Muscular Anatomy of the Gastroesophageal Junction and Role of Phrenoesophageal Ligament

Autopsy Study of Sphincter Mechanism

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It is now widely accepted that the lower esophagus possesses a sphincteric mechanism which is intrinsic to the muscle layers of the organ itself and not to contiguous structures.[3, 9, 20, 27, 41] Many investigators, using both clinical and experimental methods, have demonstrated that it is this sphincter which is the primary mechanism in the prevention of gastroesophageal reflux and its sequela, peptic esophagitis. Other mechanisms play a supporting role in the prevention of reflux but, in themselves, are insufficient if a competent sphincter is not present. Among these adjuncts to sphincter function are the sling or tunnel formed by the right crus of the diaphragm as it makes up the hiatus,[1, 2, 30, 31] the intra-abdominal segment of the esophagus,[10, 33] and possibly the acute angle of entry of the esophagus into the stomach.[2, 25] Recent investigators have attributed a supporting role to the phrenoesophageal ligament and its insertion.[34] In addition, a major role has been proposed for the phrenoesophageal ligament in the etiology of gastroesophageal reflux.[16, 17] It is the purpose of this investigation to confirm or deny this latter hypothesis anatomically in the autopsy room.

Further, physiologic evidence for the existence of a sphincter is voluminous,[12] but anatomic proof is both scanty and controversial.[9, 11, 23, 27-29, 32, 40, 44] In this investigation, we have attempted to delineate the muscular anatomy of the distal esophagus.

Gastroesophageal Junction

Anatomy. The anatomic structure of the gastroesophageal junction consists of the following (Fig. 1). The tubular thoracic esophagus progresses inferiorly, bounded in the lower thorax by the pericardium anteriorly, the aorta posteriorly and the pleurae laterally. Two to three centimeters above the hiatus of the diaphragm it is anchored at its lower end by the insertion of a tough, skirt-like prolongation of the endoabdominal fascia from the undersurface of the diaphragm, the ascending leaf of the phrenoesophageal ligament. This inserts into the esophagus and, by fascicles of fibroelastic tissue, is attached to the submucosa and intermuscular septae of the esophageal wall. According to Lerche[29] and others, at or about the insertion of

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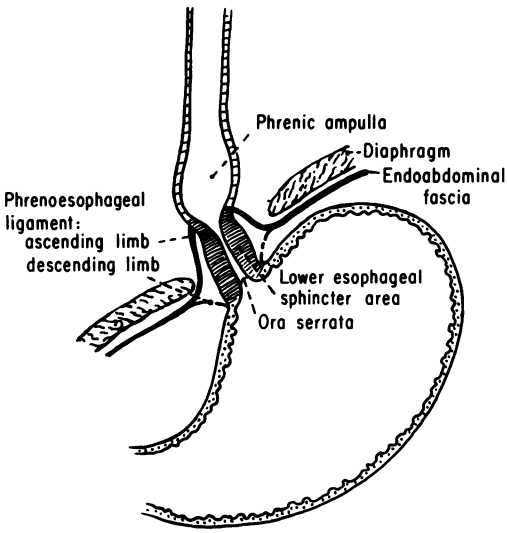

FIG. 1. Normal anatomy of the esophageal hiatus structures.

the ligament, the circular muscle layer of the esophageal wall becomes somewhat thicker than it had been at higher levels. Furthermore, the circular muscle fibers at this point and below are actually more oblique than circular. Frequently, diagonal and longitudinal muscle fascicles are found interdigitating with the oblique fibers immediately beneath the submucosa. The significance of these fibers (Laimer's bracket fibers) is not clear. They are also found in the mid-esophagus but in lesser number than in the lower portion. At a point three to five centimeters below the insertion of the phrenoesophageal ligament, the smooth, white squamous epithelium of the upper esophagus changes to the columnar epithelium of the stomach. The point of transition is marked by a jagged, serrated line (the ora serrata or "z" line) and, indeed, digital projections of gastric mucosa may extend for several centimeters up into the esophagus, although this is relatively rare. The gastric mucosa below the epithelial junction is simple columnar in type with simple tubular glands as found in the remainder of the stomach. It has been dubbed with the title "junctional epithelium" because of the demonstrated absence of oxyntic cells in this area.[26] The esophagus continues about one cm. from the epithelial junction and then flares out into the stomach.[9] The two centimeters of the esophagus immediately above the epithelial junction are contained within the sling of the right diaphragmatic crus as it forms the hiatus. The circular muscle layer of the esophageal wall continues down to the area of the esophagus below the epithelial junction where it forms the innermost two layers of the gastric muscularis. The mucosa and submucosa in the lower esophagus are thrown up into redundant folds and in some series, marked circular folds of mucosa have been noted in this area.[29, 44]

The phrenoesophageal ligament arises primarily from the endoabdominal (transversalis, subdiaphragmatic) fascia. At the lower margin of the esophageal hiatus, it decussates into an upper and a lower leaf. The upper leaf extends through the hiatus to insert into the esophagus two to three centimeters above it. The lower leaf, which exists as a loosely defined collection of fibroelastic fibers, descends to insert into the esophagus at or below the epithelial junction. The lower leaf may insert onto the gastric fundus. The upper leaf has the character of a strong, well-defined membrane rather than a ligament, despite its name.[15]

Physiology. It has been shown repeatedly by manometric technics that a sphincteric mechanism exists at the gastroesophageal junction. This barrier we refer to as the lower esophageal sphincter.

There are two conditions under which the gastroesophageal junction must maintain competence. The first is at rest and the second under conditions which raise intra-abdominal pressure generally, such as coughing, defecation, leg raising, etc. In the first, the sphincter normally maintains a pressure barrier between positive intra-

gastric and negative intrathoracic pressure. This pressure differential is approximately 10 cm. H_2O greater than intragastric pressure and 20 cm. H_2O greater than intrathoracic pressure.[12] It is present in all phases of respiration and may often be maintained even when the gastroesophageal junction is displaced into the thorax, as in sliding hiatal hernia.[13, 22] On performing the Valsalva maneuver, however, intragastric and intra-abdominal pressures can be raised to the neighborhood of 100 mm. Hg, far in excess of the values which the sphincter can withstand.[21] At this point the upper leaf of the phrenoesophageal ligament and the intra-abdominal portion of the esophagus come into play. The phrenoesophageal ligament maintains the esophagogastric junction within the abdomen, and the increased intraabdominal pressure is brought equally to bear on the abdominal esophagus containing a portion of the sphincter as well as on the stomach.[43] The sphincter, reinforced by this pressure, is able to maintain a pressure differential between the stomach and thoracic esophagus.

Pathophysiology. In hiatal hernia of the sliding type, the clinical manifestations are most commonly due to gastroesophageal reflux. Yet it is well known that hiatal hernia may exist and even attain mammoth proportions without reflux. The only mechanism which can prevent reflux under these conditions is the sphincter and then only if it can maintain the pressure relationships cited above. Thus, it may be that in hiatal hernia without reflux, the upper leaf of the phrenoesophageal ligament continues to define the abdominal compartment from the thoracic compartment or there is an extremely strong sphincter. At rest, normal pressures are maintained by the intact sphincter. When intraabdominal pressure is raised, this increase in pressure may be communicated within the hernia sac and brought to bear on the esophagus below the insertion of the phrenoesophageal ligament and reflux is prevented.

This is not to imply that the effective intraabdominal esophagus is the primary mechanism preventing reflux. This cannot be the case for two reasons. Reflux which causes peptic esophagitis primarily occurs in the resting state and not at moments when intraabdominal pressure is raised.[4] Furthermore, in the majority of instances when an individual strains, he does so after first closing the glottis and by action of accessory muscles of respiration raises the intrapleural pressure. This equalizes thoracic and abdominal pressures and diminishes the great pressure gradient which would occur if abdominal pressure alone were raised.

What then leads to the development of gastroesophageal reflux? The ready answer is that the gastroesophageal sphincter has become incompetent. The question then becomes, is it incompetent because of some intrinsic defect, or is some other correctable factor culpable. Recently, Vandertoll *et al.* have shown that an intrinsically incompetent sphincter, regardless of its location, will permit reflux.[41] Therefore, if in all cases of reflux the sphincter possesses an intrinsic defect, then the incidences of cure by operations which do nothing but replace the sphincter in its normal position are unexplainable.

Dillard recently advanced a concept [16-18] to explain those cases of sphincter incompetence in which the sphincter itself is capable of function (Fig. 2). In the normal individual, the phrenoesophageal ligament inserts into the lower end of the esophagus a short distance above the diaphragmatic hiatus. The normal sphincter is found to reside in that portion of the esophagus below this insertion. If the ligamentous insertion were to be somehow displaced lower on the esophagus, it would then insert directly into the body of the sphincter. The normal tension on the

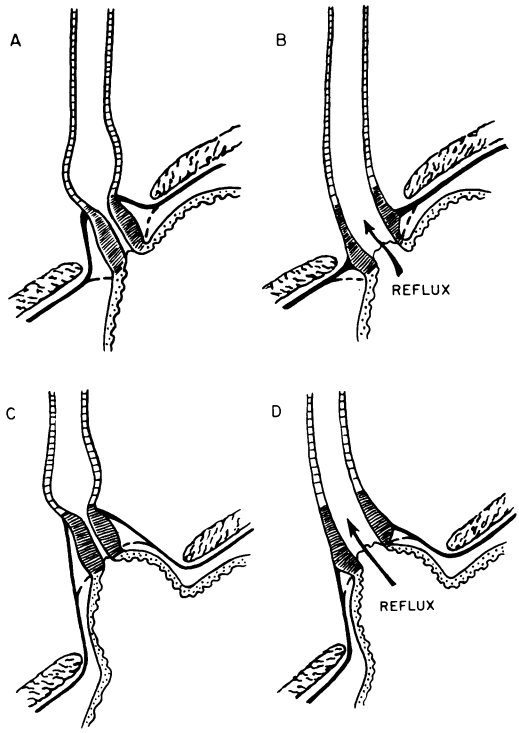

FIG. 2. The phrenoesophageal ligament dilation mechanism.

A) Normally, the ascending limb of the phrenoesophageal ligament inserts into the esophageal wall above the lower esophageal sphincter mechanism. Forces applied to the ligament by the contracting diaphragmatic musculature serve only to dilate the esophagus above the sphincter. This may be the origin of the radiologically familiar "Phrenic Ampulla."

B) If the ligamentous insertion were to be displaced inferiorly, then tension transmitted through the ligament would serve to dilate the sphincter itself, allowing gastroesophageal reflux to to occur.

C) The same situation would pertain in the patient with a sliding hiatal hernia. Normally, in the hiatal hernia patient without reflux, the ligament would insert above the sphincter area, producing the classic three radiologic criteria of hiatal hernia: The phrenic ampulla, the esophageal vestibule (corresponding to the sphincter area which has been displaced above the diaphragm), and the supra-diaphragmatic gastric loculus.

D) If the ligamentous insertion were to be displaced inferiorly, reflux would ensue. If the patient still possessed a competent sphincter, any operation which would reduce tension on the ligaments would cure the reflux.

phrenoesophageal membrane, as it anchors the esophagus, is applied parallel to the long axis of that organ. Should the hiatus be widened, however, and the ligamentous insertion displaced, tension would then be applied laterally to the sphincter, tending to distract it. Experimental evidence in our laboratories has confirmed this hypothesis in the dog.

A displaced ligamentous insertion would also explain the occurrence of gastroesophageal reflux in those individuals without herniae. In this instance (Fig. 2), all tension applied to the esophageal wall during normal respiration by the phrenoesophageal membrane would then be directed laterally and the distracting force would be enormous.

Materials and Methods

In a 6-month period at the Seattle King County Hospital, 227 autopsies were surveyed. Among these, eight cases of gross hiatal hernia and eight cases with gross evidence of acid-peptic inflammation of the esophagus, such as ulcer or distinct reddening or erosion, were found. Microscopic evaluation of all specimens subsequently showed that gross criteria of inflammation in such an organ as the postmortem esophagus are unreliable. Therefore, some cases of minimal esophagitis were probably missed in the gross survey. No specimen was included in the esophagitis group unless it met the criteria established by Moersch *et al.* on microscopic examination.[35] In addition, 48 patients who had had no disease involving the esophageal hiatus or terminal esophagus were selected as normal controls.

At autopsy, the esophagus, together with its lateral pleural covering and anterior pericardial reflection, was removed from the level of the aortic arch. A wide cuff of diaphragm, a cuff of stomach, and the involved portions of the lesser omentum

and gastrosplenic ligaments were taken "en bloc." The specimen was then placed in a wire rack so that the diaphragm was in as nearly the *in vivo* position as possible and fixed in 10% formalin for a period of 48 hours. In a few specimens, the covering pleura was dissected away in order to confirm the uniform attachment of the phrenoesophageal ligaments around the circumference of the esophagus. Dissection from below generally failed to reveal a well-defined membrane which would correspond to the lower leaf of the phrenoesophageal ligament. Instead, only a loose collection of fibro-fatty tissue was found in this area. Thick coronal and sagittal sections of 0.5 cm. were taken of each specimen, pinned to a board, and the diaphragm put on the stretch, thereby bringing the phrenoesophageal ligament into sharp relief. The distance between the epithelial junction (this being the most constant and fixed reference point in the specimen) and the upper and lower insertions of the phrenoesophageal ligament were then measured. The location of the epithelial junction relative to the diaphragm was noted. Where an obvious gross thickening in the circular muscle layer of the esophagus was noted, its length was also measured relative to the epithelial junction. Following this, a transverse section of the esophagus was taken 1 to 2 cm. above the epithelial junction and again 10 to 15 cm. above the epithelial junction. These sections were stained with hemotoxylin and eosin and submitted to microscopy. The thickness of the circular muscle layers as well as the thickness of the mucosa and submucosa was measured using a microscope with a graduated mechanical stage.

By using the criteria of Moersch *et al.*[35] the sections were graded as normal, acute inflammation, chronic inflammation and fibrosis, or ulcer. In some sections, more than one alteration was noted. All of the above inflammatory changes were taken as evidence of peptic esophagitis. All patients who had had a nasogastric tube in place or who had experienced prolonged vomiting episodes were excluded from the study.[42]

Results

Squamo-Columnar Junction. A total of 21 cases was examined for the location of the squamo-columnar epithelial junction with reference to the point at which the esophagus flared out into the stomach. In 17 of these, it was found 1.0 cm. above this point and in the other four, it ranged from 0.5 cm. to 2.0 cm. above. The average distance from the gastroesophageal junction to the squamo-columnar junction was 1.1 cm., which corresponds well with the figure reported by Botha.[9]

Lower Leaf of the Phrenoesophageal Ligament. This was a well-defined layer in only five of 56 total cases examined. In the remainder, it was represented by a diffuse collection of areolar fibers which pulled away from their insertions with ease. It was absent in seven instances, and so diffuse that its insertion could not be measured in five. In the remaining 44, the average point of insertion of this ligament was 1.4 cm. below the epithelial junction, ranging from 13 instances in which it inserted at the junction to seven cases in which it inserted 3.0 cm. below the junction onto the fundus of the stomach. In no case did this ligament insert above the epithelial junction. In no case was the ligament considered to be of significance as a pressure barrier.

Lower Esophageal Circular Muscle Layer. A specific attempt to identify this layer grossly was made in 33 of the 56 total cases. Twelve of the 33 showed no gross thickening in the circular muscle at the lower end of the esophagus. Twenty-one of the 33 (64%) did show a grossly demonstrable thickening which extended from the point at which the esophagus flared out into the stomach for an average distance

of 4.6 cm. above the epithelial junction. This ranged from 3.0 cm. in three cases to 7.0 cm. in two. This layer did not meet the criteria proposed for a sphincter,[28] but perhaps these criteria should be revised. Microscopic measurements of the thickness of the circular muscle layer were made in 42 of the 56 cases. In these, the circular muscle at the lower end of the esophagus averaged 1.8 times as thick as that higher up. This ratio ranged from 0.9 to 4.0. In all specimens, the microscopic measurements agreed well with the gross observations.

In the majority of patients, the lower portion of the esophagus was found to be in spasm and tightly constricted when removed from the cadaver. In all of these, to avoid the oft-repeated objection that any thickening observed in this region was due to smooth muscle spasm,[28] the cardioesophageal junction was dilated by the insertion of either a finger or a test tube into its lumen. When a tube was used, this was left in the esophageal lumen until the specimen was fixed. In each case, when the specimen was sectioned and examined, the lumen of the lower esophagus was the same diameter as that higher up.

Perhaps this insistence on a dilated lumen accounts for the controversy concerning this area. Certainly the lower esophagus is not dilated in the *in vivo* stage,[19, 39] or if it is, it is regarded as pathologic. In two specimens which were not dilated and which are not included in this series, circular smooth muscle thickening was readily apparent and much more pronounced than any case included here.

Muscularis Mucosa, Submucosa and Mucosa of the Lower Esophagus. Because of sporadic reports in the past that each of these layers served some function in the gastroesophageal closing mechanism,[7, 8] each was examined and measured microscopically in 42 specimens. In 12 (29%) the muscularis mucosa was more prominent in the lower esophagus than in the upper, in two it was more prominent in the upper and in 28 (67%) it was equal. In 26 of these 28, it was of no especial prominence in either layer.

When the combined thickness of all the layers other than the circular muscle was measured microscopically, both in the upper and lower esophagus, the thickness of the lower layers was found to be 1.4 times that of the upper, a difference which we do not regard as significant.

Upper Leaf of the Phrenoesophageal Ligament. This ligament was identified in all cases. In two of the 56 cases, there were two well-defined upper ligaments present, one arising from the endoabdominal or transversalis fascia, and one from the endothoracic fascia. The insertion of the upper-most ligament was taken to be the point of effective insertion of the upper leaf in these two specimens. In one specimen, the ligament inserted diffusely along the lower portion of the esophagus, and the point of primary insertion could not be determined. This specimen was discarded from the series. In the remainder of the cadavers, the point of insertion of the ligament into the esophagus covered no more than 1 cm. in vertical extent on the esophageal wall and, in the majority, it was less than this. In addition to the point of obvious effective insertion of the ligament, a diffuse fibroelastic network of fibers passed from the main, membranous body of the ligament to the sphincter area of the esophagus in all cases. The actual point of insertion of the membrane was determined by placing it on the stretch and then tracing the bundle of fibers which was carrying the most tension to its insertion. Minimal dissection was employed.

The uppermost point of ligamentous insertion was used in the measurements of the distance of the insertion from the epithelial junction. In 22 of 56 cases (39%), the ligament arose exclusively from the endoabdominal fascia. In 24 of the 56

cases, it arose from the endoabdominal fascia primarily but with some minor contribution from the endothoracic fascia. Thus, the ligament arose primarily from the endoabdominal fascia in 46 (82%) of 56 cases examined. In ten of 56 cases, the contribution of the endothoracic fascia was considered to be significant, and in two of these, as mentioned above, there were two ligaments in place of the usually single upper leaf. There was no correlation between the diaphragmatic origin of the phrenoesophageal ligament and pathologic findings. Eight specimens showed evidence of peptic esophagitis—three acute inflammation of the mucosa and submucosa, three frank ulceration and four chronic inflammation and fibrosis. There was some overlap of pathologic findings as previously mentioned. Eight had obvious hiatal herniae, with displacement of the epithelial junction above the diaphragm, an anterolateral projection of peritoneum above the diaphragm, and a variable portion of stomach above the diaphragm as well. Only three with hiatal herniae were among those with peptic esophagitis. Measurements are compared in Table 1.

In the normal patients, the upper leaf of the phrenoesophageal ligament inserted an average of 3.35 cm. above the squamo-columnar epithelial junction. In those patients with peptic esophagitis, the insertion was only 1.13 cm. above this point.

In those patients with hiatal herniae alone, the average point of insertion of the upper ligament was 3.6 cm. and in those hiatal hernia patients who also demonstrated evidence of peptic esophagitis, the insertion was only 0.5 cm. above the epithelial junction. Both differences are statistically significant.

In none of the cases examined with peptic esophagitis was there any significant inflammation found in the tissues surrounding the esophageal hiatus which could have accounted for the displaced insertion of the phrenoesophageal ligament.

TABLE 1. *Relation between Peptic Esophagitis and the Site of Insertion of the Upper Leaf of the Phrenoesophageal Ligament*

	No. Cases	Site*	Range	P
Total cases	55			
Normals	47	3.35 cm.	1.0–8.0	0.002
Peptic esophagitis	8	1.13 cm.	0.0–3.0	
Hiatus hernia only	5	3.6 cm.	2.0–5.0	<0.01
Hiatus hernia with esophagitis	3	0.5 cm.	0.0–1.0	

* Average, above the squamo-columnar junction.

Discussion

According to the concepts proposed by one of us (D. H. D.), when there is a competent lower esophageal sphincter, the presence or absence of gastroesophageal reflux depends on the height of the insertion of the upper limb of the phrenoesophageal ligament into the lower esophageal sphincter area. In our series of peptic esophagitis specimens, the insertion of the upper limb of the phrenoesophageal ligament was indeed lower than the insertion in the normal group of patients, therefore offering support to this hypothesis.

Other investigators have also reported data supporting this concept. Creamer [14] observed that reflux in his series of patients occurred only during inspiration—that is when tension is applied to the phrenoesophageal ligament by the contracting right crus of the diaphragm. Hill and associates [24] observed that when the high pressure zone in their manometric studies is located within the arms of the hiatus, reflux occurs without evidence of hernia formation. Since it is well known that this high pressure zone resides in the lower end of the esophagus and usually extends above the diaphragm, displacement of the zone downward would allow for the phrenoesophageal ligament to be

pulled laterally instead of axially, thereby distracting the sphincter. Of course an incompetent sphincter may also explain this finding. The high incidence of failure to correct reflux reported in patients after an Allison type of repair[38] also supports this hypothesis since one portion of the Allison repair depends on pulling the hernia back into the abdomen by applying tension to the severed edges of the phrenoesophageal ligament. The successful results of simple phrenic nerve section may be attributed to this same mechanism.[21]

Although we have shown that the phrenoesophageal ligament has a low insertion in our series of patients with esophagitis, we have been unable to demonstrate any mechanism to account for this displaced insertion. One possible explanation for this finding in patients with hiatal herniae has been given.[16] When the herniation first occurs and then enlarges, the phrenoesophageal ligament becomes stretched and attenuated. Eventually, it ruptures or somehow is disrupted at its insertion, leaving only those fibers which are normally present between the major body of the ligament and the sphincter area to assume the tension normally applied at the upper end of the sphincter. Inflammation may also account for the displacement of the ligamentous insertion by simply binding the upper limb of the ligament to the esophagus at some point lower than its normal insertion with fibrous adhesions. In at least one experimental series,[5] this has been the mechanism of failure when an attempt was made to relocate the sphincter entirely above the diaphragm. Adhesions formed between the incised phrenoesophageal ligaments and the site of a myotomy on the lower esophagus, effectively reanchoring the lower esophagus in the hiatus. Finally, the displaced insertion of the phrenoesophageal ligament is one of the criteria proposed by Peters[37] for the distinction of a true, congenitally short esophagus.

We have also demonstrated some evidence that there is an anatomical thickening in the circular muscle layer of the lower esophagus. Although this thickening is in the area corresponding to the physiologic sphincter and has been demonstrated before by other authors, we hesitate, in the light of conflicting evidence from still others, to claim a sphincter function for it. Since the existence of at least a physiologic sphincter is so firmly established, the point of whether this circular muscle thickening is its anatomic basis is largely academic.

The phrenoesophageal ligament dilating mechanism has obvious implications with regard to the operative treatment of reflux esophagitis. Any operation which applies circumferential tension around the insertion of the phrenoesophageal ligament may not only *not* cure the reflux, but may aggravate it. The success of operations, such as the Hill median arcuate ligament tether,[24] the Nissen fundoplication,[36] and the Boerema gastropexy,[6] may in part be attributed to their role in reducing the tension on the phrenoesophageal ligament insertion. The Nissen operation also constructs a valve mechanism which should reinforce the sphincter if it is incompetent. This last point is especially important since as yet we have no method of assessing the competence of the sphincter before returning it to the abdomen.

Summary and Conclusions

Based on a study of 227 autopsies of patients with peptic esophagitis, hiatal hernia or both, evidence is presented that the competence of the lower esophageal sphincter mechanism is dependent, in some measure, on the site of insertion of the phrenoesophageal ligament. In the control series of 48 autopsies, the insertion of the phrenoesophageal membrane was 4.4 cm. above the gastroesophageal junc-

tion, compared to 2.1 cm. above this point in the series with peptic esophagitis.

In some patients, there is a demonstrable thickening in the circular layer of smooth muscle at the lower end of the esophagus. This may be the anatomic structure responsible for the manometric sphincter in this area.

The implications of these findings with regard to the operation designed to correct gastroesophageal reflux are discussed.

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Discussion

Dr. Alan Thal (Kansas City, Kans.): I would like to present one case to illustrate the point that the columnar squamous junction is, during life, quite a mobile landmark and very likely shifts during the normal course of deglutition.

(Slide) I am presenting here a case of spontaneous perforation of the eosphagus, showing the perforation well above the diaphragm. (Slide) Here is the biopsy taken from that perforation above the insertion of the phreno-esophageal ligament. The junction of squamous and columnar epithelium is clearly seen. (Slide) I would like you to notice that this is the same patient with a barium swallow in the Tredelenberg position taken several months after the application of a fundic patch. The site of the healed perforation is at this point (indicating) and you will notice the long portion of intra-abdominal esophagus and lower thoracic esophagus. The site of biopsy and hence the squamous columnar junction is well above the phreno-esophageal ligament.

Now, this may have an important implication in understanding the persistence of symptoms after apparent reduction of hiatus hernia. The external landmarks of the esophagogastric junction may be deceptive in indicating the squamous-columnar junction and a protrusion of gastric mucosa may line the lower esophagus.

Dr. Conrad R. Lam (Detroit): The authors are to be commended for their interest in the anatomy of the region of the esophageal hiatus, and they have at least given considerable support to the fact that there is such a thing as the phreno-esophageal ligament.

At the sectional meeting of the American College of Surgeons in Cleveland last week there were at least two members of our panel on this subject who questioned the existence of the phreno-esophageal ligament, and now we learn that there not only is such a thing, but that it has an ascending and an inferior limb.

Obviously, this ligament is not like other ligaments, like the ligaments of the knee joint, but it is at least as much of a ligament as the triangular ligament of the liver or the inferior pulmonary ligament, which, of course, is only two layers of pleura.

In their anatomic studies the authors have noted that in some cases the ligament is inserted lower on the esophagus, and they have proposed the theory that radial tension on this ligament pulls open the sphincter which is doing its best to stay closed. The question which immediately arises is this: Is there really such a tension? And if so, what causes it? Because these studies were done on cadavers and fixed specimens, the actual tension was not demonstrated.

A number of years ago, Dr. Leo Kenney and I made some studies on the tension around the esophageal hiatus, and found that stimulation of the phrenic nerve or cutting of it had no effect on the pressure in the hiatus.

It is conceivable that the intra-abdominal pressure could produce this tension when the patient is in the recumbent position, which, of course, is the time when reflux occurs.

The last question which I should like to ask is: There must have been come clinical histories available on these autopsied patients. Did those in whom they found esophagitis compain of heartburn before they died?

Dr. Stanley R. Friesen (Kansas City, Kans.): Most of the recent studies concerning reflux of gastric juice up into the esophagus have been physiologic studies; and, I think, rightly so, because this is a dynamic area, between the esophagus