Pressure Transducers** transmitted the pressures from the water filled catheters and the respiratory pneumograph to an Electronics for Medicine Recorder. (See Photo II.)

An automatic flushing system prevented catheter obstruction by flushing the catheters and their transducers with approximately 0.075 ml/min. of distilled water. (See Photo III.)

In order to withdraw the catheters steadily, a Hayden Spirometer motor,*** attached pulley, and 2-0 silk suture were employed. This arrangement withdrew the catheters at a rate of 3.5 mm/sec. (See Photo IV.)

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* Clay Adams Company, Inc., New York
** Statham Laboratories, Inc., Los Angeles, California
*** Hayden Co., Torrington, Connecticut

Photo I. Polyethylene catheters used for recording intraluminal pressures.

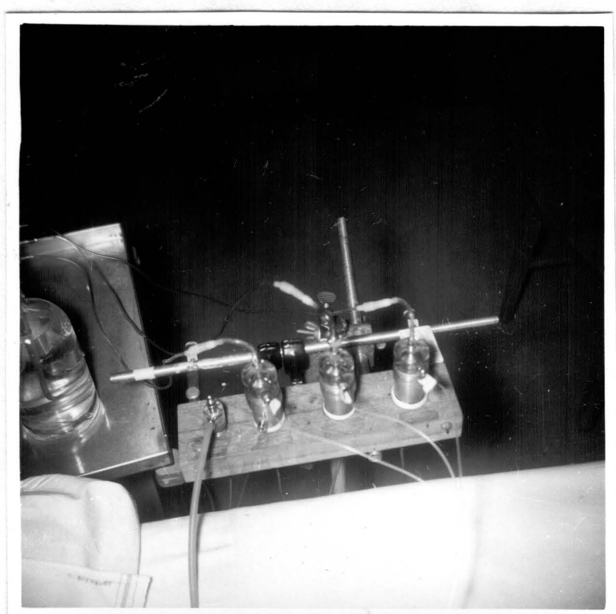

Photo II. Statham Phisiological Pressure Transducers in stand.

Photo III. Automatic catheter and transducer flushing equipment.

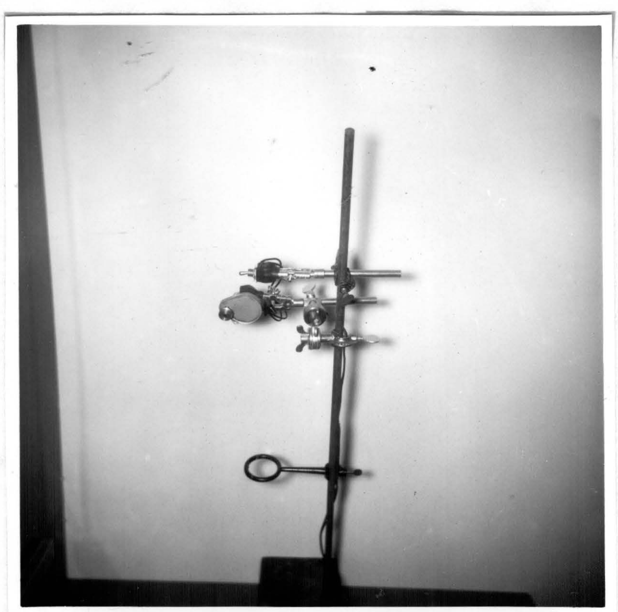

Photo IV. Catheter withdrawal motor.

The recorder utilized was a model PR-6 multichannel Electronics for Medicine Research Recorder**** containing a cathode ray recording camera and timer. (See Photo V.)

Photo VI shows the completed assembly.

Using end-expiratory pressures in all cases as the baseline pressures, the following information was calculated from each withdrawal record:

1. Length of increased pressure zone.
2. Location of the PIP: distance of the PIP from the proximal margin of the increased pressure zone.
3. Amplitude of the highest pressure in the zone relative to the abdominal baseline (excluding motility waves).
4. Difference between the gastric and the esophageal baselines.
5. Pressure of the PIP relative to the abdominal baseline.
6. Type of withdrawal curves.

Three types of withdrawal curves were recognized and defined as follows:

1. Plateau type: That pressure configuration produced by a withdrawing catheter as the catheter passes through a zone of increased pressure whose end-expiratory pressures distal to the PIP exceed the average end-inspiratory pressures distal to the pressure zone and whose duration exceeds that of one average respiratory cycle. (Figure VIII - I.)

2. Step type: That pressure configuration produced by a withdrawing catheter as the catheter passes through a zone of increased pressure whose end-expiratory pressures distal to the PIP do not exceed the average end-inspiratory pressure distal to the pressure zone and whose duration exceeds that of one average respiratory cycle. (Figure VIII - II.)

****Electronics for Medicine, Inc., White Plains, N. Y.

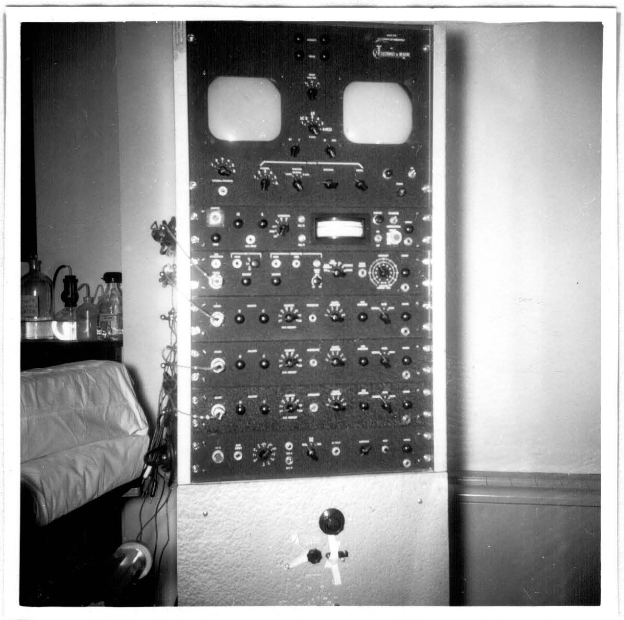

Photo V. Electronics for Medicine Research
Recorder.

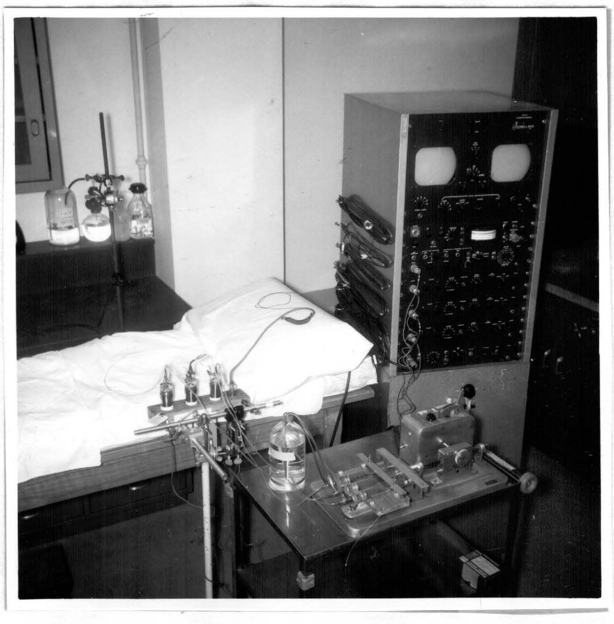

Photo VI. Assembly of Pressure Measuring
Equipment.

3. Saw-tooth type: That pressure configuration produced by a withdrawing catheter as the catheter passes through a zone of increased pressure whose duration does not exceed that of one average respiratory cycle. (Figure VIII - III.)

Figure IX shows a sample record obtained from a normal subject.

The pressure tracings on which this thesis is based were obtained from thirty-six determinations on nine normal subjects and eleven determinations on three patients with hiatus hernia. The patients with hiatus hernia were those previously diagnosed as such roentgenographically.

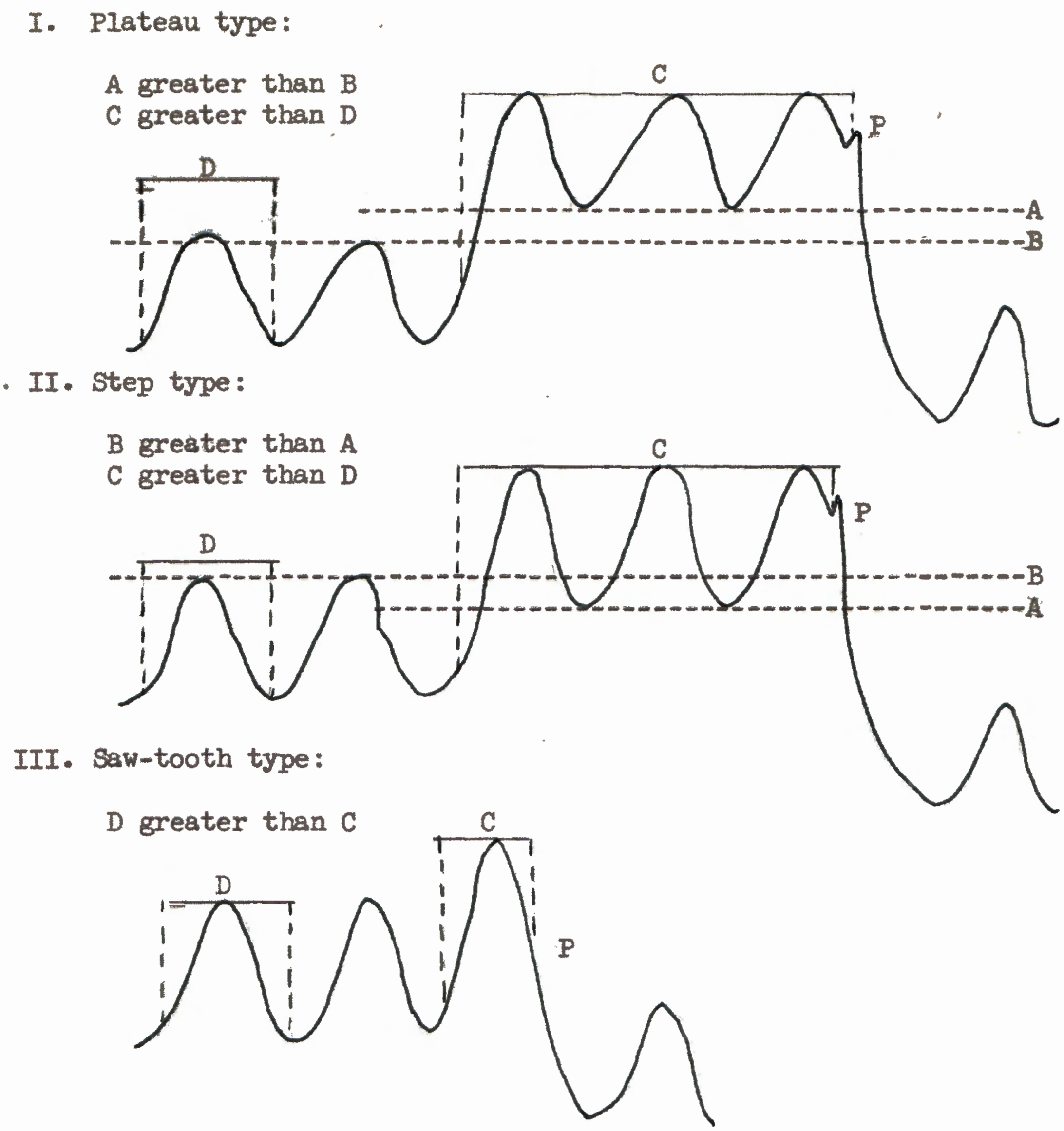

Figure VIII. The types of withdrawal curves, as defined by the author:

A. Mean end-expiratory pressure.
B. Mean end-inspiratory pressure.
C. Length increased pressure zone distal to the PIP.
D. Average duration of respiratory curve.
E. Pressure Inversion Point (PIP).

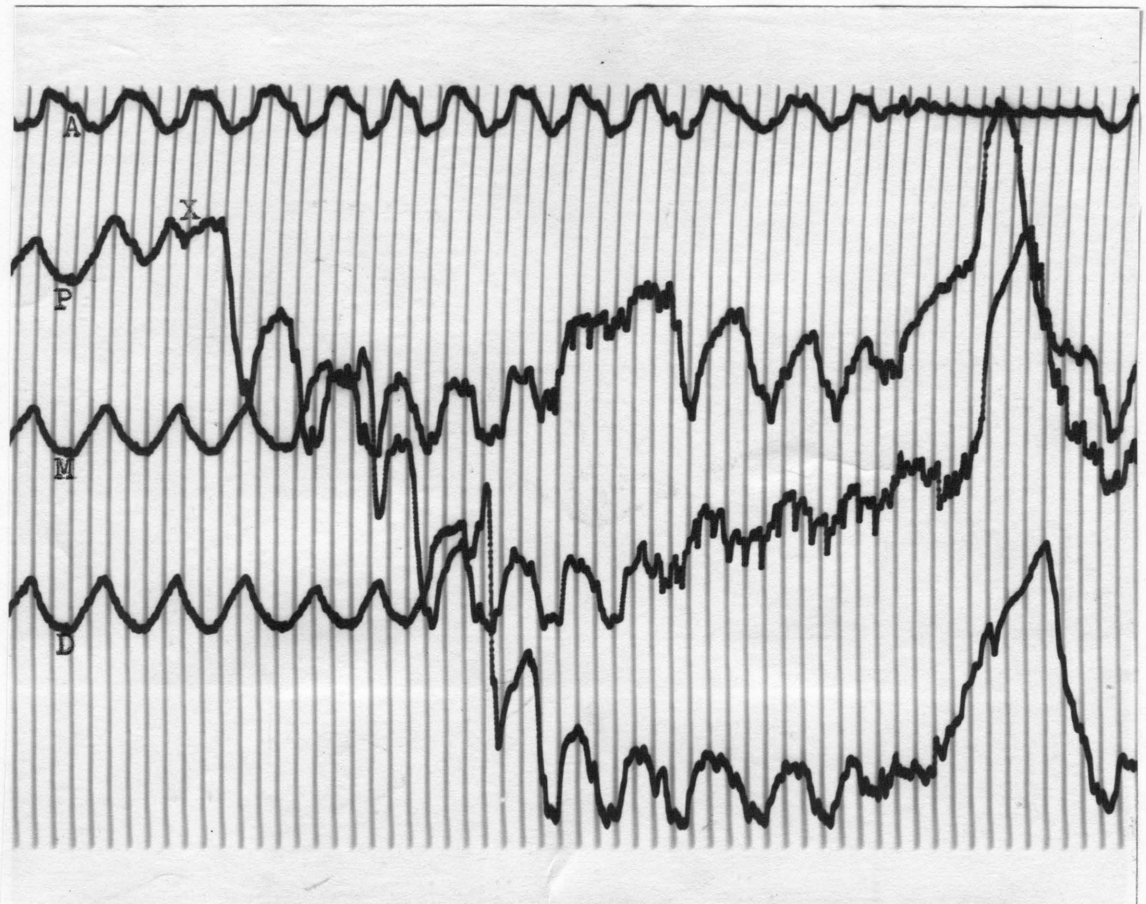

Figure IX. A sample recording of intraluminal pressures during catheter withdrawal through the gastroesophageal junction of a normal subject. (A) pneumograph tracing, (P) (M) (D) proximal, middle, and distal catheter tracings respectively. The time interval is one second. Note that curves P and M are step-type curves, while curve D is a plateau-type curve. The sphincteric segment recorded by P is 3.85 cm. in length. (X) marks the PIP, and 1.0 cm. in amplitude equals approximately 6.0 mm. Hg. pressure. The withdrawal curves are followed by deglutition complexes.

Results - Normal Subjects

Patients	Number of Determination	Length of the increased pressure zone (cm) mean	S.D.	range	Location PIP from proximal edge of increased pressure zone (cm) mean	S.D.	range
A	4	2.5	0.8	1.4 to 3.5	0.6	0.3	0.4 to 1.1
B	9	3.0	1.0	1.8 to 4.6	1.3	0.9	0 to 3.2
C	3	3.0	0.7	2.4 to 3.9	1.3	0.7	0.9 to 2.1
D	12	3.8	1.0	2.5 to 6.3	1.4	0.4	0.9 to 2.1
Accumulated	28	3.3	± 1.0	1.4 to 6.3	1.2	± 0.4	0 to 3.2

Patients	Nu Number of Determinations	Highest pressure in increased pressure zone (mm Hg) mean	S.D.	range	Gastric-esophageal pressure difference (mm Hg) mean	S.D.	range
A	4	4.4	2.2	2.6 to 7.2	7.6	1.3	6.5 to 9.1
B	9	11.0	4.6	5.7 to 21.9	9.7	0.9	8.3 to 11.0
C	3	14.3	1.9	12.1 to 15.6	8.7	0.8	8.0 to 9.5
D	12	11.8	3.1	5.8 to 16.3	8.6	2.0	3.9 to 10.4
E to I	8	8.5	3.6	4.8 to 16.8	5.9	1.7	3.1 to 8.4
Accumulated	36	10.1	± 4.2	2.6 to 21.9	8.3	± 1.9	3.9 to 11.0

Patients	Number of Determinations	Pressure at PIP (mm Hg) mean	S.D.	range	Types of withdrawal curves (%) Step	Plateau	Peak
A	4	3.3	1.2	2.0 to 7.2	50	25	25
B	9	1.0	3.2	- 3.6 to 7.3	33	56	11
C	3	6.0	1.7	4.4 to 7.8	33	67	0
D	12	4.4	3.1	0 to 9.8	0	100	0
E to I	8	4.2	0.8	3.6 to 6.0	-	-	-
Accumulated	36	3.6	± 2.9	-3.6 to 9.8	21.8	71.5	7.1

Results - Patients with Hiatus Hernia

Patients	Number of Determinations	Length of the increased pressure zone (cm)			Location PIP from proximal edge of increased pressure zone (cm)*		
		mean	S.D.	range	mean	S.D.	range
X	2	5.7	0.2	4.9 to 5.3	2.1	1.2	1.8 to 2.8
Y	3	7.2	2.4	4.9 to 10.5	2.3	0.3	2.1 to 2.8
Z	6	4.6	0.9	3.9 to 6.3	2.3	0.7	1.1 to 3.2
Accumulated	11	5.4	± 1.9	3.9 to 10.5	2.3	± 0.5	1.1 to 3.2

Patients	Number of Determinations	Highest pressure in increased pressure zone (mm Hg)			Gastric-esophageal pressure difference (mm Hg)		
		mean	S.D.	range	mean	S.D.	range
X	2	5.0	2.1	2.9 to 7.0	7.2	4.3	2.9 to 11.5
Y	3	18.6	7.0	12.6 to 29.0	8.1	3.2	5.7 to 12.6
Z	6	7.1	3.4	2.9 to 13.7	9.3	2.0	7.4 to 12.5
Accumulated	11	7.0	± 3.3	2.9 to 29.0	8.6	± 3.4	2.9 to 12.6

Patients	Number of Determinations	Pressure at the PIP (mm Hg)*			Types of withdrawal curves (%)		
		mean	S.D.	range	Step	Plateau	Peak
X	2	2.3	2.2	0 to 4.5	0	100	0
Y	3	8.5	1.9	6.0 to 10.5	0	100	0
Z	6	3.5	1.8	0 to 5.1	17	67	17
Accumulated	11	4.6	± 2.7	0 to 10.5	9	82	9

* Ill-defined measurements in some determinations.

DISCUSSION

The average normal length of the zone of increased pressure was 3.3 cm., compared with lengths varying between 1.0 and 3.0 cm. found by other investigators.[6,4,10,13,35] These measurements correlate well, in view of Marchand's finding[33] that the average hiatus moves about 2 cm. with respiration.

In patients with hiatus hernia, the measurement of 5.4 cm. as the length of the increased pressure zone coincides with the findings of Code _et al_[9] who give considerable emphasis to the increased length of the pressure zone in the diagnosis of hiatus hernia.

The location of the PIP was measured normally to be 1.2 cm. distal from the proximal edge of the 3.3 cm. increased pressure zone and in patients with hiatus hernia 2.3 cm. distal from the proximal edge of the 5.4 cm. increased pressure zone. This supports Cohen and Wolf[10,14] (Figure VI, page 19) that the PIP is located in the upper part of the increased pressure zone. If their concepts of the location of the PIP coinciding with the diaphragmatic hiatus and base of the ampulla are correct, it appears that the increased pressure zone extends a mean 1.2 cm. into the ampulla normally. The 2.3 cm. measurement in hiatus hernia patients reflects the fact that the over-all length of the zone is increased.

The highest pressure recorded in the normal increased pressure zone was a mean 10.2 mm Hg, comparing to pressure of 4.8-11.8

mm Hg and 7.4 mm Hg to 29.6 mm Hg found by other investigators.[4,6,46] These measurements were thought to generally be in agreement. The mean highest pressure of 7.0 mm Hg measured in patients with hiatus hernia falls in the same general ranges, indicating that the actual pressures in the increased pressure zone are not altered by the depth of respiration occurring during measurement. Swallowing complexes were easily recognized and the resultant pressures excluded, but the respiratory effects could not be excluded, as they were needed for PIP localization. Hightower[22] found the mean amplitude of the respiratory excursions to be 4.8 mm Hg.

Measurement revealed a mean 8.4 mm Hg difference normally between the intragastric and intraesophageal pressures. This differs from Hightower's range of 0.4-3.5 mm Hg because of a possible difference in definitions of the baselines involved. His measurements were also made in relation to atmospheric pressure while that was not possible here with the procedures used.

In hiatus hernia patients, the intragastric-intraesophageal pressure difference was a mean 8.6 mm Hg, which corresponded, as expected, to the normal difference. The intraesophageal baseline was often difficult to determine accurately, however, because of wide respiratory swings and cardiovascular pulsations not seen intragastrically.

The pressure at the PIP was found to normally be 3.6 mm Hg, compared with a pressure of 4.6 mm Hg in patients with hiatus hernia. These measurements were thought to be in accord; for, in the latter group, the PIP was often difficult to pinpoint because of double reversal of the respiratory excursions.

71.5 percent of normal withdrawal curves and 81.8 percent of the hiatal hernia withdrawal curves were of the plateau type. This compares with Botha's findings[6] that normally 46 percent were of the step type and 54 percent were of either the plateau or the saw-tooth types. These figures differ perhaps because Botha did not define his curve types and perhaps because all types can be seen in any one individual and both his group of subjects and this study's group were small. These curve types are also dependent largely upon the speed of withdrawal of the recording device. This investigator's findings tend to support Code _et al_[9] who give considerable emphasis to the plateau configuration of the pressure zone in the diagnosis of hiatus hernia by intraluminal pressures. Yet one can see that other criteria are necessary for such a diagnosis, as most normals have a majority of plateau configurations, also.

In records of patients with hiatus hernia, no wave recognized as those representing reflux were seen, nor were double peaks of pressure as described by Code _et al_. Indications of sliding as outlined by those authors were noted, however.

Thus, in view of the above results and review of literature, it appears that reflux from the stomach to the esophagus is prevented normally by a zone of increased pressure around the region of the diaphragm. This zone is seen as a 3.3 cm. length of distal esophagus, represented usually by a plateau type withdrawal curve, in which is located, 1.2 cm. from the proximal edge, a pressure inversion point (PIP) marking the diaphragmatic functional hiatus and the ampullo-vestibular junction of inferior esophageal "sphincter." The pressure in this zone is approximately 10.1 mm Hg excluding deglutition and is produced by an intrinsic muscular tonus. It is normally competent enough to prevent reflux from the 4.8 mm Hg pressure caused by inspiration. It is important to remember here that even during expiration the gastric pressure exceeds the esophageal pressure by 8.3 mm Hg.

In situations producing an increase in intraabdominal pressure greater than 10.1 mm Hg, an abdominal esophageal pinchcock mechanism probably plays a part.

When the intraabdominal pressure exceeds the pressure in the increased pressure zone, the walls of the abdominal esophagus collapse and, with the aid of opposing mucosa, aid to prevent reflux.

When intragastric pressure exceeds the pressure in the zone and exceeds the intraabdominal pressure, regurgitation takes place. This occurs normally in vomiting. While gastric contractions raise the intragastric pressure to great heights, no reflux occurs normally because of the nature of the contracting walls, which cause a pressure gradient favoring movement of contents distally down the gastrointestinal tract.

In hiatus herniation, the most obvious alteration in the gastroesophageal junction is the loss of the abdominal esophagus. This loss of the abdominal esophagus prevents action of a pinch-cock mechanism; and, as a result, reflux tends to occur when intragastric pressures exceed 7.0-10.1 mm Hg.

Apparently in this condition the increased pressure zone is relatively unaltered, as mirrored in the results seen from the records examined here. The only striking change from normal was in the increased length of the pressure zone. This can be explained in about the same way Hightower[22] explained why balloon recordings in the esophagus are higher than recording from open-tip catheters. Since the normal intrathoracic pressure is negative, the increased pressure in the pressure zone causes a ballooning elongation of the zone.

Since most hiatus hernias are of the sliding type, the features of sliding as described by Code _et al_[9] are seen on records of this region. They apparently represent only an exaggeration of that which occurs during normal 2 cm. sliding of the hiatus.

SUMMARY AND CONCLUSIONS

1. A review of the literature concerning the gastroesophageal junction and methods for studying this area was presented.
2. A description was given of the open-tip catheter pressure measuring apparatus and procedures used in this study.
3. The mean length of the normal zone of increased pressure was found to be 3.3 cm., compared with a length of 5.4 cm. in hiatus hernia patients.
4. The Pressure Inversion Point was found to lie in the proximal half of the increased pressure zone in normal subjects and patients with hiatus hernia and have a pressure of 3.6 and 4.6 mm Hg respectively.
5. The highest pressure in the zone of increased pressure was noted to be 10.2 mm Hg normally, and 7.0 mm Hg in patients with hiatus hernia, showing that the amplitude of the increased pressure zone is not markedly altered in this disorder.
6. A difference of approximately 8.5 mm Hg was found between the intragastric and the intraesophageal baseline pressures in both groups of patients.
7. A plateau type of withdrawal curve was found in 71.5 percent of the normal subjects and 81.8 percent of the hiatus hernia patients.
8. Irregularities in the lower margin of the high pressure zone

and excessive swings of pressure with breathing were noted as indications of hiatal sliding in patients with hiatus hernia.

9. The mechanics of gastroesophageal reflux prevention were discussed, in view of the results found in this study, and the current thinking mirrored in the literature reviewed. In situations of gastric pressure less than 7.0-10.2 mm Hg, the increased pressure zone serves to prevent reflux. When intragastric pressures exceed this, an abdominal esophageal pinchcock mechanism prevents reflux. Thus, in situations of pure gastric elevations of pressure, such as in vomiting, reflux occurs.

   In hiatus hernia patients, the abdominal esophageal pinchcock mechanism is lost and reflux is prevented only by action of the increased pressure zone. Thus, when intraabdominal pressures exceed 7.0-10.2 mm Hg in this condition, reflux occurs.

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