

Historic Basis for the Modern Robotic Approach to the Anatomic and Physiologic Reconstruction of Hiatal Hernias

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ABSTRACT

A hiatal hernia is a common clinical entity which has been the subject of great controversy for over a century. During this time, medical practitioners have been like “blind” men who have examined the different parts of an elephant and reached a conclusion based on the partial knowledge but have been unable to recognize the “whole elephant”. The robotic approach to the repair of Hiatal Hernias which not only incorporates the technology of robotics but is designed to return the anatomy and physiology of the hiatus to its normal function, is the culmination of the work of many surgical giants who independently identified specific parts of the “elephant” that is represented by the entity of a Hiatal Hernia. The Robotic Anatomic and Physiologic Repair of the Hiatal Hernia is also the result of a greater understanding of the very complex normal and pathologic physiology of the esophageal hiatus. Investigators from 5 specialties (cardiology, gastroenterology, pulmonary medicine, surgery and radiology), using multiple modalities such as echocardiography, computed tomography, exercise testing, respiratory function testing, have shed new light into the pathophysiology and surgical indications for the repair of Hiatal Hernias. An Understanding of the history of the surgical treatment of Hiatal Hernias sheds light on the controversies and misunderstandings that have characterized the surgical approach to this very important condition.

Keywords: Hiatal Hernia, GERD, Paraesophageal hernia, Robotic Surgery, Laparoscopic Repair, Nissen, Belsey, Gastroesophageal valvuloplasty

For the first half of the Twentieth Century, Hiatal Hernias (HHs) were repaired “Anatomically”. Since the 1950’s until recently, Hiatal hernia surgery evolved from “Anatomic Repair” to “Physiologic Restoration”. The anatomic repair of hiatal hernias was not successful in relieving the symptoms in patients with hiatal hernias and reflux disease. Therefore, with greater understanding of Gastroesophageal Reflux Disease, the pendulum moved toward purely physiologic procedures, which to a large extent, ignored the complex anatomy of the esophageal hiatus and its role in the natural antireflux mechanism. In fact, at one time in the 1970’s, investigators proposed that hiatal hernias were irrelevant, and the answer was in the relief of GERD. Today it is clear that in order to obtain the best results in symptomatic patients, both the

anatomic and physiologic aspects of the complex structure at the esophageal hiatus need to be addressed.

The era which was characterized by the anatomic repair of hiatal hernias was hampered by a lack of understanding of the actual anatomy of the hiatus and the gastroesophageal junction, as well as the shortcomings of the open surgical techniques. It is now clear that the antireflux mechanism is created by the complex anatomy at the esophageal hiatus. Therefore, restoring the complex anatomy of the esophageal hiatus, also restores the antireflux mechanism.

In the past few years, a number of factors have been responsible for a slow but methodical shift back to the correct anatomic repair. These factors have included:

1. Greater understanding of the complex three-dimensional anatomy of the esophageal hiatus.
2. The relationship of the esophageal hiatus to the gastroesophageal antireflux mechanism.
3. The importance of the esophageal hiatus in providing the “skeletal” structure onto which the gastroesophageal valve is suspended.
4. The non-gastrointestinal complications such as cardiac, respiratory and hematologic complications that are associated with hiatal hernias.
5. Change in the definition of symptomatic hiatal hernias.
6. Possibility of complex anatomic reconstruction using minimally invasive techniques which have been brought about from the advances in intraoperative three-dimensional visualization and greater instrument dexterity provided by the robotic platform.

HISTORIC PERSPECTIVE

Anatomic Approach to the repair of Hiatal Hernias: an erroneous extrapolation from the experience with Abdominal Wall Hernias:

Although congenital and posttraumatic diaphragmatic hernias were described as far back as the 16th century by Ambrose Pare (1579), Rivierius Lazari (1689), Giovanni Batista Morgagni (1761), and Vincent Alexander Bochdalek (1848), a Hiatal Hernia (HH) was not recognized as a significant clinical entity until the first half of the 20th century.(1,2)

The first report of a Hiatal Hernia was by Henry Ingersoll Bowditch, who in 1853, in a review of 88 cases with a diaphragmatic hernia which were reported between 1610 and 1846, characterized the postmortem findings in 3 cases where “esophagus presented a very abrupt change of its course. In all, it descended through the diaphragm as usual but turned back toward the left to enter the abnormal aperture caused by the hernia and to join the stomach in the chest.” (3)

The advent of radiography allowed for the antemortem visualization of the Hiatal Hernias. In 1900, Hirsch diagnosed a hiatal hernia by means of x-rays and a mercury-filled balloon prior to autopsy. (1) Four years later, Eppinger diagnosed a Hiatal Hernia in a living patient. (4) In 1911, in a summary of the literature for diaphragmatic hernias, Eppinger identified 635 cases, of which only 11 involved the esophageal hiatus. (5) In 1925, Julius Friedenwald and Maurice Feldman described the symptom of heartburn and related it to the presence of a Hiatal Hernia (HH).(6) They believed that the cause of the hernia was the failure of the muscle of the diaphragm to closely encircle the esophagus. In 1926, Akerlund in Stockholm reported 30 more cases of esophageal hiatal diaphragmatic defect, and for the first time proposed the term “hiatus

hernia” and classified hiatal hernias into 3 types. (7) Curiously this is the same classification that is used today! He also noted that patients with HH complained of pain immediately after ingestion of food, sometimes described “heartburn”, and sometimes complained of dysphagia. He noted that “a diaphragmatic hernia through the esophageal hiatus may properly be termed a Hiatus Hernia. They are most often true nontraumatic hernias and can be classified in 3 groups: a) hiatus hernias with congenitally shortened esophagus (thoracic stomach), b) paraesophageal hernias, c) hernias not included in a and b.” In 1930, Max Ritvo, a Boston radiologist, published a series of 60 cases with hiatal hernias.(8) Ritvo stated that the cause of the “acquired esophageal orifice hernia” is the increased intraabdominal tension, which can be caused by conditions such as constipation, pregnancy and obesity. He also reported epigastric pain, heartburn, nausea, vomiting, and regurgitation as clinical correlates of hiatal hernia. Despite these reports, the acceptance of Hiatal Hernia (HH) as a distinct entity was not universal, with such giants as Kirklin and Sauerbruch considering the presence of Hiatal Hernias on radiographic studies as an artifact. (9,10)

With the use of the term, “Hernia”, and the importance of inguinal hernia repair techniques in the surgical practice of the early twentieth century, surgeons began treating esophageal hiatal defects as they would “an abdominal wall hernia”. In 1919, Angelo Soresi reported the first surgical approach to the repair of hiatal hernias which consisted of reduction of the hernia and closure of the opening of the diaphragm. Interestingly he wrote: “patients suffering from this condition are not properly treated.... This lack of interest is not easily explained, because diaphragmatic hernias give rise to so many complicated and serious symptoms, which if not properly attended to, will lead the patient to an unfortunate life and premature death”. (11) In 1928, Stuart Harrington published the Mayo Clinic experience treating 27 patients using the technique of the closure of the esophageal hiatus as described by Soresi. (12,13) Harrington emphasized that: “closure of the hernia opening is essential for the relief of symptoms.” When he was not able to close the diaphragm, he sutured the herniated viscera to the edges of the hiatus (gastropexy), a procedure that he called “palliative.” He recognized that such a procedure would not correct the problem, but it was the only possible action given the techniques of the time. It is curious that many modern surgeons continue to perform “gastropexy”. He also introduced “phrenic neurectomy” via a cervical incision as an adjunct in cases of large hiatal hernias where the hiatus was difficult to close. The transection of the phrenic nerve was designed to result in a flaccid hemidiaphragm and allow for a tension-free closure of the hiatus. Unfortunately, the surgeons of this era did not appreciate the importance of freeing the esophagus and complete dissection of the gastroesophageal junction. Using the rudimentary x-ray imaging modalities of the 1930’s, they reported the recurrence rate with this technique to be 12.5%. Paralyzing the left hemidiaphragm was a technique which was rooted in the treatment of inguinal hernias, where tension-free repair was obtained by various “relaxing” procedures and was meant as a procedure to decrease the tension on the suture line. After two decades, the technique of phrenic neurectomy was abandoned due to the unpredictable results, complications from unilateral diaphragmatic paralysis, and the shift from anatomic repair to functional repair of hiatal hernias in the 1950’s. Richard Sweet from Massachusetts General Hospital first reported the application of this technique to a transthoracic versus transabdominal repair of the esophageal hiatal hernias.(14,15) He too applied the principles developed in the treatment of inguinal hernias, by reducing the hernia, crushing the phrenic nerve, and plicating the hernia sac. He then narrowed the hiatus with heavy silk sutures until he could get his index finger between the esophagus and the rim of the hiatus. Furthermore,

Sweet suggested that in some cases, Fascia Lata obtained from the left thigh would be used to reinforce the repair. Interestingly, in cases where the stomach could not be reduced under the diaphragm, he suggested that no attempt to alter the location of the cardia and stomach needed to be made. This erroneous concept has been carried until the modern age, as many surgeons still believe that an incomplete reduction of the entire hernia sac is acceptable.

It is important to note that the inguinal and other abdominal wall hernias were a significant subject of surgical research and practice in the first half of the twentieth century. Therefore, it is not surprising that a defect in the diaphragm at the esophageal hiatus was called a “Hiatal Hernia” instead of a “Hiatal Defect”, and by extension, surgical repair was to follow the, albeit erroneous, concepts which developed for other abdominal wall hernias. Consequently, at this time, surgeons focused on correcting an anatomic defect. Many surgeons believed that symptoms in patients with hiatal hernias emanated from pinching of the stomach as it traversed the hiatus. To their dismay, many patients had successful restoration of anatomy, but the symptoms continued to persist. In turn, in the face of persistent symptoms, surgeons were inclined to blame the original repair. This approach had two shortcomings: 1. Until the 1950’s there was not an appreciation for the link between hiatal hernias and gastroesophageal reflux disease, and 2. The technology of the time did not allow for a clear understanding of the complex three dimensional anatomy of the esophageal hiatus, its relationship with the gastroesophageal junction, and the varied pathologic anatomic changes that encompass the diagnosis of a Hiatal Hernia.

Functional Repair of Hiatal Defects and the Concept of Gastroesophageal Reflux

The 1950’s witnessed a shift from the focus on the anatomy of hiatal defects as a “hernia” to the functional physiologically based disorder of reflux esophagitis.

In 1951, Allison of Leeds attributed reflux esophagitis to incompetence of the gastroesophageal junction and he stated that “the cause of the incompetence is a sliding hernia of the stomach through the esophageal hiatus of the diaphragm into the posterior mediastinum.” (16) Most importantly, although it has not been recognized widely, Allison classified Hiatal Hernias (HHs) into 2 types: the sliding hernia and the paraoesophageal or rolling, and he observed that these 2 types give rise to different symptoms and had a different prognosis. Allison focused on the crural sling as the key factor in preventing reflux. He believed that these crural fibers functioned as a “pinchcock” to prevent reflux. The Allison procedure consisted of a transthoracic reduction of the herniated cardia back into the abdomen, approximation of the crural fibers posterior to the esophagus, and retention of the cardia to that position by suturing the phrenoesophageal ligament and peritoneum to the abdominal aspect of the diaphragm. At that time there was no knowledge of the gastroesophageal valve. Therefore the “Allison operation” repaired the gastroesophageal junction without creating an antireflux mechanism. It not surprising that after a long term follow up, he reported a 49% gastroesophageal reflux disease in 421 operated patients. (17)

Although Norman Barrett became more known for the condition of intestinal metaplasia that carries his name, he was the first investigator to postulate that there was a fold of mucosa at the gastroesophageal junction that acted as a functional flap valve. (18) This is especially remarkable as it predates the advent of endoscopy or any other means of intraluminal visualization of the gastroesophageal junction. As a result, he concentrated on restoring the

cardio-phrenic angle. Barrett's surgical approach can be summarized in his own words: "I believe that the hernia should be reduced because its presence permits reflux; the esophageal hiatus may sometimes require diminishing in size in the hopes that this maneuver will help to prevent a recurrence of the hernia; the esophagogastric angle should be reconstituted by fixing the cardia below the diaphragm and so allowing the fundus of the stomach to balloon up under the dome." Although instead of focusing on the reduction of the hiatal hernia and the crural sling as with the Allison Procedure, Barrett's procedure emphasized the esophagogastric junction, he was still unable to recreate the hypothesized valve by simply fixing the cardia below the diaphragm.

During this period there was great controversy as to the presence or absence of a physical barrier to gastro-esophageal reflux at the gastro-esophageal (GE) GE junction. As far back as 1902, the Radiologist Walter B. Cannon had observed two physiological phenomena during radiographic examination of the GE junction with barium: first, that there is a mechanism that prevents occurrence of reflux across the gastroesophageal junction and, secondly, that this mechanism allows brief episodes of reflux.(19) Since he could see a negative shadow at the GE junction on the radiographs, Cannon concluded, albeit erroneously, that the "cardiac sphincter" (later called the lower esophageal sphincter, LES) was a thickened band of circular smooth muscle, which he interpreted as being the mechanism that prevented reflux of gastric material back into the esophagus. However, anatomic studies of the GE junction failed to show the existence of a muscular sphincter similar to what was seen and described at the upper esophageal sphincter. Amazingly, long before a "physiologic" antireflux mechanism was recognized, Norman Barrett, denied the presence of a sphincter and supported the concept that the anatomic relationships at the esophageal hiatus accounted for the antireflux mechanism.

The controversy became more complex when in the late 1950's, Charles Code of the Mayo Clinic used an intraluminal pressure transducer and miniature balloon manometry to demonstrate that there was an intraluminal high pressure zone (HPZ) interposed between the stomach and the esophagus in humans.(20-22) The results of these findings were unfortunately erroneously interpreted as evidence for the presence of intrinsic muscles of the distal esophagus which were responsible for maintaining this pressure. (23-31) To the present day, many assume that the presence of a High Pressure Zone (HPZ) is synonymous with the presence of a Lower Esophageal Sphincter (LES), and unfortunately, and erroneously, these terms are used interchangeably.

The discovery of a High Pressure Zone at the GE junction gave rise to an "Alternate" approach to hiatal hernias. This approach emphasized a Physiologic Approach to the repair of Hiatal Hernias.

In December 1955, Rudolf Nissen of Switzerland reported an operation on a 49-year-old woman with a long history of gastroesophageal reflux disease where he enveloped the lower esophagus with the gastric fundus by suture approximation of the anterior and posterior fundal folds anterior to the esophagus. (32) This technique had been born from a technique which he had used 20 years earlier to manage a distal esophageal ulcer. (33) The history of the events that led to this landmark procedure is significant. Rudolf Nissen was a student of Professor Sauerbruch in Munich and later his assistant in Berlin. In 1933, Nissen was appointed as the Professor and Chief of the Department of Surgery at the University of Istanbul in Turkey. While

in Istanbul, in 1936, he had treated a 28-year-old man with a distal esophageal ulcer which had penetrated into the pericardium. In an effort to protect the anastomotic suture line between the esophagus and the stomach, he folded over the stomach wrapped the anastomosis with the stomach as a “Witzel” gastric tube in order to get serosal apposition. (34) Nissen left Istanbul in 1939 and after spending 13 years in the United States, became the Professor of Surgery at the University of Basel in Switzerland in 1952. Sixteen years after he has performed the procedure on the Turkish patient with the esophageal ulcer, Nissen re-examined the patient and, although he expected the patient to have reflux resulting from the original procedure, to his great surprise, there was no evidence of esophagitis and the patient had no symptoms of reflux. Nissen, who was a firm proponent of Allison’s concepts of gastroesophageal reflux disease but was unhappy with the results of the Allison Operation, applied the concept of a fundal wrap in 1955 to the treatment of gastroesophageal reflux disease.

On that day in December 1955 when confronted by a 49 old woman with a three-year history of reflux esophagitis, through a transabdominal approach Nissen divided the phrenoesophageal ligament and mobilized the esophagus. He did not divide the short gastric vessels. Using his right hand, he then passed the gastric fundus behind the stomach through an opening provided by the divided gastro-hepatic ligament and wrapped the distal 6 cm of the esophagus with the gastric fundus. During this procedure, the short gastric vessels were not divided, and the stomach was wrapped around the esophagus which had a large indwelling stent. He used 4 interrupted sutures, one of which also incorporated part of the anterior wall of the esophagus. The wrap was performed around a large-bore indwelling intra-esophageal stent. A gastropexy was added. The concept of adding a gastropexy emanated from a single case that Nissen had performed while in the US. In 1946, Nissen performed a laparotomy and anterior gastropexy on his colleague Gustav Bucky to reduce a hiatal hernia. Bucky remained symptom-free throughout his 15-year follow-up, and Nissen attributed the success of the gastropexy to the accentuation of the esophagogastric angle achieved by anchoring of the stomach to the hiatus. The clinical outcome of the first Nissen Fundoplication in 1955 was excellent, and was reproduced in a subsequent patient. These 2 cases were published in 1956, and Nissen named the operation “*gastroplication*” (35-37) The original Nissen procedure subsequently underwent modifications by Nissen and other investigators. Consequently, “Nissen fundoplication” refers to many different procedures with the unifying theme of a full gastric fundal wrap. By 1962, gastropexy portion of the operation was omitted. In a modification described by Rossetti, only the anterior wall of the stomach was used to encircle the distal esophagus. Later, in an attempt to decrease the complications from a tight wrap and the resultant dysphagia, “gas bloat” syndrome, and wrap disruption, a number of modifications were made. These have included the placement of intra-esophageal stents varying from 46 Fr. to 60 Fr., division of short gastric vessels in order to obtain a floppy wrap, closing the esophageal hiatus posteriorly, anchoring the fundoplication to the preaortic fascia, decreasing the length of the wrap to 1–2 cm, concomitant highly selective vagotomy, and design of wraps that encompass less than 360 degrees of the circumference of the esophagus. (38-45)

By contrast to the serendipitous observations resulting in Nissen’s fundoplication, Mr. Ronald Belsey’s approach to the treatment of GERD was the culmination of years of careful observation and follow-up in the clinic. Mr. Belsey, a British Surgeon at Frenchay Hospital, has been quoted as saying “The battlefields of surgery are strewn with the remains of promising new operations which perished in the follow-up clinic.” (46) His scepticism stemmed from the failures of the

anatomic repairs of hiatal hernias. In 1942, Belsey set out to develop a physiologic rather than an anatomic repair. His procedures were based on endoscopic observations using a 50 cm rigid esophagoscope. Patients underwent endoscopy under sedation in a seated position. He observed that the competence of the esophagogastric junction depended on its position below the diaphragm. If the gastroesophageal junction was displaced above the hiatus, the esophagogastric opening was seen to gape and allow a tide of gastric contents to flow out of the stomach. He referred to this condition as a "patulous cardia." The goal of his surgical procedure was to reposition the esophagogastric junction several centimeters below the diaphragmatic esophageal hiatus. The "Belsey Mark I" operation was essentially a variant of the Allison procedure. During this procedure, Belsey observed that the "naked" esophagus which lacked a serosal cover was difficult to anchor to the underside of the diaphragm. Consequently, the "Belsey Mark II" and "Mark III" procedures represented attempts at providing the esophagus with a serosal covered muscular collar of the stomach which would enable the placement of anchoring sutures to the underside of the diaphragm. Furthermore, these procedures were designed to restrain the intraabdominal segment of the esophagus from dilating. It is important to note that at no time was Belsey trying to fashion a gastroesophageal valve. However, through the application of the "scientific method" with intervention, observation, and close follow-up, the "Belsey Mark IV" procedure serendipitously created a partial gastroesophageal valve. Importantly, although today we appreciate that the "Mark IV" procedure produces a partial gastroesophageal valve by intussuscepting the esophagus anteriorly into the stomach for the span of 240 degrees of the esophageal circumference, Belsey's creation of the intussusception was designed to allow for the secure placement of sutures from the gastroesophageal junction to the underside of the diaphragm. Belsey studied the original group of 71 patients for six years before publishing the results of his procedure. Belsey held that a good antireflux procedure met the following criteria:

- It would achieve relief of reflux
- It would preserve swallowing, venting of gas, and the ability to regurgitate.
- It would be easy to teach to other surgeons.

Between 1955 and 1962, 632 patients underwent the Belsey Mark IV procedure at his clinic. He reported an overall good to excellent result of 85%. Skinner showed similar long-term results with the "Belsey Mark IV" repair in over 1000 patients. (47)

Another pioneer in the surgical repair of HHs, Lucius Hill, concentrated on studying the antireflux mechanism by use of manometry and pH testing, and applied these test to a cadaver model of HH and Gastroesophageal Reflux Disease (GERD).(48-51) He famously proclaimed that "current repair of hiatal hernia is in about the same state as repair of inguinal hernia at the time of Bassini and Halsted in 1888. Their recurrence rates for inguinal hernia were lower in 1888 than they are for hiatal hernia in current documented reports."(48) Hill's studies led to the discovery of the flap valve mechanism, "the Gastroesophageal valve" (GEV) as opposed to a "sphincter" at the GE junction. Furthermore, he proposed a grading system of the GEV that correlated with the patient's reflux status better than the measurement of the lower esophageal sphincter pressure alone. (50,51) Finally he concentrated on fixing the GE junction to the esophageal crus which became the cornerstone of the posterior gastropexy or the "Hill Repair". (49)

For the next 30 years, the open transabdominal Nissen fundoplication and the transthoracic Belsey repair were the mainstay of surgical therapy for HHs and gastroesophageal reflux disease. However, during the 1980s with the advent of better medical therapy, surgery was no longer the primary therapy for GERD, and in time, surgery was less commonly advised.

Throughout the twentieth century, the relationship between HHs and GERD remained an enigma and subject of great controversy. As the result of the development of the Nissen and Belsey procedures, greater emphasis on gastroesophageal reflux disease, and the high recurrence rates for purely anatomic repairs of the esophageal hiatus, repair of HHs became an afterthought to the fundoplication. In the latter part of the twentieth century, the key to the repair of hiatal hernias was thought to be the Fundoplication and not the Hiatal Reconstruction.

THE CONCEPT OF SHORT ESOPHAGUS

In larger hiatal hernias, there was the problem of the inability to reduce the hernia sac below the diaphragm. Surgeons had the option of a Left Transthoracic Approach, an Open Abdominal Approach and in later years a Laparoscopic Approach. Each one of these approaches had specific shortcoming when it came to the complete dissection of the hernia and reduction of the contents below the diaphragm. The left transthoracic approach was hampered by the morbidity of a thoracotomy. The abdominal approaches, although associated with lesser morbidity, did not allow for full visualization and accurate dissection in the posterior mediastinum above the diaphragm. The final outcome was that surgeons settled for the procedure with lower morbidity and resolved themselves to settle for a less than optimal dissection and reconstruction of the hiatus.

In 1957 in order to overcome the problem of an irreducible hiatal hernia, which was erroneously attributed to a “short esophagus”, J. Leigh Collis reported a procedure for lengthening the esophagus by forming a tube out of the proximal stomach. (52) Using this technique many surgeons reported significant early and late complications, some resulting from the creation of an iatrogenic Barrett's esophagus. Later the Collis Gastroplasty was combined With a Belsey procedure or a Nissen Fundoplication in patients in whom the hiatal hernia could not be completely reduced and the GE junction could not be placed below the diaphragm. When combined with an antireflux procedure, the Collis Gastroplasty had excellent results. (53,54)

A variant of the Collis Gastroplasty, wedge gastroplasty, has found its way into the modern laparoscopic surgical repair of hiatal hernias. As with the Collis Gastroplasty, this procedure represents an indirect measure designed to compensate for the inability to fully dissect the hernia sac and mobilize the esophagus by laparoscopic techniques.

It is important to note that the condition of an esophagus shortened by severe inflammatory changes from wide open reflux is rare and is even more rarely seen in the era of medical acid suppression therapy. Furthermore, in patients with giant hiatal hernias in whom the hiatal sac cannot be fully reduced and the GE junction be placed under the diaphragm, endoscopic measurement of the esophageal length and the distance of the GE junction from the incisors is similar to normal individuals. These observations have led to the conclusion that a “short esophagus” is the result of the inability to fully reduce the hiatal hernia and place the GE below the diaphragm. A more direct approach to an irreducible hiatal hernia is a more extensive

dissection of the mediastinal extension of the hiatal hernia which has been the shortcoming of the conventional surgical approaches.

The Role of the Esophageal Hiatus in the Gastroesophageal Antireflux Mechanism

What is the anatomic counterpart to the high-pressure zone (HPZ) seen on manometry?

Is there an anatomic sphincter or a functional valve at the gastroesophageal junction?

What is the relationship of the Gastroesophageal Valve to the Esophageal Hiatus?

After the description of the distal esophageal high-pressure zone (HPZ) on manometry by Fyke in 1956, all attention was focused on the HPZ. Since there was a high-pressure zone in the upper esophagus corresponding to the cricopharyngius muscle or the upper esophageal sphincter, the distal HPZ was referred to as a lower esophageal sphincter (LES). This resulted in the erroneous conclusion that there was a circular sphincter muscle at the distal esophagus which was comparable to that of an upper esophageal sphincter. However, anatomic dissections of the gastroesophageal junction failed to reveal such a sphincter. A study in 1987 by Thor, et al, clarified the presence of a functional valve at the gastroesophageal junction. (55) In this study, the stomachs of 33 cadavers without an antemortem history of reflux were distended with water. Reflux of the water from the stomach only occurred when intragastric pressure reached a pressure of 15 cm of water. Clearly a sphincter mechanism would not be functional in the postmortem state. These authors concluded that there is a functional valve which is created by the three-dimensional relationship which exists between the esophagus, the stomach, and the diaphragmatic crural sling at the level of the esophageal hiatus. The three-dimensional relationship accounts for the HPZ on manometry and represents a significant part of the antireflux barrier. In a porcine model of gastroesophageal reflux disease, Gharagozloo et. al. replicated the findings of Thor. (56) Furthermore, they showed that the creation of a hiatal hernia resulted in free reflux of gastric content at much lower intragastric pressures.

In cadaver and clinical experiments, Hill and associates have identified a gastroesophageal valve which is a musculo-mucosal fold created by the intussusception of the esophagus into the stomach spanning from the right limb of the crus to the left limb of the crus anteriorly 240 degrees of the circumference of the gastroesophageal junction. (57) The valve can be visualized by the retroflexion of the gastroscope and cephalad visualization of the gastroesophageal junction during endoscopy. Furthermore, these investigators have described a grading system for the gastroesophageal valve.

- Grade I: The gastroesophageal valve (GEV) is normal, where the musculo-mucosal fold that hugs the scope through all phases of respiration, opens during swallowing, and then closes promptly without allowing reflux.
- Grade II: GEV is a less defined fold, slightly shorter, which opens during swallowing and thereafter closes promptly.
- Grade III: GEV opens frequently, remains open for varying lengths of time, and may be associated with an intermittent hiatal hernia.
- Grade IV: No GEV is evidenced. Gastroesophageal junction is wide open and is always associated with a hiatal hernia.

Hansdotter and coworkers have used this grading system as a useful predictor of the presence of gastroesophageal reflux disease. (58)

Based on these studies and observations, the antireflux barrier appears to be the result of the intussusception of the esophagus into the stomach by 2 cm anteriorly spanning 240 degrees of the circumference of the gastroesophageal junction. The intussusception of the esophagus enters at an acute angle and is posteriorly angulated. This complex three-dimensional relationship is held in place and is suspended onto the esophageal hiatus. GERD would occur continuously without an antireflux barrier. Mittal and Balaban have described two major elements in the normal antireflux barrier [59]:

- The lower esophageal high-pressure zone which corresponds to the gastroesophageal valve
- The diaphragmatic crural arch

It appears that the relationship of the GE Valve to the Esophageal Hiatus is similar to the relationship of the Mitral valve to the Mitral Annulus. Dysfunction of the Mitral valve and dilation of the annulus result in Mitral regurgitation which is analogous to the regurgitation at the GE junction which is commonly referred to as reflux. The Mitral Valve is suspended onto the framework which is represented by the mitral annulus. The Mitral valve cannot function appropriately with a dilated annulus. A dilated mitral annulus results in valvular regurgitation. Furthermore, treatment of mitral regurgitation by repairing the mitral valve needs to be accompanied by mitral annuloplasty and suspension of the valve onto the annulus. By extrapolation, our present understanding of the Antireflux mechanism at the GE junction leads to the conclusion that treatment of GE regurgitation or Reflux needs to encompass two components: 1. Repair of the GE Valve and 2. Suspension of the GE Valve onto a repaired esophageal hiatus. These two components, in turn, would recreate the normal antireflux mechanism.

The complex three-dimensional relationship of the entry of the esophagus into the stomach is disrupted by widening of the external crural arch and the occurrence of a hiatal hernia. It is postulated that with a hiatal hernia, there is progressive enlargement of the crural arch anteriorly with stretching and redundancy of the phrenoesophageal ligament. As a result, first, the acute angulation of the gastroesophageal junction is straightened. Subsequently, with further enlargement of the crural arch and the hiatal hernia, there is greater outward traction which results in the reduction of the esophageal intussusception out of the stomach. With a reduction of the esophagogastric intussusception, the functional gastroesophageal valve is disrupted, thereby resulting in gastroesophageal reflux.

Hiatal Hernias

It is estimated that Hiatal Hernias (HHs) affect approximately 20% of the population. The incidence of HHs is 37% in patients with Morbid Obesity, defined by BMI > 43kg/m². Furthermore, 37% of HHs are defined as “Large” or >4cm on endoscopy.

Classification

Classically, hiatal hernias have been classified in two ways:

1) Type I-IV. This classification is based on the position of the gastroesophageal junction relative to the diaphragm.

Type I, commonly but erroneously known exclusively as a sliding hiatal hernia, is the most common and occurs when the phrenoesophageal ligament or peritoneum is displaced superiorly into the thoracic cavity. This anatomic configuration was originally called a “Sliding” hiatal hernia in the early part of the twentieth century, as the esophagus, a viscous, occupied

the posterior aspect of the hernia sac, and the hernia sac was confined to the anterior aspect of the defect. In the original description and classification of hiatal Hernias, this type of HH was thought to be analogous to a "Sliding" inguinal hernia where the posterior aspect of the hernia sac is occupied by the cecum. Contrary to the popular misunderstanding which has continued to the present, a "Sliding" HH does not mean that the GE junctions slides or moves up and down through the diaphragmatic hiatus.

Type II, also called "rolling" PEH, occurs when the stomach migrates into the chest and "rolls" over the esophagus with the gastroesophageal junction still laying down into the abdomen.

Type III occurs when the stomach migrates into the chest and "rolls" over the esophagus with a concomitant migration of the gastroesophageal junction into the chest.

Type IV occurs when, together with the stomach, there is herniation of other intra-abdominal contents through the hiatus (e.g., small bowel, colon, duodenum, or pancreas. (60-66)

This classification dates back to 1927 and does not have a relevance in terms of clinical decision making in patients with HH's.

2) Classification based on Clinical Presentation: Gharagozloo et. al have proposed a Classification of Hiatal Hernias based on the Clinical Stages, Stage I, Stage II. This classification proposes that Hiatal Hernias represent a spectrum of disease and therefore all Hiatal Hernias are Sliding Hernias with different amounts of intra-abdominal structures which have migrated into the hernia sac. This classification is more relevant in terms of surgical decision making. Based on this classification, HH represents a spectrum from a patulous cardia to increasingly enlarging hernias which culminate with an intrathoracic "upside-down" stomach and incarceration.

The first classification has emphasized the content of the hiatal hernia, and the latter classification focuses on the spectrum of changes which occur at the esophageal hiatus. Advent of laparoscopy has facilitated a greater understanding of the complex three-dimensional anatomy of the esophageal hiatus. Based on our observations, we subscribe to the latter classification of hiatal hernias. Different hiatal hernias represent a spectrum of defects that are associated with enlargement of the esophageal hiatus and attenuation of the diaphragmatic muscle. The right and left attachments of the right diaphragmatic crus originate from the vertebral bodies and form a crural sling around the esophagus. The enlargement of the esophageal hiatus is characterized by enlargement anteriorly of the crural sling and the preservation of the posterior portion of the esophageal hiatus. It is due to this phenomenon that the anterior phrenoesophageal ligament (peritoneal reflection) is stretched only on the anterior portion of the gastroesophageal junction from the right side to the left side of the crural sling (270 degrees). As a result, the anterior portion of the stomach and the anterior portion of the gastroesophageal junction is displaced above the diaphragm, thereby giving rise to a sliding hiatal hernia. Once again, a sliding hiatal hernia refers to the fact that the anterior portion of the hernia had a peritoneal sac and the posterior portion of the hernia is made up of the esophagus. As the hernia enlarges, progressively more and more of the stomach is displaced only anteriorly into the hernia sac (comparable to type II and III hiatal hernias). Finally, significant enlargement of the crural arch results in attenuation of the crural muscle fibers, stretching of the

diaphragmatic hiatus, and, once again, anterior migration of the stomach and other abdominal contents above the hiatus (comparable to type IV hiatal hernia). All hiatal hernias represent a spectrum from a small sliding hiatal hernia to a large hernia. All hiatal hernias result from the enlargement of the crural sling.

Finally, with the present understanding of the pathophysiology of HH's in the twenty first century it may be more accurate to refer to this condition as a "Hiatal Defect".

PATHOPHYSIOLOGY AND CLINICAL PRESENTATION

Clinical Presentation of Hiatal Hernias represents a continuum which correlates with whether the stomach or other intraabdominal tissues (a paraesophageal component) have entered into the hernia sac. The classification of HHs into Clinical Stage I and Clinical Stage II clinical presentation based on the anatomic and physiologic changes which occur in the hiatus, is more relevant for clinical decision making and indications for surgical intervention.

Clinical Stage I- Gastroesophageal Reflux Disease GERD

Patients with Small Hiatal Defects typically present with GERD symptoms. To reiterate, the antireflux barrier is the result of the intussusception of the esophagus into the stomach by 2 cm anteriorly spanning 240 degrees of the circumference of the gastroesophageal junction. This intussusception creates a three-dimensional 2cm horseshoe shaped fold, or the GE Valve, which functions like a "trapdoor". This complex three-dimensional relationship is held in place and is suspended onto the esophageal hiatus. The pathophysiology of a small HH is such that the anterior aspect of the hiatus enlarges or stretches, and the phrenoesophageal ligament migrates superiorly through the hiatus. As the phrenoesophageal ligament inserts onto the esophagus, its migration gradually pulls the esophagus out of its intussusception in the stomach. Once 2cm of the esophagus has been pulled out, the GE Valve becomes incompetent, resulting in reflux. (Fig. 6) Therefore, GERD requires a hiatal defect a of at least 2cm. This finding was described by Gharagozloo, et. al. in a prospective double-blind study of laparoscopic diagnosis of radiologically and endoscopically undiagnosed small HH's in patients with symptomatic GERD. (67)

Stage II- Gastrointestinal, Aerodigestive, Pulmonary, Cardiovascular, and Gastric, and Hematologic Symptoms

With an enlarging HH, the stomach begins to migrate through the hiatus and into the chest. While this is still a "Sliding" HH, the migration of the stomach through the hiatus results in varying degree of distal esophageal obstruction. The greater migration of the stomach through the hiatus, correlates with a greater degree of obstruction. The anteriorly herniated fundus predisposes the stomach to twist onto itself. In addition, depending on the patient's BMI and the amount of intraperitoneal and retroperitoneal fatty tissue, varying amounts of fat will also migrate through the hiatus. The fat within the hernia sac is usually omentum on the greater curve and the left lateral aspect of the esophagus, and the fatty tissue of the gastro-hepatic ligament on the lesser curve or the right lateral aspect of the esophagus. The fatty tissue in the retroperitoneum will migrate through the hiatus outside the hernia sac in a retroperitoneal and paraesophageal configuration. Consequently, the gastrointestinal symptoms in Stage II which result with a paraesophageal component in the HH, are predominantly mechanical, including esophageal and gastric obstruction, strangulation, incarceration, and ulceration. (68-71)

Esophageal Symptoms: Obstruction at the distal esophagus results in a feeling of early satiety after oral intake, and dysphagia which is described by the patients as the feeling that the “food is sticking or getting hung up at the lower esophagus or even the throat”. The stagnation of the food is associated with bacterial decomposition of the food in the moist and dark environment that is presented by the esophagus. With bacterial degradation of the food, over a period of time the food softens, liquifies, and passes into the stomach. The bacterial fluid in the esophagus has been shown to result in esophagitis and a sensation of “heartburn”. Although culture-based studies have suggested that the esophagus is either sterile or contains only few transient bacteria, in situ staining revealed association of bacteria with the esophageal epithelial cell surfaces, suggesting the presence of residential bacteria in the distal esophagus. (72-74) In addition, it has been demonstrated that esophageal bacterial composition differs under conditions of normal esophagus, reflux esophagitis, and Barrett's esophagus. Consequently, diverse bacterial communities may be associated with obstructive esophageal disease. (75,76) As the patient moves from Stage I to Stage II of the disease, pharmacologic acid suppression therapy becomes more ineffective. The patient continues to report a feeling of “heartburn”, however, in Stage II, heartburn and esophagitis are no longer caused by GERD and acid reflux. In the absence of the appropriate testing to document the progression from Stage I to Stage II of the disease, historically patients have been treated with increasing stronger acid suppression therapy with very little clinical effect.

Upper Aero-digestive symptoms: Aside from a feeling of “heartburn”, in Stage II, patients report symptoms that in the past have been attributed to “Laryngopharyngeal Reflux” or LPR. LPR is associated with symptoms of laryngeal irritation such as throat clearing, coughing, and hoarseness. In addition, patients may complain of sinus infections and other conditions affecting the upper aero-digestive system. Studies have failed to show acid reflux as the cause of LPR. (77) On the other hand, Impedance monitoring has detected episodes of non-acid or weakly acid gastric reflux in symptomatic patients, suggesting that non-acid components of the esophageal refluxate are responsible for the mucosal damage.(78) It is hypothesized that LPR is the result of the repeated reflux of the infected esophageal fluid into the upper aerodigestive tract in patient in Stage II of HH. The “esophageal Reflux” is exacerbated when the patient assumes a supine position.

Pulmonary Symptoms: The pulmonary symptoms include, shortness of breath, wheezing, and aspiration pneumonia. Wheezing and aspiration pneumonia result from aspiration of the esophageal fluid into the airway and the lungs. Historically, shortness of breath in patients with large HHs has been attributed to “thoracic displacement”. It has been erroneously suggested that dyspnea in HHs is predominantly due to a mechanical respiratory effect of a large space-occupying intrathoracic mass. Furthermore, historically, and erroneously, explanations for dyspnea in patients with large hiatal hernias have included disturbances of respiratory function, diaphragmatic dysmotility, and disturbances of ventilation and perfusion, and asthma caused by esophageal reflux (79). However, in these patients spirometry has not correlated with to the level of functional compromise.(80,81) Low and Simchuk showed only mild abnormalities of spirometry were identified (FEV1 [percentage predicted] and FVC [percentage predicted], 76% and 79%, respectively), despite moderately severe symptoms. In addition, after surgical repair of the HH, even though dyspnea completely resolved in most of their patients, there was only a mild improvement in spirometric values (absolute increase in both FEV1 [percentage predicted] and FVC [percentage predicted] of 13%).(82) Greater experience

and direct in situ observation of the effect of the contents of the hiatal hernia have also refuted the concept that the pulmonary symptoms in patients with HHs are the result of lung compression. The Hiatal Hernia extends into the posterior mediastinum and has very little direct displacement effect on the lungs.

“Asthma” in patients with large HH’s appears to be the result of inflammation of the airway from aspiration of esophageal refluxate. Shortness of breath and other pulmonary symptoms appears to be more related to the effect of the Hiatal Hernia on the cardiovascular system.

Cardiovascular Symptoms: In Stage II, large Hiatal Hernias can lead to chest pain and dyspnea, and at times result in pulmonary edema and cardiac failure. Siu et al. reported that a large hiatal hernia caused cardiac failure by the compression to the left atrium in a case presenting with recurrent acute heart failure.(82) Chau et al. demonstrated a large hiatal hernia as the cause of chest pain in patients that presented to emergency department with acute angina.(83) A hiatal hernia can cause pulmonary edema and cardiac failure through pulmonary venous obstruction. (84,85) Noam et. al. prospectively studied patients using resting and stress echocardiography, cardiac computed tomography, and respiratory function testing before and after repair of large hiatal hernias. Preoperatively, despite the presence of normal pulmonary function, 83% of these patients had exertional dyspnea, and this problem improved after surgery. Moderate to severe left atrial compression was present in 77%, and this correlated with the degree of functional impairment. The improvement of functional class and exercise capacity after surgery was associated with resolution of cardiac compression. Indeed, the change of left atrial diameter on echocardiography was the only independent correlate of the improvement in exercise capacity after surgery. (86) In addition, the results of this study provide evidence of left atrial, pulmonary venous, and coronary sinus compression by large hiatal hernias. Surgical repair of the HH results in improvement of left ventricular and left atrial dimensions, as well as a normalization of atrial inflow velocities.

Left atrial (LA) compression may cause dyspnea by increasing the pulmonary venous pressure, producing interstitial edema and reducing pulmonary compliance. Previous case reports describing cardiac failure and dyspnea attributable to LA compression by HHs support this hypothesis. (87,88) In patients with large HHs, Echocardiography demonstrates pulmonary vein compression and increased systolic and diastolic components of the pulse-wave Doppler signal at the pulmonary vein ostium. In addition, in patients with a large HH, there is increased velocities at the LA inflow, which resolve after surgery.

Extrinsic cardiac compression also appears to have an effect on left ventricular filling because patients with severe LA compression demonstrate improved ventricular volumes after HH repair. Case reports of HH causing hemodynamic instability including hypotension requiring inotropic therapy or resulting in syncope are consistent with these findings (89-92). Impaired ventricular filling due to LA compression may also contribute to exercise intolerance by preventing the necessary increase in cardiac output that normally occurs with exercise.

Naoum et. al. demonstrated compression of the Coronary Sinus (CS) in 87% of patients. (86) The anatomic course of the CS in the posterior atrioventricular groove makes it particularly susceptible to compression. CS compression can lead to diastolic dysfunction and dyspnea. (93) Previous animal studies have confirmed a relationship among CS compression and impaired

myocardial blood flow, increased ventricular blood volume, decreased ventricular distensibility, and diastolic dysfunction (94-96). These may represent further mechanisms of impaired exercise capacity due to cardiac compression by HH.

In summary, dyspnea and fatigue are underappreciated but very important complications of Stage II symptoms with HHs. Patients with large HHs have significant dyspnea and exercise impairment despite normal baseline respiratory function. (97) Significant cardiac abnormalities including compression of the Left Atrium, Inferior Pulmonary Veins, and CS are commonly seen in these patients. In addition to dyspnea, diastolic cardiac dysfunction leads to a sense of chronic fatigue. The recovery of exercise capacity with HH repair is independently predicted by recovery of the LA diameter, suggesting a significant causal role for cardiac compression in the pathogenesis of HH-associated dyspnea and fatigability. Assessment of LA compression severity pre-operatively is a useful non-invasive clinical tool for identifying those patients who will benefit most from HH repair.

Another manifestation of extrinsic compression of the left atrium is syncope. Syncope and dyspnea are provoked by lying down, typically after a large meal.

In cases of large HHs which extend posteriorly into the right chest, the inferior vena cava (IVC) and the hepatic veins are compressed. The IVC lies very close to the right limb of the esophageal crus and undergoes external compression by the large HH. Normally there is no measurable pressure gradient between the IVC and the right atrium. HHs have been reported to increase the pressure gradient between the IVC and right atrium. (98) This results in poor right atrial filling and a "tamponade" physiology, and lower extremity edema. This is yet another cause of diastolic dysfunction in patients with large HHs. Furthermore, with pressure on the hepatic veins the patients can present with ascites. (99)

Hiatal hernia appears to be associated with increased frequency of atrial fibrillation (AF) in both men and women of all age groups. In a large study from Mayo Clinic, Roy et. al. showed that the occurrence of AF was 17.5-fold higher in men with HH and 19-fold higher in women with HH compared to the frequency of AF reported in the general population. (100) Atrial arrhythmias in patients with large HHs results from pressure and stretching of the inferior pulmonary veins by the large retrocardiac mass created by the HH. In addition, this and other studies have shown that patients with AF associated with HH might have a better prognosis than patients with AF without HH. One possibility may be that patients with AF and HH represent a unique subgroup of patients with AF that are actually less likely to develop AF-related complications due to a different mechanism for the AF. These patients may be more likely to have lone AF and less structural heart disease but still develop AF due to the mechanical/neural factors from the effect of the HH on the atria and, consequently, have a lower complication rate. Many studies have suggested that the natural history of AF in patients with HH may be different from AF associated with structural heart disease.

Normally, there is no measurable pressure gradient between the inferior vena cava and the right atrium. The finding of an external pressure on the right atrium combined with a pressure gradient between the inferior vena cava and the right atrium led to the conclusion that the findings are due to external compression by the huge hiatal hernia.

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Gastric Symptoms: The strangulation of the stomach in the esophageal hiatus results in discomfort and pain in the subxiphoid region. Rarely with large HHs the patient may present with gastric volvulus.

Chronic venous congestion of the herniated gastric mucosa along with ulceration (Cameron's ulcers) can also result in occult bleeding leading to iron deficiency anemia. (101-104) Typically this anemia resolves in more than 90% of patients following the hernia repair. (105-106)

It has been shown that there is a high correlation between Delayed Gastric Emptying and "gastroparesis" and the size of the HH. This may be due to compressive effects on the vagus nerves or stretching of the gastric muscle. Delayed gastric emptying is rarely the result of vagal nerve injury at the time of the repair. Delayed gastric emptying improves after repair of HHs. Therefore, Patients with large HHs should be expected to manifest the effects of delayed gastric emptying in the postoperative period. These patients need close monitoring and symptomatic treatment until the delayed gastric emptying resolves.

Diagnosis

Although most patients with HHs are symptomatic, there is still a significant group of patients whose symptoms are not recognized as being caused by a HH and who are diagnosed incidentally during tests performed for other conditions.

The evaluation of these patients usually includes a complete history and physical examination. Standard workup typically begins with a barium swallow, followed by upper endoscopy and esophageal manometry. (107)

Barium swallow is probably the best diagnostic study and gives information about the amount of the herniated stomach and the direction of herniation.(108,109) A computed tomography scan of the chest and the abdomen because it may provide additional information on the type and location of the hernia.(101)

Upper endoscopy is useful for visualization of the esophageal and gastric mucosa, detection of Barrett's esophagus, erosive esophagitis, and Cameron's ulcers. Furthermore, it can also determine if there are any lesions suspicious for malignancy.

The role of manometry in patients with HHs is evolving. In patients with a HH in the range of 2-4cm who would undergo antireflux surgery for Stage I symptoms, manometry is useful in a) determining the pre-intervention esophageal motility and b) the type of fundoplication if the surgeon plans on performing a fundoplication. At our institution our approach to the repair of hiatal hernias does not include a fundoplication. We believe that fundoplication is a non-physiologic procedure from a different era in the understanding of hiatal hernias and GERD. Our patients who have a small hiatal hernia and GERD, undergo repair of the hiatal hernia and Gastroesophageal Valvuloplasty, which is an attempt to repair and recreate the normal

antireflux mechanism. In these patients the surgical procedure is not dictated by the findings of manometry. Rather the manometry data is used to determine the prognosis and to follow the improvement in esophageal motility after the surgical intervention. In patients with Stage II symptoms, it is believed the esophageal dysmotility may be secondary to the distal esophageal obstruction resulting from the hiatal defect. Therefore, preoperative manometry is helpful in following the progression and possible improvement in esophageal motility. Furthermore, esophageal manometry dictates the postoperative use of promotility agents which are used as a “bridge” therapy for the esophagus as it recovers its function following the correction of obstruction, the anatomic and physiologic repair of the hiatal defect and reconstitution of the normal antireflux mechanism.

In patients with small hiatal hernias (2-4cm) and Stage I symptoms, 24-hour pH monitoring may provide a quantitative analysis of reflux episodes and correlate them with patient's symptoms. However, in patients with Stage II disease, 24-hour pH monitoring is not required. (110-111) In fact, preliminary studies from our Institution have shown that in patients with Stage II symptoms, 24-hour pH monitoring will be falsely positive. Patients who are fully acid suppressed by pharmacologic therapy but continue to have symptoms of “heart burn”, have been shown to have positive pH studies. In the face of full acid suppression, it is hypothesized that in these patients, esophageal obstruction leads to bacterial overgrowth and a change in esophageal flora resulting in a low pH environment due to bacterial acid production and not gastric acid reflux.

Indications for Surgery

Gastric volvulus is an absolute indication for emergent surgical intervention and is classically described by the Borchardt Triad, which includes the inability to pass a nasogastric tube, retching without actual food regurgitation, and chest or epigastric pain. (112-113)

The surgical treatment strategy in patients with Stage I symptoms with HHs in the range of 0-2cm, is based on failure of medical therapy, young age, or contraindications for the use of pharmacologic acid suppression.

In patients with Stage II symptoms with HHs greater than 2cm, referred to as paraesophageal Hiatal Hernias, the surgical therapy has been debated extensively. Historically, due to the risks of complications and the mortality associated with emergent surgery, most surgeons opted to repair these HHs regardless of the patient's symptoms.(114) Afterward, the strategy moved away from this attitude to a more conservative one because some studies showed that elective and emergent hernia repairs were equally effective.(115-116) These studies were performed at a time when the symptoms associated with HHs were poorly understood. It is important to note that the end point for these studies was not quality of life or symptom relief, but survival. In addition, there was greater appreciation that repair of large HHs could be a difficult operation and was rarely accomplished with the use of laparoscopic techniques. These procedures were associated with high rate of recurrence and complications. The laparoscopic techniques had shortcomings in terms of two-dimensional visualization and the somewhat rudimentary instrument maneuverability which did not allow for complete dissection of the hernia sac and mobilization of the esophagus. These shortcomings were exacerbated when the HH extended significantly above the diaphragmatic hiatus. Consequently surgeons “settled” for incomplete mobilization of the hernia sac and relied on the “fundoplication” to keep the

stomach below the diaphragm. In turn, fundoplication represented an indirect solution for the anatomic and physiologic problem which was created by the hiatal defect. The shortcomings of the laparoscopic technology contributed to poor surgical results. These issues were particularly important in patients with larger HH's that required extensive mobilization of the esophagus in the posterior mediastinum. (117,118)

In 2002, Stylopoulos and colleagues examined the hypothesis that elective laparoscopic repair should be routinely performed on patients with asymptomatic or minimally symptomatic paraesophageal HHs.(119) A Markov Monte Carlo decision analytic model was developed to track a hypothetical cohort of patients with asymptomatic or minimally symptomatic paraesophageal hernias and reflect the possible clinical outcomes associated with two treatment strategies: elective laparoscopic paraesophageal hernia repair (ELHR) or watchful waiting (WW). The input variables for ELHR were estimated from a pooled analysis of 20 published studies, while those for WW and emergency surgery were derived from the surgical literature published from 1964 to 2000. Outcomes for the two strategies were expressed in quality-adjusted life-years (QALYs). The mortality rate of ELHR was 1.4%. The annual probability of developing acute symptoms requiring emergency surgery with the WW strategy was 1.1%. ELHR resulted in reduction of 0.13 QALYs (10.78 vs. 10.65) compared with WW. The model predicted that "Watchful Waiting" (WW) was the optimal treatment strategy in 83% of patients and ELHR in the remaining 17%. Based on this evaluation, they concluded that WW is a reasonable alternative for the initial management of patients with asymptomatic or minimally symptomatic paraesophageal HHs.

As a result of this study which reflected the shortcomings of the laparoscopic surgical approaches to the repair of HHs, many practitioners advised WW for patients with HHs.

A more recent study from 2018, by Morrow and colleagues, has shown that surgical repair of HHs is superior to WW in terms of quality of life. (120)

Clearly, the indications for surgical repair of HHs have evolved over the years. This evolution has been a function of:

1. Greater understanding of the complex three-dimensional anatomy of the esophageal hiatus.
2. The relationship of the esophageal hiatus to the gastroesophageal antireflux mechanism.
3. The importance of the esophageal hiatus in providing the "skeletal" structure onto which the which the gastroesophageal valve is suspended.
4. The non-gastrointestinal complications such as cardiac, respiratory and hematologic complications that are associated with hiatal hernias.
5. Change in the definition of symptomatic hiatal hernias.
6. Possibility of complex anatomic reconstruction using minimally invasive techniques.
7. Advances in intraoperative visualization and greater instrument dexterity provided by the robotic platform.

Historically, the only symptoms considered for elective repair included severe regurgitation, aspiration, cough, anemia, or dysphagia. However, recent literature suggests that symptoms associated with HHs are much broader than just gastrointestinal issues, and due to the slow progression of disease, are present in a subtle form for a long time. Furthermore, several

quality of life studies have shown that patients are severely debilitated by the extra-gastrointestinal symptoms, but due to a lack of broad appreciation among medical professionals, they are driven to attribute the symptoms to other causes. Finally, many studies have shown that the “heartburn” and other gastrointestinal symptoms which are associated with Stage II of the disease are erroneously attributed to GERD by medical professionals. Therefore, based on our present understanding of HHs, truly asymptomatic patients are rare. Carrott et al. found that symptoms are wide ranging and patients with HHs are often labeled as asymptomatic or minimally symptomatic because the hernia has been present for years in an older patient, and the gradual alterations in eating and postprandial symptoms had been attributed to aging. (121) In addition, symptoms such as dysphagia, early satiety, and postprandial dyspnea are often insidious and increase over the course of many years. While, historically, gastrointestinal symptoms of HHs have been the main focus of the indications for repair, pulmonary, upper aerodigestive, cardiovascular, hematologic and functional symptoms have been severely underappreciated. In fact, many HH repair series in the literature do not assess patients for such symptoms as dyspnea or easy fatigability, likely because in the elderly population these symptoms are often assumed to arise from other comorbidities.(122) On the other hand, patients who are younger (<50 years old) and healthier may be more likely to encounter complications from their HH given their life expectancy.

The capability to perform the operation minimally invasively with greater emphasis on the anatomic and physiologic reconstruction of the hiatus as opposed to fundoplication has provided further impetus for favoring surgical repair. (123-128)

Therefore, as we go forward, the indications for Surgery will be based on the Clinical Stage of HH's. In patients with Clinical Stage I Disease who usually present with a HH less than or equal to 2-3cm, surgery is indicated for failure of medical therapy, young age, contraindications for the use of pharmacologic acid suppression, or any extra-gastrointestinal symptoms. In patients with Clinical Stage II Disease who usually present with varying degrees of gastric migration and distal esophageal obstruction and HHs > 2-3 cm, surgical repair is indicated barring any physiologic contraindications.

The principles of the surgical repair are:

1. Complete dissection of the hernia sac.
2. Preservation of the hernia sac as opposed to resection. In larger HHs the anterior (left) vagus nerve is elevated and displaced with the phrenoesophageal ligament or the anterior sac. One of the common mistakes is to resect the sac. The hernia sac represents an extension of the peritoneum in the antero-lateral aspect of the HH. It is important to recall that a HH represents a “Sliding” HH where the posterior aspect of the hernia is made up of the esophagus as opposed to a peritoneal sac. HHs need to be approached like a “Sliding” inguinal hernia where the hernia is reduced but the sac is not resected as it would result in damage to the cecum in the case of a “sliding” inguinal hernia. In the case of a HH, all tissues should be dissected and replaced into the abdomen. Attempts at resecting the sac result in injury to the anterior vagus or the esophagus.
3. Complete mobilization of the esophagus to the level of the inferior pulmonary vein.
4. Dissection of all periesophageal fatty tissue, the so-called Mediastinal fat pad away from the esophagus.
5. Identification and preservation of both vagus nerves.

6. Dissection and removal of the fatty tissue at the esophagogastric junction (GE fat pad).
7. Posterior Closure of the hiatal "V" by crural re-approximation in a primary fashion using absorbable buttresses (pledgets) for the sutures, without the use of nonabsorbable buttressing material or mesh.
8. Suspension of the esophagus onto the right and left limb of the crus.
9. Recreation of the esophagogastric intussusception and creation of the Gastroesophageal (GE) valve.
10. Anterior closure of the hiatus in a primary fashion over a 60 French esophageal bougie.
11. Suspension of the GE Valve onto the anterior crural closure.

Traditionally, these steps have been accomplished using a left thoracotomy, direct visualization of the hernia, mobilization of the esophagus to the aortic arch, and dissection of the hernia sac. The main advantage of the transthoracic approach is the direct visualization and accessibility of the esophagus, which is essential in this procedure. Proper mobilization of the esophagus is highly correlated to the success rate of the procedure in terms of recurrence, as it ensures a tension-free repair. (129-130)

The advent of laparoscopy introduced an alternative to open procedures. However, laparoscopy has been hampered by the shortcomings of two-dimensional visualization and unwristed instruments that pivot at the level of the trocars on the abdominal wall. Although in experienced hands, these shortcomings have been largely overcome, in common practice, the essential steps of the procedure have not been adequately accomplished.

In general practice of laparoscopic repair, surgeons have used various techniques to overcome the shortcomings relating to inadequate hiatal dissection and esophageal mobilization. These techniques have included relaxation of the diaphragmatic crura, and the use of mesh. The goal of mesh repair has been to oppose the radial tension by strengthening the hiatal orifice. While many surgeons continue to use mesh, this issue continues to be debated, as many studies have shown that mesh does not improve the success of the procedure but it can cause severe complications, such as dislodgement and erosions requiring gastric resection.(131) In fact, a randomized controlled trial from Watson et al. demonstrated similar outcomes between suture and mesh repair.(132)

Another area of controversy where the shortcomings of the laparo-endoscopic techniques have dictated the surgical approach to HHs has been in morbidly obese patients. The connection between obesity, and HH is well established. Wilson et al. found that individuals with a body mass index (BMI) exceeding 30 kg/m² were 4.2 times more likely to have a hiatal hernia than those with a BMI lower than 25 kg/m².(133) However, a 10-year retrospective review of laparoscopic repair of HHs identified obesity as a risk factor for long-term adverse outcomes.(134) In other studies, obesity has also been shown to increase the failure rate of antireflux surgery.(135) Because of the increased risk of surgical failure in this challenging population, a sleeve gastrectomy or gastric bypass has been recommended. (136-138) However aside from the many potential physiologic shortcomings of this indirect approach to the repair of HHs in patients with high BMI's, there are still several sociologic obstacles, such as patient preference and lack of insurance coverage. Many patients with a hiatal hernia do not meet Medicare requirements for bariatric surgery (BMI >40 kg/m², alone, or 35-40 kg/m², with

significant comorbidities). Other patients may meet these requirements but may prefer not to undergo gastric bypass or are unwilling to comply with postoperative lifestyle modifications.

The advent of robotic technology, which provides enhanced minimally invasive capabilities such as three dimensional high definition visualization, and greater and more precise instrument maneuverability in a confined space, has facilitated more extensive mediastinal dissection, full mobilization of the HH and the esophagus, and an accurate anatomic primary reconstruction of the esophageal hiatus. Robotic Repair of HHs provides for an equivalent procedure which has been heretofore performed by a thoracotomy using laparoscopic trans-hiatal techniques. With the results of robotic repair of hiatal hernias, elective repair may be a more appropriate solution in all patients (including patients with high BMI's) with HHs.

The concept of the Robotic Anatomic and Physiologic Repair of HHs (RAPR) represents an evolution in the understanding of the anatomy of the Esophageal Hiatus and its role in the normal physiologic functioning of the Gastroesophageal Antireflux Mechanism. This is somewhat analogous to the evolution of the treatment of Mitral Regurgitation which for the purpose of this argument can be seen as “reflux” of blood through an abnormal mitral valve. Treatment of mitral regurgitation began with medical therapy until prosthetic valves became available in the 1950's and 1960's. During this era, the valve was the focus of attention and it was thought that valve replacement would be an adequate treatment. In the 1970's, 80's and 90's it became clear that the mitral annulus played a significant role in the competence of the mitral valve and that annular dilation could lead to regurgitation and valve dysfunction. Furthermore, it was discovered that the mitral valve mechanism played a significant role in left ventricular function. As a direct result of the evolution in the understanding of the anatomy and physiology of the mitral annulus and the mitral valve, and their interrelated role in preventing mitral regurgitation and preserving left ventricular and left atrial function, the modern treatment of mitral regurgitation focuses on reconstruction of the mitral annulus and the mitral valve. There are some important parallels in understanding the role of the HH (the “annulus”) and the Gastroesophageal Antireflux Mechanism (the “valve”) in the normal physiologic function of the esophagus and the stomach. To use the mitral valve analogy, the treatment of HHs has evolved from concentrating on creating an obstruction to regurgitation as with fundoplication, to a reconstruction of the complex anatomic and physiologic relationship that is present at the esophageal hiatus.

Robotic Anatomic and Physiologic Repair of HHs “stands on the shoulder of giants”. In that, the procedure represents an evolution in the understanding of the very complex anatomic and physiologic relationship at the esophageal hiatus. Furthermore, RAPR, incorporates many of the concepts in previous surgical approaches to HHs in coming closer to seeing the “whole elephant”.

Videos of this procedure can be accessed on:

Video 1: <https://youtu.be/7IM7Nvr6URY>

Video 2: <https://youtu.be/oMSr9ItiW0s>

References

1. Stylopoulos N, Rattner DW. The History of Hiatal Hernia Surgery: From Bowditch to Laparoscopy. *Ann Surg* 2005;241:185-193.
2. Hochberg LA. *Thoracic Surgery before the 20th Century*. New York: Vantage Press.
3. Bettex M, Oesch I. The hiatus hernia saga: ups and downs in gastroesophageal reflux: past, present, and future perspectives. *J Pediatr Surg*. 1983;18:670-680.
4. Schwarz GS. Historical aspects of the anatomy of the cardia with special reference to hiatus hernia. *Bull N Y Acad Med*. 1967;43:112-125.
5. Eppinger H. Allgemeine und spezielle Pathologie des Zwerchfells. In: Frankl-Hochwart L, ed. *Supplemente zu H. Nothnagel, Spezielle Pathologie und Therapie*. Wien: Holder; 1911.
6. Friedenwald J, Feldman M. Report of an interesting type of diaphragmatic hernia of the cardia of the stomach through the esophageal orifice. *Am Med Sci*. 1925;170:263.
7. Akerlund A, Onnell H, Key E. Hernia diaphragmatica hiatus oesophagei vom anastomischen und roentgenologischen gesichtspunkt. *Acta Radiol*. 1926;6:3-22.
8. Ritvo M. Hernia of the stomach through the esophageal orifice of the diaphragm. *JAMA*. 1930;94:15-21.
9. Moore AB, Kirklin BR. Progress in the roentgenological diagnosis of diaphragmatic hernia. *JAMA*. 1930;95:1966-1969.
10. Sauerbruch F, Chaoul H, Adam A. Hiatus hernia. *Dis Med Wochenschr*. 1932;82:1709-1712.
11. Soresi AL. Diaphragmatic hernia: its unsuspected frequency: diagnosis and technique for radical cure. *Ann Surg*. 1919;69:254-270.
12. Harrington SW. Diaphragmatic hernia. *Arch Surg*. 1928;16:386-415.
13. Harrington SW. The surgical treatment of the more common types of diaphragmatic hernia: esophageal hiatus, traumatic, pleuroperitoneal hiatus, congenital absence and foramen of Morgagni: report of 404 cases. *Ann Surg*. 1945;122:546-568.
14. Sweet RH. Diaphragmatic hernia. In: Sweet RH, ed. *Thoracic Surgery*. Philadelphia: WB Saunders; 1950:316-334.
15. Sweet RH. Esophageal hiatus hernia of the diaphragm: anatomical characteristics, technique of repair, results of treatment in 111 consecutive cases. *Ann Surg*. 1952;135:1-13.
16. Allison PR. Reflux esophagitis, sliding hiatal hernia and anatomy of repair. *Surg Gynecol Obstet*. 1951;92:419-431.
17. Allison PR. Hiatus hernia (a 20 year retrospective survey). *Ann Surg*. 1973;178:273-276.
18. Barrett NR. Hiatus hernia. *Br J Surg*. 1954;42:231-243.
19. Cannon WB, Moser A. The movements of the food in the esophagus. *Am J Physiol*. 1898;1:435-444.
20. Code CF, Creamer B, Schegel JF, Olsen AM, Donoghue FE. *An Atlas of Esophageal Motility in Health and Disease*. Springfield, IL: Charles C. Thomas, 1958.
21. Fyke FE, Code CF, Schiegel JF. The gastroesophageal sphincter in healthy beings. *Gastroenterologia*. 1956;86:135-150.
22. Kramer P, Ingelfinger FJ, Atkinson M. The motility and pharmacology of the oesophagus in cardiospasm. *Gastroenterologia*. 1956;86:174-178.
23. Ingelfinger FJ. Esophageal motility. *Physiol Rev* 1958; 38: 533-84.
24. Code CF, Fyke FE Jr, Schlegel JF. The gastroesophageal sphincter in healthy human beings. *Gastroenterologia* 1956; 86: 135-50.

-
25. Pope CE 2nd. A dynamic test of sphincter strength: its application to the lower esophageal sphincter. *Gastroenterology* 1967; 52: 779–86.
 26. Winans CS, Harris LD. Quantitation of lower esophageal sphincter competence. *Gastroenterology* 1967; 52: 773–8.
 27. McNally EF, Kelly JE Jr, Ingelfinger FJ. Mechanism of belching: effects of gastric distension with air. *Gastroenterology* 1964; 46: 254–9.
 28. Dodds WJ. Instrumentation and methods for intraluminal esophageal manometry. *Arch Intern Med* 1976; 136: 515–23.
 29. Dent J, Dodds WJ, Friedman RH, et al. Mechanism of gastroesophageal reflux in recumbent asymptomatic human subjects. *J Clin Invest* 1980; 65: 256–67.
 30. Wyman JB, Dent J, Heddl R, Dodds WJ, Toouli J, Downton J. Control of belching by the lower esophageal sphincter. *Gut* 1990; 31: 639–46.
 31. Pandolfino JE, Ghosh SK, Zhang Q, Jarosz A, Shah N, Kahrilas PJ. Quantifying EGJ morphology and relaxation with high-resolution manometry: a study of 75 asymptomatic volunteers. *Am J Physiol Gastrointest Liver Physiol* 2006; 290: G1033–40.
 32. Nissen R. Eine einfache Operation zur Beeinflussung der Retluxoesophagitis. *Schweiz Med Wochenschr* 1956; 86: 590–2.
 33. Nissen R. Die transpleurale Resektion der Kardia. *Deutsche Z Chir* 1937 ; 249:311–6.
 34. Nissen R. The treatment of hiatal hernia and esophageal reflux by fundoplication. In: Nyhus LM, Harkins HH, eds. *Hernia*. Philadelphia: J.B. Lippincott, 19W;488–96, 1964.
 35. Nissen R, Rossetti M. La fundoplication et la gastropexie dans l'énorme Hiatal. *Ann Chir* 16:825–36, 1962.
 36. Nissen R, Rossetti M. Die Behandlung von Hiatushernien und Refluxoesophagitis mit Gastropexie und Fundoplication. Stuttgart: Thieme, 1959.
 37. Nissen R, Rossetti M. Surgery of hiatal and other diaphragmatic hernias- *J Int Coll Surg* 43:663–74, 1965.
 38. DeMeester TR, Sleisner HJ. Minimizing the side effects of antireflux surgery. *World J Surg* 16:335–6, 1992.
 39. Donahue PE, Samelson S, Nyhus LM, Bombeck CT. The floppy Nissen fundoplication: effective long term control of pathologic reflux. *Arch Surg* 120:663–7, 1985.
 40. Herrington JL Jr, Meacham PW, Hunter RM. Gastric ulceration after fundic wrapping. Vagal nerve entrapment, a progressive causative factor. *Ann Surg* 195:574–81, 1982.
 41. Siewert JR, Feussner H, Walker SJ: Fundoplication: how to do it? Peri-esophageal reflux prevention. *World J Surg* 16:326–34, 1992.
 42. DeMeester TR, Bonavina L, Albertucci M.: Nissen fundoplication for Gastroesophageal reflux disease: evaluation of primary repair in 100 consecutive patients. *Ann Surg* 204: 9–20, 1986.
 43. Ellis FH Jr. Nissen fundoplication. In: Braasch JW, et al eds. *Atlas of abdominal surgery*. Philadelphia: Saunders, 1990 pp.10–6
 44. Codiano C, Revere GQ, Agugiaro S, et al. Technical modification of the Nissen fundoplication procedure. *Surg Gynecol Obstet* 143:977–8, 1976.
 45. Jordan PH Jr. Parietal cell vagotomy facilitates fundoplication in the treatment of reflux esophagitis. *Surg Gynecol Obstet* 147:593–5, 1978.
 46. Hiebert CA: Surgical management of esophageal reflux and Hiatal hernia. *Ann Thorac Surg* 52:159–60, 1991.
 47. Skinner DB, Belsey RH: Surgical management of esophageal reflux and hiatus hernia: Long-term results with 1030 patients. *J Thorac Cardiovasc Surg* 1967; 53:33-54.
 48. Hill LD, Tobias J, Morgan EH. Newer concepts of the pathophysiology of hiatal hernia and esophagitis. *Am J Surg*. 1966;111:70–79.
-

49. Hill LD. An effective operation for hiatal hernia: an eight-year appraisal. *Ann Surg*. 1967;166:681-692.
50. Hill LD, Kozarek RA. The gastroesophageal flap valve. *J Clin Gastroenterol*. 1999;28:194-197
51. Hill LD, Kozarek RA, Kraemer SJ, et al. The gastroesophageal flap valve: in vitro and in vivo observations. *Gastrointest Endosc*. 1996;44:541-547.
52. Collis JL. An operation for hiatus hernia with short esophagus. *J Thorac Surg*. 1957;34:768-788.
53. Pearson FG, Henderson RD. Long-term follow-up of peptic strictures managed by dilatation, modified Collis gastroplasty and Belsey hiatus hernia repair. *Surgery*. 1976;80:396-404.
54. Orringer MB, Sloan H. An improved technique for the combined Collis-Belsey approach to dilatable esophageal strictures. *J Thorac Cardiovasc Surg*. 1974;68:298-302.
55. Thor KBA, Hill LH, Mercer CD, et. al.: Reappraisal of the flap valve mechanism : A study of a new valvuloplasty procedure in cadavers. *Acta Chir Scand* 153:25-28, 1987.
56. Gharagozloo F, Pourshahmir M, Deters K: Comparison of Nissen fundoplication and gastroesophageal valvuloplasty in an experimental model of gastroesophageal reflux disease. *Surg Forum* 10:73-86, 1998.
57. Hill LD, Kraemer SJM, Aye RW, et. al.: Laparoscopic Hill repair. *Cont Surg* 44: 13-20, 1994.
58. Hansdotter I, Björ O, Andreasson A, Agreus L, Hellström P, Forsberg A, Talley N, Vieth M, Wallner B: Hill classification is superior to the axial length of a hiatal hernia for assessment of the mechanical anti-reflux barrier at the gastroesophageal junction. *Endosc Int Open*. 2016; 4(3): E311-E317.
59. Mittal RK, Balaban DH. The esophagogastric junction. *N Engl J Med* 1997; 336: 924-32.
60. Hashemi M, Peters JH, DeMeester TR, et al. . Laparoscopic repair of large type III hiatal hernia: Objective followup reveals high recurrence rate. *J Am Coll Surg* 2000;190:553-560; discussion 560-561.
61. Lal D. Laparoscopic repair of paraesophageal hernia. *Surg Clin North Am* 2005;85:105-118.
62. Landrenau RJ, Del Pino M, Santos R. Management of paraesophageal hernias. *Surg Clin North Am* 2005;85:411-432.
63. Scheidler MG, Keenan RJ, Maley RH, Wiechmann RJ, Fowler D, Landrenau RJ. "True" paraesophageal hernia: A rare entity radiologic presentation and clinical management. *Ann Thorac Surg* 2002;73:416-419.
64. Maziak DE, Todd TR, Pearson FG. Massive hiatus hernia: Evaluation and surgical management. *J Thorac Cardiovasc Surg* 1998;115:53-60; discussion 1-2.
65. Altorki NK, Yankelevitz D, Skinner DB. Massive hiatal hernias: The anatomical basis of repair. *J Thorac Cardiovasc Surg* 1998;115:828-835.
66. Luketich JD, Raja S, Fernando HC, et al. . Laparoscopic repair of giant paraesophageal hernia: 100 consecutive cases. *Ann Surg* 2000;232:608-618.
67. Gharagozloo F, Soberman M, DuBree K, Tempesta B, Gurfinkel G: Correlation of a hiatal hernia and gastroesophageal reflux disease: A prospective blinded study in patients undergoing laparoscopy. *Chest* 1999;116:4:371.
68. Maziak DE, Todd TR, Pearson FG. Massive hiatus hernia: Evaluation and surgical management. *J Thorac Cardiovasc Surg* 1998;115:53-60; discussion 1-2.
69. Wo JM, Branum GD, Hunter JG, et al. . Clinical features of type III (mixed) paraesophageal hernia. *Am J Gastroenterol* 1996;91:914-916.
70. Velanovich V, Karmy-Jones R. Surgical management of paraesophageal hernia: Outcome and quality of life analysis. *Dig Surg* 2001;18:432-437.
71. Andolfi C, Jalilvand A, Plana A, Fisichella PM. Surgical Treatment of Paraesophageal Hernias: A Review. *J Laparoendosc Adv Surg Tech A*. 2016; 26: 778-783.
72. Gagliardi, D., Makihara, S., Corsi, P. R., Viana Ade, T., Wiczer, M. V., Nakakubo, S. & Mimica, L. M. (1998) *Dis. Esophagus* 11, 248-250.

73. Pajecski, D., Zilberstein, B., dos Santos, M. A. A., Ubriaco T. A., Quintanilha, A. G., Cecconello, I. & Gama-Rodrigues, T. (2002) *J. Gastrointestinal Surg.* 6, 723-729.
74. Pei Z, Bini EJ, Yang L, Zhou M, Francois F, Blaser MJ. Bacterial biota in the human distal esophagus. *Proc Natl Acad Sci U S A.* 2004; 101: 4250–4255.
75. Pei Z, Yang L, Peek RM, Jr Levine SM, Pride DT, Blaser MJ. Bacterial biota in reflux esophagitis and Barrett's esophagus. *World J Gastroenterol.* 2005 Dec 14;11(46):7277-83.
76. Liu N1, Ando T, Ishiguro K, Maeda O, Watanabe O, Funasaka K, Nakamura M, Miyahara R, Ohmiya N, Goto H. Characterization of bacterial biota in the distal esophagus of Japanese patients with reflux esophagitis and Barrett's esophagus. *BMC Infect Dis.* 2013;13:130.
77. Johnston N, Yan J C, Hoekzema C R. et al. Pepsin promotes proliferation of laryngeal and pharyngeal epithelial cells. *Laryngoscope.* 2012;122:1317–1325.
78. Samuels T L, Johnston N. Pepsin as a causal agent of inflammation during nonacidic reflux. *Otolaryngol Head Neck Surg.* 2009;141:559–563.
79. Havemann B.D., Henderson C.A., El-Serag H.B. (2007) The association between gastro-oesophageal reflux disease and asthma: a systematic review. *Gut* 2007;56:1654–1664.
80. Zhu J.C., Becerril G., Marasovic K., Ing A.J., Falk G.L. Laparoscopic repair of large hiatal hernia: impact on dyspnoea. *Surg Endosc.* 2011 Jun 3.
82. Low D.E., Sim C.-W. Siu, M.-H. Jim, H.-H. Ho et al., "Recurrent acute heart failure caused by sliding hiatus hernia," *Postgraduate Medical Journal* 2005; 81:268–269.
83. A. M. T. Chau A, R. W.-L. Ma, and D. M. Gold, "Massive hiatus hernia presenting as acute chest pain," *Internal Medicine Journal* 2011; 41:704–705.
84. Lim HS, Leong DP, Alasady M, "Massive hiatus hernia mimicking a left atrial mass," *Heart, Lung and Circulation* 2013; 22: 875–876, 2013.
85. Sahin C, Akin F, Cullu N, Ozseker B, Kirli I, Altun I. A Large Intra-Abdominal Hiatal Hernia as a Rare Cause of Dyspnea. *Case Reports in Cardiol* 2015; Article ID 546395, 4 pages.
86. Naoum C., Falk G.L., Ng A.C.C., et al. Left atrial compression and the mechanism of exercise impairment in patients with a large hiatal hernia. *J Am Coll Cardiol* 2011;58:1624–1634.
87. Siu C.W., Jim M.H., Ho H.H., et al. Recurrent acute heart failure caused by sliding hiatus hernia. *Postgrad Med J* 2005;81:268–269.
88. Raza S.T., Mukherjee S.K., Danias P.G., et al. Hemodynamically significant extrinsic left atrial compression by gastric structures in the mediastinum. *Ann Intern Med* 1995;123:114–116.
89. Devbhandari M.P., Khan M.A., Hooper T.L. Cardiac compression following cardiac surgery due to unrecognised hiatus hernia. *Eur J Cardiothorac Surg* 2007; 32:813–815.
90. Neumann L., Poulton B., Ridley S. Life-threatening complications of hiatus hernia. *Anaesthesia* 1999; 54:93–94.
91. Oishi Y., Ishimoto T., Nagase N., et al. Syncope upon swallowing caused by an esophageal hiatal hernia compressing the left atrium: a case report. *Echocardiography* 2004; 21:61–64.
92. Maekawa T., Suematsu M., Shimada T., Go M. Unusual swallow syncope caused by huge hiatal hernia. *Intern Med* 2002; 41:199–201.
93. Park S.M., Shim C.Y., Choi D., et al. Coronary sinus obstruction by primary cardiac lymphoma as a cause of dyspnea due to significant diastolic dysfunction and elevated filling pressures. *J Am Soc Echocardiogr* 2010; 23:682.
94. Miura T., Hiramatsu T., Forbess J.M., Mayer J.E. Jr. Effects of elevated coronary sinus pressure on coronary blood flow and left ventricular function: Implications after the Fontan operation. *Circulation* 1995; 92:II298–II303.

95. Gaasch W.H., Bing O.H., Franklin A., et al. (1978) The influence of acute alterations in coronary blood flow on left ventricular diastolic compliance and wall thickness. *Eur J Cardiol* 1978;7(Suppl):147–161.
96. Vogel W.M., Apstein C.S., Briggs L.L., Gaasch W.H., Ahn J. Acute alterations in left ventricular diastolic chamber stiffness: Role of the “erectile” effect of coronary arterial pressure and flow in normal and damaged hearts. *Circ Res* 1982; 51:465–478.
97. Marwick TH. When the Stomach rules the heart: Dyspnea as a neglected complication of a large Hiatal Hernia. *J Am Coll Cardiol* 2011; 58:1635-36.
98. Asti E, Bonavina L,a, Lombardi M, Bandera F, Secchi F, Guazzic M. Reversibility of cardiopulmonary impairment after laparoscopic repair of large hiatal hernia. *Int J Surg Case Rep*. 2015; 14: 33–35.
99. Stavi D, Sztarkier I, Chudakov G, Kalchiem-Dekel O. Ascites Worsening Due to a Large Hiatal Hernia Compressing Hepatic Venous Outflow: A Case Report. *Journal of medical cases* 2014; 5:573-575.
100. Roy RR, Sagar S, Bunch TJ, MD, Aman W, Daniel J Crusan DJ, Srivathsan K, Asirvatham S, Shen WK, Jahangir A. Hiatal Hernia Is Associated With an Increased Prevalence of Atrial Fibrillation in Young Patients. *J Atr Fibrillation*. 2013; 6: 894-98.
101. Schieman C, Grondin SC. Paraesophageal hernia: Clinical presentation, evaluation, and management controversies. *Thorac Surg Clin* 2009;19:473–484.
102. Weston AP. Hiatal hernia with Cameron ulcers and erosions. *Gastrointest Endosc Clin N Am* 1996;6:671–679.
103. Maganty K, Smith RL. Cameron lesions: Unusual cause of gastrointestinal bleeding and anemia. *Digestion* 2008;77:214–217.
104. Haurani C, Carlin AM, Hammoud ZT, Velanovich V. Prevalence and resolution of anemia with paraesophageal hernia repair. *J Gastrointest Surg* 2012;16:1817–182.
105. Pauwelyn KA, Verhamme M. Large hiatal hernia and iron deficiency anemia: Clinico-endoscopic findings. *Acta Clin Belg* 2005;60:166–172
106. Hayden JD, Jamieson GG. Effect on iron deficiency anemia of laparoscopic repair of large paraesophageal hernias. *Dis Esophagus* 2005;18:329–331.
107. Bonrath EM, Grantcharov TP. Contemporary management of paraesophageal hernias: Establishing a European expert consensus. *Surg Endosc* 2015;29:2180–2195.
108. Linke GR, Borovicka J, Schneider P, et al. . Is a barium swallow complementary to endoscopy essential in the preoperative assessment of laparoscopic antireflux and hiatal hernia surgery? *Surg Endosc* 2008;22:96–100.
109. Kahrilas PJ, Kim HC, Pandolfino JE. Approaches to the diagnosis and grading of hiatal hernia. *Best Pract Res Clin Gastroenterol* 2008;22:601–616.
110. Wolf PS, Oelschlager BK. Laparoscopic paraesophageal hernia repair. *Adv Surg* 2007;41:199–210.
111. Fuller CB, Hagen JA, DeMeester TR, Peters JH, Ritter M, Bremmer CG. The role of fundoplication in the treatment of type II paraesophageal hernia. *J Thorac Cardiovasc Surg* 1996;111:655–661.
112. Perdakis G, Hinder RA, Filipi CJ, et al. . Laparoscopic paraesophageal hernia repair. *Arch Surg* 1997;132:586–589.
113. El Khoury R, Ramirez M, Hungness ES, et al. . Symptom relief after laparoscopic paraesophageal hernia repair without mesh. *J Gastrointest Surg* 2015;19:1938–1942.
114. Lebenthal A, Waterford SD, Fisichella PM. Treatment and controversies in paraesophageal hernia repair. *Front Surg* 2015;2:13-18.
115. Horgan S, Eubanks TR, Jacobsen G, et al. . Repair of paraesophageal hernias. *Am J Surg* 1999;177:354–358.
116. Floch NR. Paraesophageal hernias: Current concepts. *J Clin Gastroenterol* 1999;29:6–7.
117. Oelschlager BK, Yamamoto K, Woltman T, et al. . Vagotomy during hiatal hernia repair: A benign esophageal lengthening procedure. *J Gastrointest Surg* 2008;12:1155–1162.

118. Horvath KD, Swanstrom LL, Jobe BA. The short esophagus: Pathophysiology, incidence, presentation, and treatment in the era of laparoscopic antireflux surgery. *Ann Surg* 2000;232:630–640.
119. Stylopoulos N, Gazelle GS, Rattner DW, MD. Paraesophageal Hernias: Operation or Observation? *Ann Surg*. 2002; 236: 492–501.
120. Morrow EH, Chen J, Patel R, Bellows B, Nirula R, Glasgow R, Nelson RE. Watchful waiting versus elective repair for asymptomatic and minimally symptomatic paraesophageal hernias: A cost-effectiveness analysis. *Am J Surg*. 2018;216(4):760-763.
121. Carrott PW, Hong J, Kuppusamy M, Koehler RP, Low DE. Clinical ramifications of giant paraesophageal hernias are underappreciated making the case for routine surgical repair. *Ann Thorac Surg* 2012;94:421–122. Carrott PW, Hong J, Kuppusamy M, Kirtland S, Koehler RP, Low DE. Repair of giant paraesophageal hernias routinely produces improvement in respiratory function. *J Thorac Cardiovasc Surg* 2012;143:398–404.
123. Wiechmanm RJ, Ferguson MK, Naunheim KS, et al. . Laparoscopic management of giant paraesophageal herniation. *Ann Thorac Surg* 2001;71:1080–1087.
124. Hawasli A, Zonca S. Laparoscopic repair of paraesophageal hiatal hernia. *Am Surg* 1998;64:703–710.
125. Fullum TM, Oyetunji TA, Ortega G, et al. . Open versus laparoscopic hiatal hernia repair. *JSLs* 2013;17:23–29.
126. Zehetner J, Demeester SR, Ayazi S, et al. . Laparoscopic versus open repair of paraesophageal hernia: The second decade. *J Am Coll Surg* 2011;212:813–820.
127. Hashemi M, Sillin LF, Peters JH. Current concepts in the management of paraesophageal hiatal hernia. *J Clin Gastroenterol* 1999;187:227–230.
128. Oddsdottir M. Paraesophageal hernia. *Surg Clin North Am* 2000;80:1243–1252.
129. Fullum TM, Oyetunji TA, Ortega G, et al. . Open versus laparoscopic hiatal hernia repair. *JSLs* 2013;17:23–29.
130. Zehetner J, Demeester SR, Ayazi S, et al. . Laparoscopic versus open repair of paraesophageal hernia: The second decade. *J Am Coll Surg* 2011;212:813–820.
131. Tam V, Winger DG, Nason KS. A systematic review and meta-analysis of mesh vs suture cruroplasty in laparoscopic large hiatal hernia repair. *Am J Surg* 2016;211:226–238.
132. . Watson DI, Thompson SK, Devitt PG, et al. . Laparoscopic repair of very large hiatus hernia with sutures versus absorbable mesh versus nonabsorbable mesh: A randomized controlled trial. *Ann Surg* 2015;261:282–28.
133. Wilson LJ, Ma W, Hirschowitz BI. Association of obesity with hiatal hernia and esophagitis. *Am J Gastroenterol* 1999;94:2840–2844.
134. Luketich JD, Nason KS, Christie NA, et al. . Outcomes after a decade of laparoscopic giant paraesophageal hernia repair. *J Thorac Cardiovasc Surg* 2010;139:395–404.
135. Anvari M, Allen C, Marshall J, et al. . Randomized controlled trial of laparoscopic Nissen fundoplication versus proton pump inhibitors for treatment of patients with chronic gastroesophageal reflux disease: One-year follow-up. *Surg Innov* 2006;13:238–249.
136. Merchant AM, Cook MW, Srinivasan J, et al. . Comparison between laparoscopic paraesophageal hernia repair with sleeve gastrectomy and paraesophageal hernia repair alone in morbidly obese patients. *Am Surg* 2009;75:620–625.
137. Davis M, Rodriguez J, El-Hayek K, et al. . Paraesophageal hernia repair with partial longitudinal gastrectomy in obese patients. *JSLs* 2015;19:e2015.00060.
138. Patel AD, Lin E, Lytle NW, et al. . Combining laparoscopic giant paraesophageal hernia repair with sleeve gastrectomy in obese patients. *Surg Endosc* 2015;29:1115–1122.